FAMILY PATHOLOGY AND INTERVENTION IN THE TREATMENT OF ALCOHOLISM

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Over the last two decades, consumption of alcohol has been increasing on a global scale. In some third world countries the increase is more rapid than in others. If the steady rise in the per capita consumption of alcohol is continued for another generation, these countries will attain or exceed the present level of consumption in the developed world. It may also lead to a higher incidence of alcohol related problems and prove to be a substantial drain on already scarce economic and social resources. Such alarming possibilities make it imperative that strategies be developed for the management and prevention of alcohol dependence. In this context, the different sociocultural status of these countries becomes significant so far as the choice of methods of treatment is concerned. The vast majority of the Indian population does not approve of alcohol consumption as an acceptable social behaviour. However, the increase in alcohol consumption and its acceptability is readily manifest in the urban, industrialised section of the society, probably due to a shift from the traditional values of a joint family to the modern values of the nuclear family and the resultant lack of social support systems. If this change is considered as a significant one, then the inclusion of family intervention as an adjunct to the other modes of management becomes important. Whereas previously alcoholics were taken to be homeless, jobless, physically ravaged individuals with meagre psychological resources, it is now clear that the 'end stage' alcoholic is not representative of the patient population that abuses alcohol. A significant proportion of the alcoholic population continues to function in within intact and stable family systems.

The family has only recently been viewed as a possible focus of intervention by professionals. Clinical interest has focussed on disturbed communication and structural patterns within the family. Several theories of family functioning, pathology and therapeutic change have been developed, and certain core concepts have gained widespread acceptance. Drawing from the concepts of the general systems theory, the family may be viewed as a primary

organizational unit. Individuals thus represent the component parts of this unit. The emphasis is on the patterns of interrelationships between the component parts, hence the focus on interactional behaviour, structural patterning within the family and the balance or stability of the system as a whole. Any single piece of behaviour in the family has to be understood first in terms of how all the individuals are contributing to make the behaviour possible, and secondly, how the behaviour is affecting all the individuals in the family. Pathology becomes redefined as a structural or functional imbalance in the family, rather than as difficulties being experienced by any single individual in the family.

Families tend to establish a sense of stability and have mechanisms to resist any change. This stability does not necessarily imply a healthy state of affairs. The family might for example, include as part of this stabilization, a form of psychopathology such as chronic alcohol abuse. An extension of this concept implies that an individual might, through his or her symptom expression, be stabilizing the level of functioning of other family members. It may thus be more profitable to view the whole family, rather than the individual alone, as the basic unit of pathology. The behaviour of each spouse may be rigidly controlled by the other. As a result, any effort by one to alter the typical role behaviour threatens the family equilibrium and provokes renewed efforts by the others to maintain the status quo.

Within an alcoholic's family of origin, there is frequently a lack of constructive pressure for change, either because the alcoholism is accepted or because the alcoholic is viewed as helpless and discounted as a person. These influences from the family of origin are carried over into the alcoholic's current nuclear family, and are especially evident in the husband – wife interation. Often the alcoholic marriage can be viewed as a struggle for control. The nonalcoholic spouse may appear overly responsible and dominant, and assume an 'overfunctional' role in contrast to the alcoholic spouse who underfunctions. The alcoholic's drinking may be viewed as neutralizing the overfunctioner's apparent control in

the relationship. The overfunctioner may further perpetuate the drinking by exhibiting anxiety, criticizing and attempting to forbid the drinking, thereby initiating another round of 'counter-control' drinking behaviour by the alcoholic spouse. The overfunctioning spouse may also perpetuate the drinking cycle by indirectly reinforcing the drinking. The spouse may support the drinking behaviour by hiding it from public gaze or at times by even bringing alcohol for the partner. Thus each spouse contributes to the drinking behaviour and each has some needs satisfied by it. However, both spouses also needs that are not met by the drinking behaviour and hence the relationship remains conflictual. The shared fears of separation often keep the couple from risking honest, angry confrontations regarding their conflicting views and they continue in a highly competitive relationship. The alcoholic repeatedly tries to control the situation and yet avoids responsibility through subtle, passive-dependent techniques. The spouse tries for control by being forceful, active, blunt and domineering. Neither achieves dominance, but the fight continues indefinitely.

The male alcoholic gives up his role as a father. Other roles are also rapidly abandoned and taken over by other family members. The wife may encourage an older son to take over the responsibilities abdicated by the father, thus placing the son in a position of overt competition with the father. As the nonalcoholic members take over the full management of the family functioning, the alcoholic is relegated to child status, which perpetuates his drinking. In the immediate situation of the alcoholic family, the children are also severely victimized. They have growth and development problems, school and learning problems, develop emotional problems and may exhibit significant behaviour dysfunction. Further, the children are subject to gross neglect and abuse. The family of the alcoholic provides him with his most important, and potentially his most destructive, interpersonal contacts.

Treated alcoholic patients frequently relapse into former drinking habits when they return to their preexisting dysfunctional family lives. Although

sobriety brings with it the desired goals of behavioural stabilization, it often carries with it seemingly undesirable aspects such as lack of familiarity with new patterns of the marital relationship and new demands for intimacy. Such a model could explain the appearance of significant depression in a nonalcoholic spouse when the alcoholic stops drinking. In fact, many families seek help, not because of the alcohol problem, but because of severe problems in family communication, parent-child conflict, sexual difficulties and the like. When the drinking is under control and the underlying marital and family problems surface, the alcoholic may resume drinking to cover them up, or the couple may may become too threatened and terminate therapy.

There is continuing debate over the structural component of the therapeutic situation in alcoholism, for example, who should the therapy be addressed to. However, family therapy techniques have been used with increasing enthusiasm. In recognition of this trend, the second special report to the U.S Congress on alcohol and health (1974) called family therapy the most notable current advance in the area of psychotherapy of alcoholism. Once attention is focussed on the families of alcoholics, it becomes obvious that the relationship between the alcoholic and his family is not a simple one-way relationship. The family also affects the alcoholic and his illness. The family can either help or interfere with the treatment process. Communication, both verbal and nonverbal, is viewed as reflecting the basic structural and interactional pattern governing the family's behaviour, and therefore frequently becomes the primary focus of attention in therapy. The marital partners go through an initial resistance in which each attempts to pin the rap on the other as the trouble maker in the marriage, followed by a period of insight into their own roles in the maintenance of the marital 'homeostasis'. For this reason it increases the likelihood

that the drinking problem is acknowledged as a problem and also stimulates the motivation for change. The goal of communication centered therapy is to correct discrepancies in communication styles. This is achieved by having messages clearly stated, clarifying meanings and assumptions and permitting feedback. The therapist acts as an objective governor of communication who teaches people to speak clearly and directly in a structured, protected situation.

When working with couples where alcohol is part of the relational system, it is useful to identify one or more functions that alcohol is serving for the couple and to make this the focus of intervention. Focussing on the marital interaction can allay some of the guilt and anxiety of the alcoholic and place responsibility for the situation on both spouses. Thus the emphasis is on an alcohol related problem rather than on labelling one family member as an alcoholic. Joint admission of the spouses to the hospital not only gives the staff an opportunity to observe the couple's interaction, provide feedback to the couple and integrate the spouse into the therapeutic milieu, but also facilitates the couple in learning new ways of adaptive sober behaviour.

Multiple family therapy is the treatment of several families simultaneously through the vehicle of group meetings led by a therapist. It uses the group setting and group processes to assist the couples in examining their marital interactions and the relationship between those patterns of interactions and drinking behaviour. It is unique in contemporary society in that families expose themselves to one another and try to exercise significant effects on one another's way of life. In the process the technique reduces premature dropouts, acts as a preventive mental health measure for other family members, builds an extended good family subculture and creates and supports structural family changes that facilitate abstinence. In the first few sober days of an alcoholic he is so needy that his resistance to the group is low and he has the best acceptability of the group. After a

Chapter VII

Treatment: Emerging Trends in Research and Practice

During the past decade, several issues surrounding the treatment of alcoholism have been subject to controversy. Does the unitary disease concept permit reliable diagnosis for the purposes of early detection and treatment planning? Is treatment effective and, if so, can the costs of treatment be contained? Are there distinct subgroups of alcoholic persons? Can treatment efficacy be improved by matching individual patients to specific types of treatment? Should abstinence be the singular goal of treatment or can some problem drinkers engage in nonabusive drinking? If some can, how can these individuals be identified? The issues raised by these questions imply much about the theoretical and practical challenges facing the treatment field.

This chapter will focus on the state of the art of alcoholism treatment, giving special attention to developments not covered in the Fourth Special Report (USDHHS 1981). These developments include innovations in diagnosis, improved procedures for screening and early intervention, new findings on the costs and benefits of treatment, a continuing debate on the use and viability of controlled drinking therapies, and emerging models of treatment efficacy. An encouraging development in recent years has been the enhanced investment in research and clinical training in the alcoholism field, and the concomitant emergence of a multidisciplinary research community actively involved in the problems of treating alcoholism. As the quality and sophistication of clinical research improve, it is likely that many of the unanswered questions will be solved.

Recent Advances in Diagnosis and Nomenclature

Diagnosis, the process of identifying and labeling specific disease conditions, uses precise attributes, or diagnostic criteria, to classify a sick person as having a disease. The importance of diagnostic criteria derives from their usefulness in making clinical decisions, estimating disease prevalence, understanding etiology, and planning treatment. While the diagnosis of alcoholism may seem straightforward to the concerned layperson, it is in fact a complex process.

Limitations in current diagnostic procedures were recently reviewed by McIntosh (1982), who compared data collected in 31 studies of the prevalence of alcohol-related problems in general hospital populations. Variations in the use of diagnostic criteria, as well as lack of conceptual precision in differentiating alcohol dependence from related disabilities, have led to widely differing prevalence estimates of the number of alcoholic patients. In spite of the fact that these patients were in hospitals, most individuals with drinking problems had neither been diagnosed as such nor received appropriate treatment for their alcohol problems.

The traditional unitary disease concept of alcoholism has been challenged by the observation that there may be multiple patterns of dysfunctional alcohol use, which result in multiple kinds of disability. A corollary of the unitar, disease concept has been the assumption that alcoholics could be clearly differentiated from nonalcoholics on the basis of their distinctive disease characteristics. Known as the binary classification rule, this assumption has led to the search for universal and singular criteria applicable to all alcoholics. This dichotomous approach has not been particularly helpful to programs interested in early intervention (namely, secondary prevention), nor has it been useful in differentiating prognoses within patient samples in ways that could clarify important questions about treatment efficacy. An alternative approach (Mariani 1981) characterizes alcohol dependence along a continuum of severity, with no clear demarcation between the beginning of alcoholism and the end of social drinking. An advantage of this approach is that early detection may be improved when levels of risk have been established.

An important development in the area of diagnosis has been the publication in 1980 of the third edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 1980), better known as DSM-III. As a major revision of the manner in which various disorders, including alcoholism, are diagnosed and classified, DSM-III introduced several innovations designed to address problems in earlier classificatory systems (Spitzer et al. 1980). In contrast to previous editions of DSM, alcoholism is now included within the separate category of substance use disorders rather than as a subcategory of personality disorder. Reflecting a trend toward increased semantic precision, the term "alcohol dependence" is used in preference to the more generic "alcoholism." In addition, a separate category of "alcohol abuse" is added to permit greater differentiation. As delineated in table 1, alcohol dependence is differentiated from alcohol abuse by the presence of tolerance or withdrawal symptoms. Both diagnoses include a pattern of pathological use or impairment in social or occupational functioning due to alcohol. Modeled after diagnostic procedures initially developed for research purpos , (Feighner et al. 1972), these criteria provide a systematic, standardized approach to diagnostic decisionmaking.

DSM-III permits evaluation of the individual's condition in terms of five independent dimensions or axes. Axis I describes the major clinical syndromes, including substance use disorders, while Axis II is reserved for concomitant personality disorders. Axis III classifies physical disorders and conditions, a number of which can be coded as alcohol-induced, including alcohol intoxication, alcohol withdrawal, alcohol withdrawal delirium, alcohol hallucinosis, alcohol amnestic disorder, and dementia associated with alcoholism. Axis IV draws attention to the severity of psychosocial stress (e.g., occupational, interpersonal, physical) that may modify the course of the current disorder. Finally, Axis V permits the clinician to indicate the patient's highest level of adaptive functioning during the past year in terms of social relations, occupational functioning, and use of leisure time.

Preliminary evaluations of DSM-III suggest that it provides reliable and valid identification of alcoholics in clinical settings, but that it may be less useful in detecting alcoholics who are not institutionalized (Mulford and Fitzgerald 1981; Helzer et al. 1981; Hesselbrock et al. 1982). Thus, while the DSM-III alcohol-related diagnoses contain many innovative features, the usefulness of this approach for early identification, epidemiological surveys, and treatment planning remains to be evaluated.

The recent development and successful field testing of two standardized interview schedules, the Schedule of Affective Disorders (Weissman et al. 1980) and the Diagnostic Interview Schedule (Helzer et al. 1981), represent a major advance in clinical diagnosis and psychiatric epidemiology. Both schedules provide objective, standardized procedures for diagnosing alcoholism and other clinical syndromes using DSM-III criteria. As these instruments are used with greater frequency to study clinical and population samples, they promise to advance basic knowledge about alcoholism and its relationships to psychiatric syndromes and personality disorders. Data from household surveys using the Diagnostic Interview Schedule in three National Institute of Mental Health Epidemiology Catchment Areas suggest that, at some time during their lives, one in seven adults 18 years of age or older met criteria for alcohol abuse or alcohol dependence.

A related development in the area of alcoholism diagnosis is the international program on diagnosis and classification sponsored jointly by the WHO School, the Rutgers and Mental Health Administration (ADAMHA) and the World Health Organization (WHO 1982). The purpose of this project is to recommend improvements in sections of the International Classification of Diseases (ICD) dealing with alcoholism, drug abuse, and mental health. The first phase was devoted to a systematic examination of concepts and nomenclature pertaining to alcohol and drug abuse (Edwards et al. 1981). Of particular interest to the alcoholism treatment field is the emerging set of concepts, definitions, and criteria that may well provide the basis for the next revision of ICD. Central to the WHO approach to alcoholism is the concept of a dependence syndrome that is distinguished from alcohol-related disabilities (Edwards et al. 1976, 1981). As outlined in table 2, the dependence syndrome is an interrelated cluster of cognitive, behavioral, and physiological symptoms. Alcohol-related disabilities, on the other hand, consist of those physical, psychological, and social dysfunctions that follow directly or indirectly from excessive drinking and dependence.

Table 1. DSM III criteria for alcohol abuse and alcohol dependence

Diagnostic criteria for alcohol abuse	Diagnostic criteria for alcohol dependence
A. Pattern of pathological alcohol use: need for daily use of alcohol for adequate functioning, inability to cut down or stop drinking, repeated efforts to control or reduce excess drinking by "going on the wagon" (periods of temporary abstinence) or restricting drinking to certain times of the day, binges (remaining intoxicated throughout the day for at least 2 days), occasional consumption of a fifth of spirits (or its equivalent in wine or beer), amnestic periods for events occurring while intoxicated (blackouts), continuation of drinking despite a serious physical disorder that the individual knows is exacerbated by alcohol use, drinking of nonbeverage alcohol.	A. Pattern of pathological alcohol use: need for daily use of alcohol for adequate functioning, inability to cut down or stop drinking, repeated efforts to control or reduce excess drinking by "going on the wagon" (periods of temporary abstinence) or restricting drinking to certain times of the day, binges (remaining intoxicated throughout the day for at least 2 days), occasional consumption of a fifth of spirits (or its equivalent in wine or beer), amnestic periods for events occurring while intoxicated (blackouts), continuation of drinking despite a serious physical disorder that the individual knows is exacerbated by alcohol use, drinking of nonbeverage alcohol.
B. Impairment in social or occupational functioning due to alcohol use: violence while intoxicated, absence from work, loss of job, legal difficulties (e.g., arrest for intoxicated behavior, traffic accidents while intoxicated), arguments or difficulties with family or friends because of excessive alcohol use.	B. Impairment in social or occupational functioning due to alcohol use: violence while intoxicated, absence from work, loss of job, legal difficulties (e.g., arrest for intoxicated behavior, traffic accidents while intoxicated), arguments or difficulties with family or friends because of excessive alcohol use.
	Either tolerance or withdrawal:
C. Duration of disturbance of at least 1 month.	C. Tolerance: need for markedly increased amounts of alcohol to achieve the desired effect, or markedly diminished effect with regular use of the same amount.
	D. Withdrawal: development of alcohol withdrawal (e.g., morning "shakes" and malaise relieved by drinking) after cessation of or reduction in drinking.

Source: American Psychiatric Association 1980.

According to the WHO dependence model, a complete description of an individual's alcohol-related pathology must include statements concerning the nature and severity of dependence, the kinds and degrees of disability, and the personal and environmental factors that influence the drinking problem. The WHO model departs significantly from DSM-III and the older binary classification schemes by emphasizing that both dependence and alcohol-related disabilities exist in degrees rather than in an all-or-none state.

In the brief period since it was first introduced, the dependence syndrome construct has received considerable attention from researchers and clinicians. A number of instruments have been developed to assess its usefulness (Stockwell et al. 1979; Skinner and

Table 2. Constituent elements of the alcohol dependence syndrome

Elements	Interpretation/examples
Narrowing of the drinking repertoire	The drinking pattern tends to become stereotyped around a regular schedule of almost continuous daily consumption.
Salience of drink-seeking behavior	Drinking is given higher priority than other activities in spite of its negative consequences.
Increased tolerance to alcohol	More and more alcohol is required to produce behavioral, subjective, and metabolic changes; large amounts of alcohol can be tolerated.
Repeated withdrawal symptoms	Tremulousness, sweatiness, nausea, etc., appear after short periods of abstinence.
Relief drinking	Withdrawal symptoms are relieved or avoided by further drinking, especially in the morning.
Compulsion to drink	Subjective awareness of craving for alcohol exists, as well as impaired control over quantity and frequency of intake.
Readdiction liability	The syndrome tends to be rapidly reinstated when drinking is recommenced after a period of abstinence.

Source: Adapted from Edwards et al. 1976, 1981.

Allen 1982; Hesselbrock et al. 1983). Research in both experimental and clinical settings suggests that the severity of dependence can correlate positively with attendance at a treatment clinic (Skinner and Allen 1982), cravings for alcohol after a "priming" drink (Kaplan et al. 1983), and failure to control drinking following relapse (Orford et al. 1976; Polich et al. 1981). While further research is needed, the dependence syndrome construct shows considerable promise for early detection, diagnosis, and treatment planning.

Differential Assessment

Both DSM-III and the WHO dependence model represent a general movement toward differential assessment as evidenced by the use of multiple criteria to provide a comprehensive evaluation for treatment planning. Ideally, specificity of diagnosis should lead to specificity of treatment, an important goal in the delivery of treatment services for three essential reasons: (1) economy of cost, (2) avoidance of inappropriate or ineffective treatments, and (3) increased efficacy. Surprisingly, outside the area of personality assessment, little interest has existed until recently in differential assessment of the alcoholic person. This is due in part to assumptions about the unitary nature of alcoholism, and in part to the limited methods available to evaluate alcoholics.

Recent advances in evaluation methodology (Meyer et al. 1981) have stimulated research in this area, and a number of assessment procedures have been developed. In addition to those already discussed, several investigators (Skinner and Allen 1982) have demonstrated the usefulness of assessing drinking patterns and alcohol-related problems in an effort to identify individual differences related to

treatment outcome. Other instruments have been developed recently to provide differential assessment of stages of alcoholism (Mulford 1980), client liabilities associated with poor treatment response (Costello and Baillargeon 1981), severity of addiction-related problems (McLellan et al. 1980), and stability of the male alcoholic's marital situation (O'Farrell et al. 1981). With the burgeoning application of computer technology to individual testing and statistical data analyses, the benefits of differential assessment may well become routine in clinical settings.

Screening and Early Detection of Alcoholism

Consonant with recent conceptual developments in the diagnosis of alcoholism, there has been increasing interest in the development of a simple and accurate screening procedure that could facilitate early identification of alcohol dependence. Clearly, early casefinding can improve the effectiveness of treatment and reduce its costs. Research on early identification has progressed on two relatively independent planes: (1) the search for biochemical markers for alcoholism and (2) the elucidation of psychosocial indicators.

Biochemical Markers

The accumulation of research findings, some of which have already been noted in previous chapters of this report, suggests that several laboratory tests may be useful in the early detection of alcohol abuse. Serum gamma-glutamyl transpeptidase (GGTP) has been suggested as an indicator of heavy alcohol consumption among problem drinkers (Reyes and Miller 1980). Although its usefulness may be affected by concurrent liver pathology, drug use, and individual differences among heavy drinkers (Garvin et al. 1981), its discriminative ability is enhanced considerably when interpreted in conjunction with mean corpuscular volume (MCV) and other tests (Mayfield and Johnston 1980; Chick et al. 1981). That routine blood chemistries may be a useful adjunct for detecting alcoholism is suggested by the results of several studies (Ryback et al. 1980, 1982) that have found that the profiles of 25 biochemical tests differentiated between known alcoholics and nonalcoholics with a high degree of accuracy.

Psychosocial, Clinical, and Combined Assessments

Because some psychosocial symptoms (e.g., escape drinking, concern about a drinking problem, alcohol-related accidents) may appear relatively early in the alcoholic's drinking career, their assessment by means of interviews or self-administered questionnaires has received increasing attention by researchers interested in screening and early diagnosis (Skinner et al. 1981).

Clearly, the most investigated self-administered diagnostic instrument continues to be the Michigan Alcoholism Screening Test (MAST). Consisting of 25 true/false statements describing the typical medical, social, and behavioral problems associated with excessive drinking, the MAST has demonstrated a considerable degree of validity in distinguishing between known groups of male alcoholics and male nonalcoholics (Brady et al. 1982). Concurrent with validation of the MAST has been the development of a variety of similar screening instruments (Brown and Lyons 1981; Skinner et al. 1981). These tests are rapid, inexpensive, and relatively accurate when used under proper conditions. Their simplicity, or gender specificity, however, may limit their usefulness for general population screening, particularly as the obvious intent of the questions leaves them vulnerable to deliberate falsification or unconscious denial.

Recognizing that psychosocial tests have limitations when used in isolation, investigators have begun to use clinical, laboratory, and psychosocial tests in combination. The results of one study (Skinner et al. 1980) underscore the advantages of this approach. Using the Munich Alcoholism Test, a new instrument that combines clinical signs and symptoms (as identified by the physician) with self-report information provided by the patient, it was found that the two types of indicators occurred with relative independence of each other. Psychosocial problems predominated in the younger patients, while clinical signs and symptoms were more frequent in the older drinkers.

From Screening to Early Intervention

Developments in several countries indicate that concerted efforts are now underway to link new screening technologies to low-cost early intervention strategies. Building on previous research (Edwards et al. 1977), which suggested that one session of advice can be as effective as conventional treatment for some alcoholics, a pilot program is currently being evaluated in Scotland to determine the effectiveness of a brief intervention with problem drinkers identified in a general hospital setting (Chick et al. 1982). Preliminary results of a 1-year followup indicate that a single 35-minute counseling session may reduce drinking and its consequences in a significant proportion of newly identified problem drinkers.

In Sweden, an early intervention program was conducted with middle-aged heavy drinkers identified by means of elevated GGTP values (Kristenson 1982). Individuals randomly assigned to a control group received advice to limit their alcohol consumption. Those assigned to an intervention group were repeatedly encouraged to drink less and, in addition, received feedback about their GGTP levels. Compared with the controls, the intervention group showed significant reductions in absenteeism, hospitalizations, and mortality up to 6 years after the initial screening.

In France, screening for problem drinking and alcohol dependence is conducted routinely in industry, health care settings, and the courts. Individuals identified by means of a simple clinical exam (the Le Go grid method) and biochemical tests (GGTP and MCV) are referred to a nationwide system of more than 130 early intervention clinics. While the effectiveness of these clinics has not been studied systematically, the program demonstrates that, from a public health perspective (Babor et al. 1983), early intervention in a large population is now feasible.

Job-Based Interventions

The process by which problem-drinking employees within a work organization are identified and engaged in treatment has been the subject of much discussion (Googins and Kurtz 1980; Roman 1982). Initially, it was advocated that supervisors and managers be trained to identify impaired job performance, and then to confront the employee to determine the necessary corrective action. Supervisors have been found to be inconsistent in confronting their problem-drinking employees (Kurtz et al. 1980), depending on their own attitudes about alcohol use and abuse and their perceptions of the union's support for the program (Beyer et al. 1980). With the impressive expansion of job-based employee assistance programs during the past decade, there has been a shift in emphasis away from the sole identification of alcohol problems to include any employee problem that adversely affects performance (Roman 1981). In the more recently developed employee assistance programs, the role of supervisor as diagnostician has been deemphasized in favor of the more traditional roles of supervising performance and focusing on unacceptable changes in performance as the basis for intervention and referral.

One recent study investigated how the threat of disciplinary action affects treatment outcome (Freedberg and Johnston 1980). Men who chose to enter treatment as an alternative to disciplinary action were compared with others who sought treatment voluntarily. Both groups were similar with respect to job functioning and drinking behavior after 1 year, a finding that denotes the disciplinary process as a useful way to engage employees in treatment. Moberg et al. (1982) report followup data on several hundred inpatients from an abstinence-oriented industrial program. Forty-six percent were abstinent 3 months after treatment, while 37 percent were abstinent after 9 months. About 20 percent were drinking moderately at each followup, but those listed as moderate drinkers at 3 months had a high relapse rate 9 months after treatment. Although social support and employer involvement had a positive influence on outcome, type of referral (voluntary vs. coercive) made little difference.

Despite increasing interest in the evaluation of occupational programs, the relative lack of studies in this area, together with limited access to the worksite, have made it difficult to assess the overall contribution of these programs to improved job performance and reduced health care costs.

Alcoholism Treatment: Programs and Therapeutic Approaches

The treatment system that emerged following the Second World War is varied, complex, and still in the process of development. Many of its programmatic components (e.g., detoxification facilities, inpatient hospital and residential programs, halfway houses, and outpatient clinics), and the therapeutic approaches employed in these components, have only recently begun to receive systematic research attention. What is the most appropriate setting for treatment? What are the most efficacious therapeutic approaches? What is the

optimal reimbursement policy for medicare, medicaid, and private insurance carriers?

Detoxification

The management of acute alcohol intoxication and the concomitant alcohol withdrawal syndrome is commonly referred to as detoxification. The late 1960s, specialized detoxification facilities were developed to address the needs of the public inebriate; these facilities expanded rapidly following widespread adoption by the States of the 1976 Uniform Alcoholism and Intoxication Treatment Act decriminalizing public intoxication. At first these facilities were closely associated with hospital emergency services, but in recent years several alternatives to hospital-based detoxification have emerged. A recent survey of State Alcoholism Authorities (Den Hartog 1982) indicated a threefold increase in the number of States operating or purchasing non-hospital-based detoxification since 1975. The most controversial of these alternatives has been "social setting" detoxification, in which the use of drugs such as diazepam and chlordiazepoxide is avoided in the management of withdrawal in favor of a supportive social climate designed to engage the patient in further treatment.

Because the different detoxification methods have not been assessed directly in controlled comparisons, little is known about the relative merits of various approaches in relieving the distress associated with alcohol withdrawal, preventing the risks of medical complications, and facilitating the process of referral to long-term treatment. The need for hospital-based, medical detoxification, as well as the referral to social setting facilities, depends on the patient's physical condition, nutritional status, severity of alcohol dependence, and overall medical evaluation.

The growth of social setting detoxification has resulted from its lower cost, the changing attitudes about the use of sedatives and tranquilizers in withdrawal management, and evidence indicating that a significant number of patients do not experience serious medical complications during withdrawal (Diesenhaus 1982). A recent review of the published literature in the United States, Canada, and Europe (Den Hartog 1982) concluded that detoxification could occur humanely, safely, and efficiently in nonhospital settings. These reports need to be substantiated by systematic research, since it is not clear what proportion of alcoholics require detoxification in hospital settings. Moreover, while the proponents of social setting detoxification contend that it will facilitate referrals to long-term treatment, insufficient evidence exists to conclude that this type of referral will occur more efficiently in either social or medical settings.

What is clear from the experience of nonhospital detoxification programs is that medically oriented inpatient detoxification may not be necessary for the majority of referrals who are not severely dependent and who are otherwise in good health (Diesenhaus 1982). With the increasing availability of ambulatory and inpatient social setting detoxification, careful study of the nature and role of detoxification in the rehabilitation process would seem warranted. The long-range implications of this trend need to be considered.

The high rate of relapse among detoxified patients is another major concern to service providers. Although it may be unrealistic to expect an initial care component to accomplish much more than detoxification and referral, at present a small proportion of the public inebriate population utilizes a disproportionate share of detoxification resources. Further, these patients often refuse recommended referrals to intermediate care facilities. One study of a predominantly skid row population (Fagan and Mauss 1978) found that fewer than one-third accepted their recommended treatment referral after detoxification. Another study (Edelman and Smart 1981) found that the probability of further referral was lowest in those patients having a history of multiple detoxifications. Two possible solutions to the problem of "resource absorption" by detoxification repeaters have been suggested. The first is the greater availability of comprehensive services to the skid row population; the second is the use of incentives to motivate the public inebriate to enter and remain in treatment (Fagan and Mauss 1978).

Benefits of Treatment

The rapid growth of insurance coverage for alcoholism treatment has been predicated in part on the assumption that such treatment is cost-beneficial, i.e., the long-term costs to both the individual and society will be reduced. Alcoholics and their families have been found to use a disproportionate amount of inpatient and outpatient medical services for a wide variety of physical problems related to excessive drinking (Roghmann et al. 1981; Putnam 1982a). Reflecting an increased concern with the implications of treatment reimbursement policy, a number of new studies have used the improved methods of cost-effectiveness and cost-benefit analysis to show that cost savings can be accrued over a period of time (Swint and Nelson 1977). The first major review of research conducted during the 1970s (Jones and Vischi 1979) found surprising consistency across 12 studies. Despite some methodological problems, these studies indicated significant reductions in medical care and sick leave expenditures related to various kinds of alcoholism treatment, amounting to a 90-percent median reduction in sick days and accident benefits. Additional studies since that time have confirmed this conclusion (Saxe et al. 1983).

Substantiating these findings is a recently conducted study of the employees of California in which 90 families with an alcoholic member, all enrolled in Blue Cross/Blue Shield, were followed for a period of 3 years. Results indicated that total medical care costs per family member decreased substantially over time once the alcoholic member entered treatment (Holder and Hallan 1981).

These studies have been conducted predominantly within health maintenance organizations (HMOs), primarily because alcoholics are relatively easy to track within these comprehensive health programs. A major study of alcoholism treatment in HMOs recently completed by the Group Health Association of America (Plotnick et al. 1982) compared the utilization of four HMO outpatient alcoholic treatment programs by alcoholic persons from 2 years before entering treatment to 48 months after treatment. The study revealed significant reductions in general health care use for the alcoholic persons and their family members. In another HMO study (Forsythe et al. 1982), treated alcoholics were compared with nonalcoholics over a 4-year period. Although the cost differential between the two groups was substantial during the entire study periods, there was a significant decline in alcoholics' demand for services following referral to treatment.

Focusing on the illnesses that may precipitate alcoholics' referral to treatment, Putnam (1982a) found that alcoholics manifested a high number of physical and emotional illnesses. Problems most likely to be associated with the alcoholics' hospitalizations entailed psychosocial difficulties such as anxiety, depression, and marital discord, as well as accidents, drug overdoses, and violence. In many cases, the alcoholics were found to be receiving inappropriate care and receiving inappropriate treatment, since the alcoholism underlying their illness was often not properly diagnosed.

In a related study of the same HMO (Putnam 1982b), alcoholics who received outpatient alcoholism treatment were compared with alcoholics who were identified but refused to accept treatment. An examination of the utilization rates for medical care services before and after referral to treatment revealed that both groups showed a higher level of service use in the 4-month period prior to referral. Those who accepted alcoholism treatment, however, showed a progressive decrease in use of services during the subsequent period, while those who refused treatment increased their demand for services approximately 13 months after the attempted referral. This increase is related to a high incidence of problems associated with accidents, drug overdoses, and violence, and highlights the importance of appropriate diagnosis and treatment to reduce both the human and the social costs of alcoholism.

Finally, a significant non-HMO-based study (Holder and Hallan 1981) followed the families of 90 State employees in California for a period of 3 years. Each family had an alcoholic member and all were enrolled in Blue Cross/Blue Shield. The study indicated that total medical care costs per family member decreased substantially over time once the alcoholic family member entered treatment. At the end of the study, inpatient costs per person per month of both the comparison families (N=83) and the alcoholic families (N=90) were similar, and outpatient costs of the comparison families were actually higher.

Although many of the studies of alcoholics' use of health services are limited in scope, methodology, and populations investigated, the evidence suggests that (1) alcoholics and their families initially use more health care services than nonalcoholics; (2) this elevated demand can be reduced substantially by treatment for alcoholism; and (3) the benefits of alcoholism treatment clearly outweigh its costs.

Treatment Settings

Studies comparing the effectiveness of inpatient programs with outpatient and day hospital treatment are important because of the potential for improved cost-effectiveness and the appeal these approaches may have for alcoholics who reject inpatient or residential care. In one study (Longabaugh et al. in press), patients participating in a day hospital pro-

gram (while returning home at night) were given the same behavioral treatment program that hospitalized inpatients were receiving. At 6-month followup, both groups were comparable on measures of drinking behavior, need for rehospitalizations, and social and occupational functioning. The partial hospitalization group was superior on measures of psychological well-being. Given the lesser cost of partial hospitalization, the authors conclude that this form of treatment is more cost-effective. In another study, comparable findings are reported after 1-year followup of 100 patients who were randomly assigned either to a day clinic or to an inpatient program offering a similar treatment program (McLachlan and Stein 1982). These results support the conclusion that the two types of treatment setting may be equally effective.

Studies comparing the results of inpatient and outpatient programs have generally not shown significant differences in the effects of treatment setting (see review by Cole et al. 1981). However, it seems likely that day hospital and outpatient treatment programs will prove to be substantially less effective for certain groups of patients, and Spicer et al. (1981) caution that conclusions regarding equal effectiveness should be limited to clients who are appropriate for each type of program. Many studies comparing outpatient with inpatient treatment have not controlled for the possibility that patients choosing outpatient settings are less severely dependent. Inpatient treatment seems indicated when motivation is weak to continue treatment, when patients are psychotic, depressed, or suicidal, and when complicating physical disabilities are present. Other factors which may also influence the choice of treatment setting include patients' social stability and the number and severity of their symptoms, as well as the ability of programs to respond to individual needs (Cole et al. 1981; Spicer et al. 1981). Research is needed to identify the characteristics of patients most likely to benefit from an outpatient program, followed by treatment-matching studies to test the validity of the identified patient types. An additional obstacle to the substitution of residential treatment by outpatient treatment is the high rate of client attrition usually encountered in the outpatient setting.

Matching Clients with Programs

Considerable selection takes place on the part of both the client and the referral agent in the natural process of identifying alcoholics and referring them to treatment (Pattison and Kaufman 1981; Beckman and Kocel 1982). Despite the increasing variety of programs, settings, and treatment modalities, many alcoholics do not have the opportunity to find an informal match between their own specific needs and the type of treatment available. This is particularly true of special needs populations, such as women, ethnic minorities, the multidisabled, the elderly, and skid row alcoholics. For these groups, access to treatment and successful rehabilitation are often impeded by cultural barriers, financial constraints, and program design characteristics. Since considerable attention has been and is being given to the needs of these groups in the Fourth Special Report (USDHHS 1981) and other sources (Diesenhaus 1982; NIAAA 1982), this section will provide a brief update on the issues common to all special needs populations.

Because middle-aged white men represent the typical clientele of most treatment facilities, programs often are not designed with the needs of other groups in mind. These groups may thus be reluctant to seek help because of a number of structural and group-specific barriers that restrict access to appropriate facilities. For example, a survey of 93 California treatment facilities (Beckman and Kocel 1982) suggested that many alcoholics were less likely to enter programs lacking child care services, professional staff, and aftercare programs. However, Institute experience in providing services for women reveals that many women do not utilize child care services even when provided. Other barriers to treatment for some groups are language differences and composition of treatment staff. Minority staff members represent only a small proportion of the Nation's alcoholism treatment personnel (NIAAA 1980). Yet another barrier may be financial constraints. Women and minorities tend to be overrepresented in publicly funded facilities and underrepresented in private ones (NIAAA 1980). Without programs' sensitivity to the needs of these populations, many individuals may fail to seek treatment until their alcoholism has reached a severe stage of development.

With the growing recognition that utilization rates may be improved by removing barriers to access, greater attention is now being given to special population groups in the design of treatment programs. Some facilities seeking to attract Hispanic and Native American clients are using folk medicine and native healing approaches as alternatives or adjuncts to traditional medical and psychiatric treatments. Counselors and other treatment staff members are being matched to the sex and ethnic background of their clients. Also, conveniently located, community-based programs, at times staffed and planned in collaboration with special population groups, are appearing more frequently (NIAAA 1982).

What is uncertain at this time, is the impact the ADM block grant program will have on treatment programs. Of particular interest will be the differential rates of service use across States, by both the general population and special population groups. As these programs become available to special population groups, research is needed to learn from program descriptions to actual evaluation studies in which programs designed for special population groups are compared with more traditional approaches.

One exception to the trend to develop services for special population groups and subtypes of alcoholic persons has been the small number of combined programs serving both alcoholics and drug dependent persons. Although combined treatment for alcoholics and drug abusers has not been widely adopted, the feasibility of such programs may have important implications for the planning of future treatment services, given the increased tendency for patients to present multiple abuse or addiction patterns at the time of treatment (Sokolow et al. 1981).

Therapeutic Approaches

Several diverse treatments are often delivered within the context of alcoholism services, depending on the resources and needs of clients, as well as the specific training or orientation of the staff. In many cases, a combination of therapeutic interventions is provided to all clients, under the assumption that multiple treatments stand a good chance of meeting at least some of each client's needs. As the alcoholism treatment system has grown in size and complexity, evaluating individual therapeutic approaches in isolation from one another has become more difficult. Many studies reported in the literature involve adding a treatment of interest to a facility's standard therapeutic program, or are descriptive evaluations rather than clinical trials comparing one or several treatments with the absence of treatment. Studies discussed here generally employed an experimental methodology, as firm conclusions about efficacy can be made only through systematic comparisons.

Behavior Therapy

Behavioral elements most frequently applied in treatment programs include social skills and assertiveness training, contingency management, deep muscle relaxation, self-control training, and cognitive restructuring (Miller and Mastria 1977). Despite their widespread adoption, these methods have not been subject to systematic evaluation with random assignment of large numbers of patients to various treatment conditions.

Aversion therapy.--This approach to treatment grew from Pavlovian conditioning theory, which predicts that the sight, smell, and taste of alcohol will acquire aversive properties if repeatedly paired with noxious stimuli. A thorough review of the research in this area (Miller and Hester 1980) concluded that emetic therapy producing) aversive conditioning can be effective for employed, married, well-motivated alcoholics, but that the effectiveness of electrical aversion is doubtful. These conclusions are supported by recent work (Cannon et al. 1981) comparing these two aversion procedures when used in addition to a standard treatment program. Patients exposed to emetic conditioning exhibited significantly greater improvement after 6 months than patients exposed to conditioning with electric shock, but the advantage after 1 year was not significant.

Work has also proceeded on covert sensitization, a procedure in which imagined scenes of alcoholic consumption are paired with nausea induced by verbal suggestion. This method may overcome some of the practical disadvantages of emetically based aversion therapy. Some recent studies have obtained support for this procedure. One study found that patients receiving covert sensitization remained abstinent longer and demonstrated better psychosocial adjustment than patients in control groups (Elkins 1980). In a related study, patients receiving covert sensitization in a standard inpatient treatment program showed greater improvement over a 4-year period than patients who received insight-oriented therapy (Olson et al. 1981).

Extinction.--Recent experiments have shown that alcohol-related stimuli (such as the sight or smell of an alcoholic beverage) can induce both physiological changes and altered behavior in alcoholic patients (Kaplan et al. 1983; Pomerleau et al. 1983). Because condi-

alcohol abuse rather than alcoholism. Similar semantic problems clouded the meaning of moderation. Several studies (Armor et al. 1978; Moos et al. 1981) have included amounts of up to 5 ounces of absolute alcohol per day within their definitions of moderate drinking. While many patients were drinking far below this limit, the use of such a high cutoff point would tend to inflate estimates of the proportion of moderate drinkers. Furthermore, to the extent that cognitive impairment and other alcohol-related disabilities may result from consuming these amounts (Wilkinson and Sanchez-Craig 1981), this liberal definition of moderation may be questioned in terms of its health implications.

The controversies generated by such studies as the Rand reports (Armor et al. 1978; Polich et al. 1981) illustrate the need to apply sophisticated and systematic research methodology to explorations of the following questions: In the small number of alcoholic subjects who have reportedly established a pattern of moderate drinking, how stable is that pattern over time? How much is this drinking pattern altered by stress, anxiety reactions, and environmental factors, such as media advertising and the cost of alcoholic beverages? What are the risks of resuming drinking compared with abstinence in this group? What are the characteristics of patients who have developed moderate drinking patterns in terms of age, sex, socioeconomic level, marital status, and drinking history? To what extent does the existence of a belief in moderate drinking increase the risk of relapse in alcoholic patients?

Despite questions raised about treatment goals for alcoholic persons, research has nevertheless proceeded on the effects of teaching moderation to socially stable problem drinkers (Heather and Robertson 1981). Pomerleau et al. (1978) have demonstrated that behavioral treatment can be used effectively for teaching moderation to middle-income nonalcoholic problem drinkers, while others have explored some dimensions of teaching nonproblem alcohol consumption skills. Effective elements of treatment that have been identified include modeling and repeated practice of a clearly delineated pattern of moderation (Strickler et al. 1981), and empathy on the part of therapists (Miller et al. 1980). These must be viewed as suggestive findings, since the results of these treatment studies have yet to be confirmed with various subgroups of problem drinkers, in natural environment settings, or with studies of long-term outcomes.

In summary, the issues surrounding moderate, nonproblem drinking as a viable treatment option for nonalcoholic problem drinkers are not likely to be completely resolved until progress has been made in those areas most relevant to this controversy: improved nomenclature, more precise diagnostic criteria, new techniques for differential assessment, earlier treatment interventions, better matching of client needs to specific treatments, and improved research methodologies. It should be clear that while continued exploration and assessment of a variety of treatment options and goals for nonalcoholic and/or prealcoholic persons are appropriate endeavors, the consensus of clinical opinion is that the most appropriate goal for alcoholic persons is abstinence.

Alcoholics Anonymous

With nearly 50 years of service to alcoholic persons and their families, the fellowship of Alcoholics Anonymous (AA) has not been the subject of systematic research to study its long-term and short-term efficacy. To this end, Glaser and Ogborne (1982) reviewed the clinical and research literature, and have proposed a number of research questions and designs to highlight the most effective elements of the AA program and describe those persons for whom AA participation is the preferred approach.

Efforts to identify patients who are best suited to the AA approach have thus far sought to identify characteristics of those who have become actively involved in the AA program (e.g., Boscarino, 1980; O'Leary et al. 1980). Some common characteristics have been identified, including need for structure and for affiliation with a group. As yet, no consistent profile has emerged to characterize successful AA members in terms of degree of impairment, social stability, or emotional disruption. This state of affairs has been attributed to inconsistencies of findings among studies, differing focuses of attention across studies, and methodological inadequacies (Ogborne and Glaser 1981).

Alcoholics Anonymous is a major voluntary resource in the treatment of alcoholism, with a reported membership of 476,000 in the United States and Canada in 1980. The most comprehensive information on membership comes from the organization itself (Alcoholics Anonymous 1981). The triennial sample sur-

veys conducted since 1968 profile characteristics and trends of the membership. For example, membership has increased from 170,000 in 1968 to 676,000 in 1980; the proportion of women increased from 22 percent in 1968 to 31 percent in 1980; the proportion of people 30 and under has increased to 14.7 percent in 1980 from 11.3 percent in 1977 when this trend toward younger membership was first noted; and the proportion who state counseling agencies and treatment facilities as important in their attending their first AA meeting increased from 19 percent in 1977 to 26 percent in 1980. (Of those members beginning AA since the 1977 survey, 13 percent indicate counseling and treatment referrals were most responsible for first attendance.)

Dynamic Psychotherapy and Group Therapy

Numerous descriptions of the application of dynamic psychotherapy to the treatment of alcoholism exist (Dean and Zinberg 1981; Zimberg, 1982), but treatment in outcome studies evaluating this approach to treatment have been conducted. In recent years, psychotherapeutic approaches have given way to, or have been combined with, behaviorally oriented treatment in many settings. Some clinicians have argued against the use of psychotherapy on the grounds that it may strip patients of defense mechanisms that could be used in the service of sobriety (Wallace 1978), and that it makes demands that alcoholics are unready to meet in the early phases of recovery (Vaillant 1981). Nevertheless, there are indications that psychodynamically oriented group psychotherapy may in fact be preferred for certain subgroups of alcoholic persons (Kripp 1977).

A considerable body of literature describes group therapy techniques in the treatment of alcoholism (e.g., Vannicelli 1982), but with only a few evaluative studies to support its efficacy. A recent study by Oei and Jackson (1980) compared group with individual therapy, each of these approaches being used to provide social skills training to some patients and traditional supportive therapy to others. Significant improvement was found only in patients given social skills training, with greater improvement in those trained in a group setting than among those treated individually. Given the potential cost-effectiveness of group therapy, as well as its widespread use, these findings should be followed up with comprehensive evaluative studies.

Family Treatment

The accumulating evidence documenting the deleterious effects of alcoholism on families has prompted heightened interest in family treatment of alcoholism, as described in some detail in the Fourth Special Report (USDHHS 1981). Although the therapeutic value of family treatment is well documented and heralded for many related problem behaviors (Pattison and Todd 1982), its unique contribution and efficacy with alcoholism treatment remain to be fully demonstrated and quantified (Steinglass 1979; Pattison and Kaufman 1981). Preliminary results from a study comparing degrees of spouse involvement in outpatient alcoholism treatment indicate certain advantages for marital therapy, but 18-month outcome data have not yet been reported (McCrady and Noel 1982). Considerable work in evaluating family therapy with alcoholic families is needed, especially efforts to match specific treatments to the problems presented by different families.

Pharmacotherapy

The value of pharmacological agents such as disulfiram (Antabuse) as a deterrent to drinking and various patient factors associated with favorable outcome is of continuing research interest. In recent work, Fuller and Roth (1979) randomly assigned 128 men to receive either a standard dose of disulfiram, a pharmacologically inactive dose, or a daily dose of the vitamin riboflavin. The standard dose of disulfiram was more effective than riboflavin in producing abstinence after 1 year, although differences were small (Fuller and Williford 1980).

One aspect of treatment with disulfiram has been patient noncompliance, that is, the patient's unwillingness to continue regular ingestion. Azrin et al. (1982) studied compliance-enhancement procedures with patients in a rural outpatient clinic. A behavioral "disulfiram assurance" program, involving five sessions of stimulus control training, role playing, and communication skills training, was highly effective in promoting abstinence in married clients, but had little effect on single clients. However, the addition of behavior therapy (training in drink refusal, social skills, and muscle relaxation, and counseling in recreation activities and job-finding) was sufficient to produce nearly complete abstinence for single clients. These are intriguing results, especially since they were obtained in relatively few sessions and with only monthly followup contacts thereafter.

Another study tested the effects of a compliance-enhancement procedure in an industrial setting (Robichaud et al. 1979) and demonstrated the effectiveness of closely supervised ingestion of disulfiram for reducing absenteeism, but only during the treatment period. The findings regarding disulfiram should be regarded as preliminary, but they do suggest that disulfiram may have a useful role in treatment if compliance can be ensured. Future studies of compliance with broader ranges of clients are needed and may be facilitated by newly developed methods for monitoring use of disulfiram (e.g., Paulson et al. 1977; Neidenhiser and Fuller 1982).

A possible role for lithium in the treatment of alcoholism has been considered because of its effectiveness in treating affective disorders and the relatively high incidence of these disorders in alcoholics. The findings thus far have been equivocal. In one study, depressed patients taking lithium had fewer drinking episodes compared with placebo control patients, but without any greater alleviation of the depression than occurred in the control patients (Kline et al. 1974). In other studies, however, depressed patients did not consume less alcohol during lithium therapy (Pond et al. 1981). The possible value of lithium in treating alcoholics thus remains uncertain, with more definitive studies required, employing double-blind procedures and comparisons with other forms of treatment (McMillan 1981).

Ciraulo and Jaffe (1981) have reviewed the use of tricyclic antidepressants in alcoholics and report success in treating initial symptoms of withdrawal, such as anxiety, depression, and somatic discomfort, only within the first 2 to 3 weeks after cessation of drinking. In a subsequent study, however, they demonstrated that alcoholics whose greater relapse rate and lower plasma levels of imipramine than nonalcoholics, suggesting that previous studies which found persistent restlessness and malaise may have utilized dosages that were inadequate for alcoholics (Ciraulo et al. 1982). Future studies should seek to define subtypes of depression in alcoholics, monitor plasma levels of antidepressants, and evaluate drug effects both on depression and on drinking behavior.

Factors Affecting Treatment Outcome

Quality of Treatment

As in other areas of psychotherapy, treatment outcome in alcoholism is affected by the perspectives and attitudes of the therapists, which in turn are functions of their training, experience, and self-esteem (Cartwright 1980). Treatment outcome is also affected by the interpersonal skills of the alcoholism counselor. Valle (1981) evaluated treatment outcomes in patients randomly assigned to eight recovered alcoholic counselors and found that counselors having a higher level of interpersonal functioning tended to have patients who drank less, relapsed less often, and recovered more quickly after a relapse.

Evaluations of training programs for alcoholism counselors indicate that they are able to achieve gains in such areas as participants' knowledge, attitudes, ego strength, capacity for self-disclosure, and effectiveness of counseling (Gideon et al. 1980). The success of alcoholism counselor training programs has prompted their use as a treatment method with chronic alcohol- and drug-dependent individuals, with positive results sustained for at least 1 year (Kahn and Stephen 1981).

Response to Treatment

Treatment research has increasingly focused on identifying personal and environmental factors that predict positive or negative treatment outcomes. Positive outcomes are no longer conceived solely in terms of total abstinence from alcohol, since posttreatment functioning in such domains as physical health, psychological adjustment, social functioning, and occupational performance may not deteriorate automatically with relapse to drinking (Finney et al. 1979); moreover, successful abstinence is not necessarily associated with good functioning in other areas of adjustment.

One form of negative outcome is attrition, that is, failure to complete the treatment program. Client factors found to be related to attrition include inadequate financial resources, low social stability, and youth (Weiss et al. 1981; Keil and Esters 1982). Treatment variables such as program duration and size of treatment groups also can influence the

dropout rate (Schroeder et al. 1982). Undoubtedly, attrition results from an interaction between individual and program factors and may be reduced by improvements in program design and the method of assigning patients to treatments.

It is generally believed that treatment outcome is affected less by the treatment process itself than by the personal resources and characteristics the client brings to the treatment situation. Numerous studies have shown that prognosis to be associated with social stability and marital adjustment (Baekeland 1977). Personal characteristics recently reported to be associated with poor prognosis include cognitive impairment (Abbott and Gregson 1981) and depression (Hatsukami et al. 1981). While client characteristics seem to be important determinants of outcome regardless of the quantity or quality of treatment, other research has shown that this effect may be mediated indirectly by the environment to which the client returns after residential treatment (Cronkite and Moos 1980). Another study found that three situational factors (negative mood states, interpersonal conflicts, and social pressures to drink) were most likely to precipitate relapse after treatment (Cummings et al. 1980). These studies indicate that therapeutic efforts must deal not only with the individual characteristics of the patient but also with the environmental contexts in which the patient is expected to function after treatment.

With this in mind, some have advocated giving more attention to restructuring of the client's environment through marital or family therapy, while others have called for more general approach focusing on frequent aftercare contact in the period after treatment (Costello 1980). As a neglected dimension in the treatment system, aftercare consists of (a) ongoing supportive activities, such as professional and self-help programs designed to maintain treatment gains, (b) prevention of costly rehospitalizations, and (c) improvement in social and occupational functioning. At present, the resources most frequently available to fulfill these functions are halfway houses, AA groups, and program-sponsored support groups. In general, affiliation with aftercare groups is associated with better treatment outcome (Costello 1980). Further research is needed to determine the relative efficacy of different kinds of aftercare, the optimal frequency and duration of aftercare, and how clients can be induced to comply with aftercare without dropping out.

Matching Patients to Therapies

The emerging concept that alcoholism is not a unitary disorder has stimulated renewed interest in delineating different subgroups or types of alcoholic persons. As discussed previously, the goal of this research is to facilitate treatment planning and improve treatment outcome by matching types of alcoholic persons with the most appropriate treatment interventions. Although this typological approach is not new to the field of alcoholism, recent studies have benefited from improvements in assessment technology (Meyer et al. 1981) and from the application of more sophisticated statistical techniques (Skinner 1982).

Attempts to differentiate alcoholics on the basis of personality characteristics account for most typological research efforts (Morey and Blashfield 1981). Several independent studies have denoted two common subgroups (1) passive-dependent alcoholic persons characterized by antisocial personality disorder and (2) neurotic alcoholic persons who may use alcohol as a coping mechanism. In a study designed to explore the often-noted association between alcoholism and antisocial (psychopathic) personality, Hesselbrock et al. (in press) compared alcoholics having an early history of social problems (i.e., those who were relatively free from problems before the onset of alcoholism. Alcoholics with antisocial personality were found to have an earlier onset of alcoholism, as well as a more rapid and severe progression of drinking problems. Several other studies (McLellan et al. 1981; Zivich 1981) suggest the importance of psychiatric disturbance, particularly in the alcoholic's response to treatment. Alcoholic subgroups characterized by poor psychological adjustment were found to show little or no improvement following treatment, whereas those having high adjustment levels showed significant improvement. McLellan et al. (1981) conclude that because patients having less severe psychological disturbance respond to most kinds of treatment, cost considerations would recommend this subgroup to outpatient settings.

The probability of a genetic predisposition to alcoholism has prompted the search for an alcoholic subtype related to family pedigrees. One study of more than 7,000 alcoholic men found that those with a family history of alcoholism had more severe symptomatology, more antisocial behavior and other psychopathology, less stable employment histories, and more severe physical symptoms (Goodwin et al. 1980). Another approach to the classification of alcoholics is based on certain indicators known to predict treatment outcome. One study (Gibbs 1981) classified alcoholics on the dimensions of social stability and intellectual functioning and showed that mutually exclusive types were often assigned to the same treatment regimen despite their widely different rehabilitation needs.

In an attempt to directly test the clinical implications of differentiating subgroups of alcoholics, several investigators have studied how certain types of patients respond to different treatments. Using a classification system that differentiated alcoholics from a less severe type of behaviorally impaired drinker, Brown and Lyons (1981) found that alcoholics did not benefit from programs having a high medical orientation, while the behaviorally impaired drinkers respond better to treatment having a high psychological rehabilitation orientation. Using a different classification scheme, Finney and Moos (1979) studied the treatment response of alcoholics classified in terms of high or low social competence. Contrary to the findings of Brown and Lyons (1981), no evidence was found to indicate that various treatment programs were differentially effective for different types of patients.

Although evidence supporting treatment matching remains equivocal, there may be important methodological reasons why the matching hypothesis has not been adequately tested. First, despite improvements in the methods and theory of classification, the optimal classification system has yet to be developed. Even though family history, psychiatric disorder, alcoholic symptomatology, and organic brain dysfunction seem to be promising diagnostic considerations, there has been little attempt to integrate information from these disparate levels of analysis into more comprehensive typologies. Second, many matching studies have been designed to detect predicted interactions. In a recent review of the matching literature (Skinner 1981), it was found that evidence supporting the matching hypothesis came primarily from those experimental studies that randomly assigned patients to treatment conditions, while the results of nonexperimental correlational studies tended to be less supportive. Finally, in a number of studies it is possible that the assigned treatments were not sufficiently distinct to produce a differential effect. Thus, while the matching hypothesis still holds promise as an avenue to improved treatment efficacy, the systematic study of patient-treatment interactions will have to await the development of better typologies and improved scientific research methodology.

Emerging Trends and Future Directions

A consensus appears to be developing among clinicians, researchers, and policymakers that treatment of alcoholism is passing through a transitional period during which basic assumptions are being reevaluated and a new approach to treatment efficacy is emerging. The elements of this trend include refinements in the definition of terms, improvements in the technology of diagnosis, a more sophisticated approach to the planning of treatments, and a new awareness of the complexity of evaluation methodology.

The advances noted in this chapter in the areas of conceptualization, assessment, and treatment intervention can only serve to enhance the quality of services available to alcoholic persons. The notion that different degrees of alcohol dependence can be measured, that different types of alcoholic persons can be classified, and that different types of disability can be diagnosed has important implications for treatment and research. Recent reviews of treatment-related priorities conducted by the Institute of Medicine (1980), the Journal of Studies on Alcohol (Keller 1979), and the World Health Organization (Edwards et al. 1981) suggest that these themes will constitute a promising but ambitious agenda for the 1980's. As articulated in the writings of many specialists in the field, a new approach is emerging regarding the ways in which treatment is conceptualized, conducted, and evaluated.

The traditional model, which still dominates much thinking about alcoholism treatment, describes how heterogeneous groups of patients are assigned to multimodal treatment programs. After a period of time, the relative success or failure of treatment is evaluated primarily on the basis of the proportion of patients remaining abstinent, and secondarily by global assessments of functioning in other areas of living. Because both treatment variables and client variables are aggregated in this approach, treatment effects may be obscured when the improvements of some patients are averaged with the lack of improvement or even deterioration of other patients. Even when treatment effects are observed, it

is not clear which parts of the treatment process are responsible. In addition to the conceptual limitations of this model, much of the evaluation research conducted within this tradition is difficult to interpret because patient characteristics differ from one study to another, outcome criteria have not been sufficiently specified, comparison groups have not been included in the research design, and the treatment process has not been adequately described. Furthermore, the posttreatment environment has not been taken into account, and there have been unrealistic expectations about what the treatment will accomplish.

In contrast to the traditional model of treatment, the emerging model stresses the heterogeneous nature of the client population, the need for more specific and efficient treatments, and the importance of maintaining gains after treatment. This model differentiates among alcoholics (e.g., depressed vs. nondepressed) and attempts to match each type with the most appropriate combination and configuration of treatments.

Recognizing the importance of accurate patient descriptions, the American Psychiatric Association has developed systematic criteria for classifying alcoholic patients along a number of dimensions. The technique, a part of DSM III (Diagnostic and Statistical Manual of Mental Disorders) promises to provide more standardized and comprehensive patient diagnoses.

Several methods are currently used to identify alcoholics before they come for treatment. Laboratory tests for biological markers or indicators can be a powerful aid to detecting alcoholism. Current research on the use of biochemical indicators in the early detection of alcoholism suggests that a single specific biochemical marker for alcoholism may be elusive. The combination of GGTP, MCV, and several other tests, however, appears to offer, at relatively low cost, a strong indication of recent excessive alcohol consumption. In addition, a widely used and validated self-administered test, the Michigan Alcoholism Screening Test (MAST), elicits responses to medical, social, and behavioral statements. The MAST is rapid, inexpensive, and relatively accurate.

Studies show that untreated alcoholics and their families are characterized by high users of medical services. Insurance programs, especially HMOs, in recognition of the cost implications have begun to identify alcoholics for increased attention. Preliminary studies suggest that partial hospitalization or outpatient programs may be as effective as inpatient programs for some patients; moreover, the lower costs of the former may lead to increased use.

Treatments continue to rely largely on psychotherapy and behavior therapy including social skills and assertiveness training, self-control training, cognitive restructuring, and aversion therapy, which pairs alcohol with unpleasant stimuli. In addition, group approaches like Alcoholics Anonymous are widespread. Behavior therapy in conjunction with disulfiram (Antabuse) seems to be highly effective in producing abstinence; however, abstinence alone does not imply successful psychosocial adjustment.

Summary

The traditional concept of alcoholism as a unitary disease has been challenged. Over the past decade, researchers and clinicians have come to realize that multiple patterns of alcohol use may result in multiple forms of disability. Accordingly, a new emerging model of treatment stresses the heterogeneous nature of the client population, the need for more specific and efficient treatments, and the importance of maintaining gains after treatment. This model differentiates among alcoholics (e.g., depressed vs. nondepressed) and attempts to match each type with the most appropriate combination and configuration of treatments.

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DRUGS WHICH INTERFERE WITH ALCOHOL CONSUMPTION

DISULFIRAM. Disulfiram, at an adequate level in the body, will interact with ingested alcohol to induce the following symptoms in this general order: flushing, sweating, palpitations, dyspnea, hyperventilation, tachycardia, hypotension, nausea, and vomiting. These events are usually followed by drowsiness with complete recovery generally prevailing after sleep.[1] These reactions are presumably dose dependent, but the minimal amounts of either agent needed to produce the adverse reaction are unclear in the literature. Presumably 250 mg of disulfiram a day, perhaps less, is enough; as little as 7 ml of ethyl alcohol can produce the reaction. Since disulfiram is fat soluble and long acting, patients may still react for as long as seven days after they stop taking disulfiram.

Our experience at McLean Hospital is that 125 mg of disulfiram a day in the a.m. is a satisfactory, relatively trouble-free initial and maintenance level for patients weighing under 170 lbs. Patients on this dose who start to drink report typical disulfiram-alcohol reactions of moderate intensity which are aversive without being catastrophic.

Disulfiram probably acts by blocking the metabolism of alcohol, inhibiting the further oxidation of acetaldehyde. The drug also inhibits the conversion of dopamine to norepinephrine by blocking the enzyme dopamine-β-hydroxylase. This

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[1] It was discovered by mistake. Two Danish scientists took the drug themselves to assess its safety as a vermifuge. While on disulfiram, they went to a cocktail party with disastrous results. They inferred, correctly, that the drug might be useful in preventing alcohol consumption.

second action may explain the paradox that acetaldehyde alone causes a rise in blood pressure. The alcohol-disulfiram interaction may have the opposite (hypotensive) effect because of disulfiram's interference with the synthesis of norepinephrine, a biogenic amine active in elevating blood pressure (Ritchie, 1965). Side effects of the drug, other than the alcohol reaction, include fatigue, lethargy, a metallic taste, impotence, and, rarely, toxic psychosis, perhaps secondary to increased brain dopamine.

Although two good psychologically and psychiatrically oriented reviews exist (Lundwell & Baeckeland, 1971; Mottin, 1973), and although disulfiram has been used in alcoholism for almost 30 years, there is no good compilation of the frequency or severity of the various possible side effects at various maintenance dosage levels. These range from 1 gram to 125 mg per day. Rumor suggests that some alcoholics manage to drink and take disulfiram. In fact, this old drug has almost never been exposed to double blind controlled clinical trial. One Russian study, showing no difference between disulfiram and placebo, is described in Mottin's review. Wallerstein's 1957 study compared disulfiram with conditioned reflex therapy, hypnotherapy, and a control group. After two years 53% of the disulfiram patients were doing well as against about 30% of the patients in the other three treatment groups. Hoff (1953), in a large comparative study, found alcoholic clinic patients who were willing and medically able to take disulfiram did better than patients who refused the drug. The fact that a third group, patients willing to take disulfiram but denied it for medical reasons, did as well as the group who actually received it, makes one su that the drug is only a test of motivation. An inter-

study by Gallant and his associates (Gallant, Bishop, Faulkner, Simpson, Cooper, Lathrop, Brisolara & Bossetta, 1968) compared court enforced disulfiram therapy with court enforced group psychotherapy and a voluntary program (court referral without enforcement). They found no treatment differences. However, they suggest that the study patients were too ill to understand clearly the court's actions and believe an inpatient detoxification phase prior to court assignment to the various treatments might have led to a more valuable study. Over the years a shift has occurred in the practice of disulfiram therapy. Originally, all patients were given a test dose of alcohol early in their disulfiram therapy to acquaint them with the full unpleasantness of the alcohol reaction. This practice has now almost completely stopped. It is unfortunate that the contribution, if any, of this aversive experience to the patient's future abstinence has never been adequately studied.

Recently, Rosenberg's group (Gerrein, Rosenberg, & Manohar, 1973) has taken a lead from methadone maintenance and compared disulfiram therapy given on two schedules--once a week administration by a nurse and twice a week administration by a nurse--with the patients being expected to dose themselves the rest of the time. This was compared to non-disulfiram programs involving once or twice weekly clinic visits. Interestingly, the biweekly disulfiram group did very well in terms of clinic attendance (55% at 6 weeks as opposed to about 25% for the other groups). In addition, 40% of the biweekly disulfiram patients remained abstinent for 8 weeks as opposed to about 10% in the other treatment groups (including once a week disulfiram). A more recent study (Rosenberg, 1974) compared this successful regimen

with chlordiazepoxide, vitamins, and no drug. The results were slightly less favorable, but disulfiram was still superior to either vitamin or no drug treatment and equal to chlordiazepoxide. In this study, disulfiram (which is said to cause depression as a side effect) actually acted to decrease depression and anxiety. Patients who continued to return to the clinic for up to 26 weeks tended to be sober as a group; disulfiram's sobriety rate of 85% was a bit higher than the 75% for the other treatment groups.

Lundwell and Baeckeland (1971), in their review, observe that successful disulfiram patients tend to be older, more socially stable, more highly motivated, better able to form dependent relationships, less depressed, and less likely to have blackouts or sociopathic traits. The problem, of course, is that these same traits may be associated with a better prognosis in chronic alcoholism in the absence of disulfiram.

Despite the limited evidence favoring disulfiram therapy, it seems to have an assured place in the outpatient therapy of chronic alcoholics. The position in favor of its use can be stated as follows: disulfiram is not a cure but affords the patient with a sincere desire to stop drinking support in avoiding an impulsive drink. The rationale for its use is that the patient knows that he has to avoid alcohol at least three days after taking disulfiram or he will experience some or all of the previously mentioned symptoms. In this way, the patient's self-control is increased. He also allows himself an interval of sobriety, with regular use of disulfiram, in which to learn to deal with his life without alcohol. Moreover, taking disulfiram is something positive the patient is doing to help himself. This

self-imposed treatment can increase the patient's self-esteem and encourages his family and friends to regain confidence in him. Many patients also see disulfiram as a "good friend" which they take each morning with their orange juice. This "friend" sees that they stay out of trouble as any good friend would.

Patients on disulfiram should avoid disguised forms of alcohol, such as cold alcohol-laden sauces, fermented vinegar, and certain cough syrups. Absorption of alcohol through skin is minimal and, consequently, after-shave lotions are not contraindicated at the disulfiram dosages previously suggested. Gourmets need not become disheartened as Julia Child, "The French Chef" (1974), has suggested the substitution of "chicken broth, fish stock, and beef stock in fish or meat dishes and orange juice or cider in desserts, along with appropriate spices and herbs in meat dishes and cloves, cinnamon, cardomon, etc., in sweet dishes" for wine. Finally, well-cooked dishes will evaporate off all the alcohol during preparation.

Disulfiram should not be given to organically confused, psychotic, severely depressed, or suicidal patients. The confused or psychotic patient may inadvertently drink alcohol for a variety of reasons (e.g., memory deficits, voices telling him to), while the severely depressed or suicidal individual may use disulfiram in an attempt to kill himself (Jacobsen, 1952). Accordingly, because of the latter, as well as the necessary responsibility for daily or bi-weekly self-administration, the patients chosen for this therapy have to have a reasonably sound personality structure. Of course, "an alcoholic who becomes determined to desert his sobriety will simply stop taking the pills for a few days

until he is safe, or he may even start drinking right through the disulfiram, accepting the frightful resulting symptoms as a kind of punishment in advance for his break, with authorization to go on drinking thereafter with impunity" (Solomon, 1966). Finally many patients are resistant to taking disulfiram, particularly men. They see the drug as taking away the opportunity to "do it on their own" and in that sense undermining their masculinity. In term "crutch" is often used, suggesting that their having to take disulfiram infers that they are crippled or at best unreliable. More suspicious patients are concerned that the drug will control them or place them under the physician's control. Education is directed at helping the patients recognize that disulfiram will support their own self-control as the result of their own decision to take it. Low doses (125 to 250 mg a day) are usually clinically useful and avoid almost all side effects other than occasional transient skin rashes, dizziness, headache, or gastric irritability. Most doctors feel disulfiram is contraindicated during pregnancy because of possible danger to the fetus, and in patients with psychosis, and even heart disease (due to its B-hydroxylase inhibiting properties); (Kissin & Gross, 1968). Moreover, if a patient is taking diphenylhydantoin, the dose must be decreased or toxicity may result since disulfiram slows its metabolism.

It is, however, always possible that, in the social context described, a placebo which was believed by both parties to be disulfiram would do as well. It is unclear to what extent actual disulfiram-alcohol reactions play a significant role in the long-term success of disulfiram in some patients.

DISULFIRAM IMPLANTS. A long acting disulfiram implant which may possibly last as long as six months has been developed and is in clinical trial. The most positive and only comparative published study (Whyte & O'Brien, 1974) compares outcome in 22 "implanted" male alcoholics with 23 "matched controls," apparently not randomly assigned to nonimplant therapy. The treatment group averaged 5.4 months until their first drink as opposed to 1.9 months in the comparison group. In terms of the implant itself, the eight tablets formed a sore, inflamed nodule which was uncomfortable for the first week; the operation was done under local anesthetic; no implants were rejected in the series. Local infection or rejection of the tablets occurred in 13 out of 70 implants in the series described by Malcolm and Madden (1973). These patients were, as a group, significantly longer abstinent than had been the case at any time in the two years prior to implantation. Nine patients drank and had a reaction in the first six months after implantation, but only four of these reactions, were judged typical of disulfiram-alcohol reaction and twelve of the 46 patients increased their use of sedatives.

In a South African study of 19 patients (Obholzer, 1974) over half the implants were complicated by infection. Four out of six patients began drinking within seven months of the implant, and only two experienced the expected adverse reaction. There is more extensive data in the Polish literature, which is summarized in the above article.

In summation, it is still too early to assess the ability of disulfiram implants to actually induce reliable alcohol reactions for any fixed duration of time. If they do, it is unclear whether they will be of use in other than specially selected and motivated patients. The potential

medico-legal problems attached to the use of this agent are bothersome.

CITRATED CALCIUM CYANAMIDE. The literature on this shorter acting disulfiram-like drug is well reviewed by Mottin (1973). Its alcohol reactions are usually considered to be generally milder than those occurring with disulfiram, and the alcohol effect is no greater two hours after a single dose than two hours after the seventh of seven daily 100 mg doses (Lader, 1966). In the only comparative study (Levy, Livingston, & Collins, 1967, calcium carbimide (the generic name) was compared to disulfiram. During alcohol challenges in patients, abnormal electrocardiograms were less common when on carbimide (4 of 19) than on disulfiram (3 of 7). Seven of the 19 calcium carbimide patients did well as against an unknown number (less than 5) of the 11 disulfiram patients. It appears that the carbimide patients were local alcoholics; whereas, disulfiram was given to patients likely to leave the area. Hence conclusions about the real relative efficacy of the two drugs cannot be drawn. The drug is marketed in England and Canada but not in the United States.

METRONIDAZOLE. This drug, marketed for use in Trichomonas infections, achieved a certain notoriety after Taylor (1964) reported that males taking the drug reported a distaste for alcohol and a reduction in drinking. This led to a surprising number of studies (9 controlled) which have been well reviewed by Mottin (1973). Although three studies were positive, most were depressingly negative or showed only a small subsample of patients with the metronidazole effect -- i.e., alcohol tastes different, alcohol is disliked, intoxication occurs on lesser amounts. All in all, the effects were generally

small or nonexistent. Thus, the ability of the drug to actually produce disulfiram-like effects when combined with alcohol is dubious, with Gelder and Edwards study (1968) testing this most directly. Unfortunately, the positive placebo-controlled studies were in obscure languages and not available to the present reviewers. In an interesting side study, Wilson, O'Brien, and MacAirt (1973) showed that metronidazole at 800 mg a day (but not at 600 mg) was able to raise taste thresholds for alcohol for both aversive and pleasurable (e.g., sweet) thresholds. (800 mg is higher than the daily dose 500-750 mg used in most controlled studies).

The intriguing aspect of the metronidazole story is that this weak drug has inspired such a range of investigative activity while disulfiram has remained almost unstudied by identical controlled trial methodology.

DRUGS AS AVERSIVE STIMULI

There is modest literature on the aversion conditioning treatment of chronic alcoholism, which is well reviewed by Costello (1969) and less clearly reviewed by Mottin (1973). The general principle, of course, is to associate a highly unpleasant stimulus (the unconditioned stimulus--UCS) with an alcoholic beverage (the conditioned stimulus--CS). This is usually done in a Pavlovian rather than an operant conditioning paradigm in the hope that the unpleasant effects will be associated with the alcohol and lead the patient to avoid, or be upset by, alcohol in the future. In past decades, drugs causing vomiting were generally used--emetine or apomorphine. Here there was a problem in timing since,

ideally, the UCS and CS should be placed only seconds apart for maximal effect. A complex emetine regimen has been used clinically for years at the Schick-Shadel Hospital in Seattle and is well detailed by Costello (1969). No controlled studies of its efficacy are available although Lemere of Schick-Shadel Hospital reports excellent results (60% abstinent for at least one year). One wonders whether going to a hospital for enforced vomiting may not imply strong motivation to stop drinking and whether being able to afford a private sanatorium may not be associated with above average social stability. Both are predictors of good outcome in alcoholism independent of the specific treatment used.

In an effort to develop even more drastic and closely timed aversive stimuli, succinylcholine and other drugs which cause total, brief paralysis of all muscles (as well as severe anxiety) have been tried. Here, a pair of controlled studies have been done. In both studies, patients not receiving both the UCS and the CS have done about as well as patients receiving the full pairing of stimuli.

Overall, the results of these therapies are not impressive. Neither approach is free from danger. One hopes that other nonpharmacological behavioral methods using more active operant procedures with provision for positive reinforcing alternative behavioral outlets would be more useful.

PSYCHOTOMIMETIC DRUGS

Early reports from hospitals in Saskatchewan, Palo Alto, and Spring Grove, Maryland, led to a presumption that the administration of lysergic acid diethylamide (LSD) in a

context of psychotherapy resulted in remarkable changes in drinking behavior of a favorable sort (Cole & Katz, 1964). The Josiah Macy, Jr., Foundation Conference on "The Use of LSD in Psychotherapy" (Abramson, 1960) documents the flavor of that exciting and controversial time.

The early claims have now been tested in at least five well-designed controlled trials. In the most elaborate of these, Ludwig and Levine, who earlier had found short-term favorable effects of LSD therapy in institutionalized drug addicts, compared hypnodelic therapy (an LSD experience guided by hypnosis), psychedelic therapy (LSD and psychotherapy during the experience), LSD without psychiatric intervention and, as a no-treatment control, an equivalent period of quiet solitary contemplation of personal problems by the patient. Throughout a year of detailed and successful follow-up studies of the 44 patients in each treatment group, no differences were found on any of a range of drinking, social adjustment, or subjective state measures between any of the four groups of chronic alcoholics (Ludwig, Levine, Stark, & Lazar, 1969). Half the patients were randomly placed on disulfiram. This also had no effect on any of the measures used.

Hollister, Shelton, and Krieger (1969) compared 600 micrograms LSD with 60 milligrams dextroamphetamine in 72 alcoholics. The experience was intended as a several hour period of self-examination. At two months the LSD group was somewhat better off, but at six months the drinking and social status of the two groups were identical.

Johnson (1970) compared about 550 micrograms LSD intravenously with psychiatric psychodynamic interviewing; a similar period after LSD with attention from a nurse; a

psychiatric interview after intravenous administration of sodium amobarbital and methamphentamine; and routine care. There were approximately 25 patients per treatment group. After one year, there were no differences in outcome between the four groups.

Smart, Storm, Baker, and Solursh (1966) compared 800 micrograms with 60 milligrams of ephedrine and a no-drug session control group (10 subjects per group). Interviews with the drug patients emphasized dynamic insight. The two drug groups showed improvements in abstinence over the 6 month period (34% and 32%) while the no-drug group showed 20% improvement. Overall, no significant differences were found between groups on any of several outcome measures.

Only the Spring Grove group recorded favorable results, but failed to use a placebo or no treatment control. In comparing high dose LSD with low dose LSD or comparing patients who had or failed to have intense transcendental LSD experiences independent of dose (Pahnke, Kurland, Unger, Savage, & Grof, 1970), they observed somewhat better social and drinking behaviors after one year in the high dose and the intense peak experience groups. However, this treatment program involved 12-15 hours of counseling prior to the LSD experience, an eight-hour elaborately programmed LSD experience with constant presence of a therapist plus extensive counseling after the session to consolidate and digest insights and experience.

In sum, LSD therapy for alcoholism is generally ineffective and, at best, is only partially effective even when elaborate, intensive, and expensive psychotherapy is interwoven into the experience. Other hallucinogens have been studied in preliminary trials in alcoholism with little in

the way of substantial findings worthy of detailed review (Denson & Sydiaha, 1970; Grof, Soskin, Richards, & Kurland, 1973; Simonopoulos, Pinto, Babikow, Kurland, & Savage, 1970).

CONDLUSIONS

The available clinical literature assessing drug therapies in various phases of alcoholism has its limitations and is often either inconclusive or controversial. Nevertheless, some general assertions are possible:

1. In the treatment of withdrawal symptoms chlordiazepoxide (and probably other benzodiazepines) is safe and effective and can abort or avert delirium tremens.

2. Promazine, in patients with severe withdrawal symptoms or delirium tremens, is contraindicated. It appears to increase the occurrence of full delirium tremens and has been associated with a higher death rate than any other treatment studied

3. No antianxiety or antidepressant drug (or lithium carbonate) has been shown to offer any clear advantage over placebo in the treatment of detoxified alcoholics in the community. The one available study on lithium carbonate in alcoholics with associated depression is promising and deserves replication.

4. Although disulfiram is widely used and popular with both doctors and patients and probably is useful in selected patients, studies comparing it with placebo or no drug treatment generally show it to have little or no overall clinical value. Nevertheless, it appears to have a firm place in the therapy of chronic alcoholism. The newer variants, calcium carbimide and metronidazole, are equally ineffective and lack

the virtue of wide clinical exposure. Disulfiram implants also seem to offer no advantage over oral medication and may not, in fact, really provide their promised chronic anti-alcohol effect.

5. Aversion techniques -- using either emetics or paralyzing agents -- appear crude and unvalidated and may merely be tests of motivation.

6. LSD and related psychotomimetics used as adjuncts to psychotherapy of alcoholics have been well studied and are generally without any prolonged effect on drinking behavior.

COMMON PROPRIETARY NAMES OF DRUGS MENTIONED

amitriptyline hydrochloride	Elavil (U.S.), Tryptizol (U.K.) Laroxyl (U.K.)
chlordiazepoxide hydrochloride	Librium
chlorpromazine hydrochloride	Thorazine (U.S.), Largactil (U.K.)
chlorprothixene	Taractan
citrated calcium carbimide (cyanamide)	Abstem (U.K.)
diazepam	Valium
diethylpropion hydrochloride	Tenuate (U.S. & U.K.), Apisate (U.K.)
diphenylhydantoin, sodium	Dilantin (U.S.), Epanutin (U.K.)
disulfiram	Antabuse
dixyrazine	Esucos (Belgium), not marketed in U.S.
emylcamate	Nuncital (Sweden) not at present marketed in U.S. or U.K.
haloperidol	Haldol (U.S. & U.K.) Serenace (U.K.)
hydroxyzine hydrochloride	Atarax
hydroxyzine pamoate	Vistaril

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REVIEW ARTICLE

Early identification of alcohol abuse: Critical issues and psychosocial indicators for a composite index*

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Traditional approaches to the medical management of alcohol-related disorders have met with limited success in altering the prevalence of alcohol abuse. Evidence suggests that identifying early those who drink to excess and intervening with low-cost educational and motivational programs could significantly reduce the prevalence of alcohol-related disabilities. However, physicians must take systematic steps to detect alcohol abuse. Part 1 of this two-part series discusses the need for early identification of individuals who drink to excess and the factors that may either facilitate or hinder the development of effective programs for detecting alcohol abuse.

A profile is given of important psychosocial indicators of alcohol abuse, including the classic signs of alcohol abuse, the early manifestations of heavy drinking, the predisposing or high-risk factors for alcohol abuse, and the precipitating events and correlated habits of excessive drinking.

L'approche traditionnelle au traitement medical des problemes relies a l'alcoolisme n'a su modifié avec beaucoup de succès la prévalence de l'abus de l'alcool. Les donnees actuelles indiquent que l'identification precoce de ceux qui boivent avec exces et qu'une intervention à l'aide de programmes éducatifs et de motivation peu coûteux pourraient réduire significativement la prévalence des incapacités dues à l'alcoolisme. Cependant les médecins doivent adopter une démarche systématique afin de deceler l'abus de l'alcool. La premiere partie de cette série à deux volets discute de la necessite d'une identification precoce des individus qui boivent excessivement et des facteurs qui peuvent soit faciliter ou nuire au developpement de programmes efficaces pour deceler l'abus de l'alcool.

On presente un profil des indicateurs psychosociaux importants de l'abus de l'alcool, y compris les signes classiques de l'abus de l'alcool, les manifestations précoces de l'éthylisme, les facteurs prédisposants à l'abus de l'alcool, et les évenements precipitants ainsi que les habitudes menant à une consommation excessive d'alcool.

A new patient walks into your office. After a few introductory comments the 40-year-old man describes his nausea, diarrhea, heartburn and indigestion. During the course of doing a routine physical examination and taking a medical history you detect several interesting items. The examination reveals evidence of hypertension and periorbital edema. You note several scars on the chest and limbs, and tobacco stains on the left hand. Other than treatment for a peptic ulcer 3 years ago, the patient states that he has had no other medical problems in the past 5 years. However, further questioning about the body scars reveals that he has been to the emergency department of a local hospital on three occasions for the treatment of accidental injuries that necessitated leaves of absence from work. In response to further questions the patient states that he has had difficulty sleeping and is troubled by growing stresses at work. You sit back in your chair for a few moments and attempt to synthesize this array of clinical information. Could excessive drinking be a key determinant of his physical complaints?

As there is growing evidence that alcohol-related disabilities are a major cause for medical consultation, there is reason to presume that your patient may indeed have a drinking problem. Along with cancer, cardiovascular disorders and mental illness, alcohol abuse is a major public health concern.[1] However, at the clinical level, evidence has accumulated that physicians are experiencing a "tip of the iceberg" phenomenon. Studies on the prevalence of alcohol abuse in hospital and private practice populations indicate that many patients have drinking problems that are unrecognized.[2,3] A study at the Massachusetts General Hospital,

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*Part 2 of this review, which considers the clinical and laboratory indicators for a composite index of alcohol abuse, will be published in the May 15, 1981 issue of the Journal.

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Reproduced on the cover of this issue of the Journal is "Gin Lane", an eloquent engraving by William Hogarth (1697–1764) depicting results of the abuse of gin by the working class of 18th-century London. The slide was kindly supplied by the Addiction Research Foundation of Ontario through Dr. Mary Jane Ashley.

Boston, found that the chief medical officers failed to detect alcohol abuse in approximately half of the alcoholics in the emergency ward.[12] Rubington[7] has described the situation as analogous to a game of "hide and seek" in which both the hiders (the alcoholics) and the seekers (the physicians) participate either actively or passively. Research suggests that treatment is most successful in patients who have not reached an advanced stage of alcohol abuse and are not yet suffering its many medical and social sequelae.[13] This underscores the need for clinicians to employ identification procedures that can lead to early intervention and an improved prognosis.

Part 1 of this review has several aims. The need is raised for physicians to take more steps to detect alcohol abuse. This need is buttressed by the staggering cost of medical services attributable to alcohol-related disabilities, and by the tendency for some patients to minimize or even deny a drinking problem.

Next, issues relevant to the development of screening programs for alcohol abuse are discussed. Can physicians rely solely on biomedical abnormalities to detect excessive drinking, or must social and psychologic variables also be considered for accurate early diagnosis? More fundamentally, should busy physicians concern themselves with detecting alcohol abuse when their ability to treat and significantly alter the natural course of the disorder is in question?

Then a profile is given of important psychosocial indicators of excessive drinking, including the predisposing factors, likely precipitating events and early behavioural manifestations.

The second part of this review discusses the clinical signs and symptoms and the laboratory findings that are related to excessive alcohol consumption, and describes the morbidity profile at various stages in the development of alcohol abuse. The objectives of these papers are to provide the physician with an up-to-date review of alcohol-related disabilities, to illuminate key indicators of alcohol abuse that should routinely be considered when assessing hospital and private practice patients, and to point out some of the more promising directions of current research into the identification of alcohol abuse.

Early identification

Why it's so important

The economic costs of alcohol abuse are substantial. In the United States the National Institute of Alcohol Abuse and Alcoholism (NIAAA) estimated that alcohol-related problems cost nearly $43 billion in 1975 as a result of lost production, motor vehicle accidents, crime, social problems and demands for health care services.[14] Indeed, over 12% of the total expenditure on health care for adults ($13 billion) was for alcohol-related medical services. Closer to home, the Addiction Research Foundation of Ontario estimated that in the province during 1976 additional medical treatment due to alcohol-related problems cost $320 million, approximately 11% of the total cost of medical services in Ontario for that year.[15]

The mortality associated with alcohol abuse is well documented.[16] In 1975 the NIAAA estimated that 11% of all deaths were alcohol-related.[14] Ashley[17] reported that the risk of death for those abusing alcohol or drinking heavily was two to four times that for the general population, and that hazardous drinking accounted for a 10% loss of potential years of life. The mortality from cirrhosis of the liver is accepted as one of the best indicators of the prevalence of alcohol-related health problems. This disorder ranked as the sixth most common cause of death in the United States in 1975, and up to 95% of those deaths were thought to be alcohol related.[14] In Ontario from 1950 to 1973 cirrhosis of the liver was the most rapidly increasing cause of death in the adult population, followed by lung cancer and suicide.[18] Furthermore, the increase was greatest among persons in the productive age range of 35 to 49 years. These data firmly establish alcohol abuse as a major health concern.

Hindering factors

The importance of identifying alcoholism early has been recognized for over a century. In 1867 Day,[19] the first superintendent of the Washington Home for Fallen Men, indicated that there was a general lack of appreciation of the magnitude of the problem presented by alcoholism, and referred to what we would call the "hidden alcoholic". Earlier Marcet,[20] a physician at the Westminster Hospital, London, England made one of the first systematic studies of alcohol as a predisposing cause of a variety of diseases. Emphasizing that patients may attempt to conceal their drinking habits, Marcet stated: "On first applying to his medical adviser, the patient will probably not state the cause of his illness, and thus seriously mislead the physician in his estimation of the nature of the complaint." More recent studies have confirmed that the attitude of those who abuse alcohol has not changed.[4] Moreover, many physicians are not trained to recognize symptoms of excessive alcohol use or do not consider it their duty to detect alcoholics or both.[14,21]

Clinicians frequently use data from reports on alcohol consumption made by the patient as a basis for diagnosing alcohol abuse. However, physicians' lack of standardized methods for eliciting and recording such information and sometimes their lack of awareness of the many factors that influence the reliability and validity of these reports lead to problems in data collection. These factors, combined with the patient's motivation and ability to minimize drinking problems, may lead to inaccuracies in diagnosis.

Studies on the reliability and validity of patients' reports of alcohol consumption have produced conflicting results.[22-25] Generalizations are difficult to make since the accuracy of these reports depends upon the context in which they are obtained, the patients' characteristics and the types of behaviour being assessed. Armor and colleagues[26] concluded that patients' estimates of frequency of drinking are quite satisfactory, but that es-

timates of the actual quantity of alcohol consumed, while moderately reliable for nonalcoholics, are low for alcoholics. Hence, patients may be fairly consistent when describing how often they drink, but less accurate about how much they drink. Furthermore, Sobell and coworkers[24] underscored the need for ensuring that patients are sober, as verified by the blood alcohol level, when a history of drinking habits is examined. They found that alcoholics' reports of recent alcohol consumption were often invalid, consumption being underreported, when the patients had been drinking.

An important extension of this observation concerns how much credence the physician should give to a patient's complaints when the patient has been drinking. This issue is especially relevant in an emergency department, where some patients may have been drinking or may even be intoxicated. For instance, a recent study found that 32% of the patients admitted to the emergency department of a teaching hospital in Edinburgh, Scotland had a blood alcohol level exceeding 17.4 mmol·l (80 mg/dl).[25]

Diagnostic instruments presently available

Instruments for the diagnosis of alcoholism that incorporate medical data include the National Council on Alcoholism (NCA) criteria,[26] the Michigan Alcoholism Screening Test (MAST)[27] and the Munich Alcoholism Test (MALT).[28] The NCA criteria provide a reasonably comprehensive list of the main physical, social and psychologic sequelae of alcoholism. However, many of these criteria may be redundant for identifying the alcoholic patient since the predictive ability of a number of them has not been fully established; in one study there was no significant difference between alcoholic and control patients according to 38 of 86 of the NCA criteria.[29]

The MAST is a widely used instrument containing 25 items that refer to the medical, social, intrapersonal and legal consequences of problem drinking;[27] the total score classifies patients along a continuum according to the degree of alcohol misuse. The test can be rapidly completed by interview or by the patients themselves, and encouraging data have been reported on its reliability and validity.[27] In one clinical population of alcohol abusers the MAST yielded scores that were relatively unaffected by any tendency the patients might have to deny problem areas.[30]

By including objective data such as clinical signs and laboratory findings that indicate the presence of alcohol-related diseases, it may be possible to corroborate interview and self-reported data, and thus to obtain a more accurate assessment of alcohol abuse. This approach was used by Feuerlein and associates[28] to develop the MALT, a test that contains two sections: part A is to be completed by the clinician, and part B, which contains 24 items pertaining to alcohol abuse and its adverse social and somatic effects, is to be completed by the patient. Although the MALT has produced encouraging results, it seems that the medical items contained in part A are sensitive only to disorders that develop in the later stages of alcohol abuse.[31] Nevertheless, this test is a good prototype of a short test that combines medical and psychosocial indicators of alcohol abuse.

Issues that must be addressed

The economic, social and health costs associated with alcohol abuse provide powerful incentives for early identification and treatment. To date, most efforts have concentrated on tertiary care for persons with relatively advanced medical and social disorders, here rehabilitation is the goal. Considerably less attention has been directed toward secondary care, which involves detecting alcohol-related disabilities early and intervening at a stage when the individual's drinking behaviour should be more amenable to treatment. Available research indicates a more favourable prognosis for individuals who are socially stable and have not yet accrued adverse medical or social consequences of their drinking.[32] In an editorial published in 1978 Lieber[33] called for a major public health strategy against alcoholism. A basic issue is whether early identification will lead to an improvement in the outcome of treatment and a concomitant reduction in the overall costs related to alcohol abuse. To answer this fundamental question four issues must be addressed.

Lack of precise definitions: Consensus is lacking on definitions for the disorders that need to be identified. Most existing criteria for alcohol abuse, such as those of the NCA,[26] are relevant primarily to the later stages of the disorder, which require tertiary care. More recent recommendations for diagnostic classes suggest including the alcohol dependence syndrome and alcohol-related disabilities, as proposed by the World Health Organization (WHO),[34] as well as distinguishing between alcohol abuse and alcohol dependence, as advocated by the American Psychiatric Association.[35] Although both systems represent needed steps in the standardization of terminology, they have not adequately taken into account the indicators and problems related to the early stages of excessive drinking. There is a conspicuous neglect of the middle ground between social drinking and chronic alcoholism.

Undoubtedly one explanation for the lack of precise definitions is the complexity of disorders that are influenced either directly or indirectly by alcohol abuse. The traditional concept of alcoholism as a single specific disorder has failed to adequately represent the diverse and multifaceted problems related to drinking, and a multiple-syndrome concept is gaining ascendancy.[36-40] However, considerable work is needed to refine the definitions of these alcohol-related syndromes. Our approach, consistent with the WHO definitions,[36] is to view alcohol abuse as lying along a continuum of severity; that is, it exists in degrees and may be manifest in multiple syndromes or alcohol-related disabilities.

Questionable effectiveness of current interventions: An axiom of preventive medicine is that it is inappropriate to detect a disease for which effective treatment is lacking. This raises a serious question. Do we at this time have interventions

that can significantly alter the course of alcohol abuse? The available evidence is far from convincing. A consistent finding from research on the treatment of alcohol abuse is that patient characteristics have a greater effect on the outcome than the kind of treatment given. In their exhaustive review Baekeland and collaborators[41] concluded that "over and over we were impressed with the dominant role the patient, as opposed to the kind of treatment used on him, played in his persistence in treatment and in his eventual outcome." The different forms of treatment seem to produce the same relative level of success with individuals who have advanced to the later stages of alcohol abuse.

If we lack interventions that are powerful enough to alter the course of alcohol abuse, then the early identification of cases may yield meagre results. Fineberg and Hiatt[42] described how a 10-fold increase in the use of radionuclide scanning for brain tumours over the past decade has shortened the interval between the onset of symptoms and surgical treatment from 4 years to 1. Clearly this was a significant improvement in the early detection of tumours, yet survival after the operation remained unchanged: during a decade less than 50% of the patients who underwent an operation each year survived for 2 years.

Moreover, the mere identification or labelling of patients can produce deleterious effects. This problem was illustrated quite dramatically by a study of hypertension in an industrial setting. Haynes and colleagues[43] found that the labelling of patients as hypertensive resulted in increased rates of absence from work: an 80% increase in absenteeism was found in the identified group, compared with a 9% increase in the general employee population during the study period. Similarly, numerous critics have voiced concern over labelling and self-fulfilling prophecies, particularly for psychiatric disorders.[44,45]

Need for innovative approaches to treatment: If valid procedures for the early detection of alcohol abuse were devised, there is a danger that those identified could swamp existing treatment resources. Indeed, Plaut[46] estimated that the treatment of every "alcoholic" in California would require the full-time service of every psychiatrist and social worker in the United States. However, the intensity of present treatment methods, which are aimed primarily at rehabilitation, may be unnecessary for helping those at an early stage of alcohol abuse. There are indications that a lower-cost intervention, consisting of assessment, brief counselling and follow-up, can yield results that are comparable to those of traditional inpatient and outpatient programs for alcohol abuse.[47,48] This basic intervention, summarized in Table I, could be readily adapted to private practices and general hospitals. Although further clinical investigation is needed, it appears that a brief intervention by physicians in the earlier stages of excessive drinking could have the widespread impact of curtailing the prevalence of alcohol-related disabilities.

The impact of a brief intervention was clearly evident in a study of the effects of advice against smoking from general practitioners. Russell and coworkers[49] assigned 2138 cigarette smokers consulting 28 general practitioners to one of four groups: nonintervention control subjects; questionnaire-only control subjects; subjects advised to stop smoking; and subjects advised to stop smoking and given a leaflet to help them. Follow-up data collected 1 month and 1 year later revealed a significant difference across the groups. The percentages of patients who had stopped smoking within 1 month and remained abstinent for 1 year were 0.3%, 1.6%, 3.3% and 5.1% respectively. The Russell team estimated that if all the general practitioners in the United Kingdom participated, the brief advice plus leaflet intervention would yield over half a million ex-smokers a year. Indeed, this success rate could not be matched by increasing the number of specialized smoking-withdrawal clinics in England from the present 50 to over 10 000. Thus, this study dramatically underscores the potential impact of collective efforts by physicians in general practice.

In addition to low-cost clinical interventions, another approach is a large-scale prevention program, like the heart disease prevention program of Stanford University in Palo Alto, California.[50] In this study, involving three communities, intensive instruction given to individuals identified as being at high risk for heart disease significantly reduced such physiologic indices of risk as blood pressure, relative weight and serum cholesterol concentration. This finding suggests that mass media educational campaigns directed at entire communities can be effective in reducing the risk of cardiovascular disease. Although a similar program may prove successful in reducing the prevalence of alcohol abuse, especially for individuals identified as being at risk, research to date in-

Table I — Basic treatment strategy for alcoholism[47]

Comprehensive assessment
Record medical history, current status and physical disabilities related to alcohol use
Obtain an alcohol and drug use history
Describe psychosocial problems related to drinking
Estimate level of dependence on alcohol
Single counselling session
Review assessment findings with the patient and the immediate family
Emphasize responsibility of the patient and the family for working out goals and dealing with problems
Set goals with respect to alcohol use (i.e., abstinence v. controlled drinking)
Set goals in other key areas (e.g., personal health, work, leisure, finances, family relationships)
Provide educational material on alcohol use, self-monitoring of drinking and other relevant subjects
Follow-up
Contact the patient and the immediate family regularly (by telephone or letter, or in person)
Monitor progress in goal attainment

dicates that influencing patients' attitudes toward alcohol use will not necessarily change their behaviour to healthier patterns.[51]

Purposes of identification. The early detection of disease has generated much controversy. A real danger is that unproven or marginally effective treatments will be employed merely because patients have been identified. The absolute necessity for long-term evaluation of interventions has been repeatedly stressed. For example, Favus and associates[52] found that thyroid cancer occurred as a late consequence of radiotherapy for benign childhood conditions of the head and neck. There is ample justification in the history of medicine for Bergman's words of caution:[53] "A special section in my personal demonology of the health field is reserved for the perpetrators of mass screening. With some gadget or laboratory test to peddle, they fall upon their prey with self-righteous zeal, selling promises of prevention or cures like indulgences to heaven."

Sackett and Holland[54] have contended that much of the controversy in the detection of disease arises from inconsistent definitions and a lack of understanding of the different purposes of detection. *Epidemiologic surveys* involve the assessment of a carefully selected sample of a population to obtain new knowledge; no health benefit to the participants is implied. The aim of *screening*, on the other hand, is to detect groups at high risk for a disorder that is more effectively treated at an early stage. Here the implicit promise is that volunteers will receive treatments of proven efficacy. Sackett and Holland argued that "far greater certainty of efficacy is required when recommending treatment at the community level, especially when patients are solicited through screening". In comparison, *case-finding* is initiated by a patient seeking help from a physician; a comprehensive health assessment may reveal a disorder that was unrelated to the patient's initial complaints. Whereas case-finding may seem optional to the busy clinician, *diagnosis* is mandatory and entails establishing the exact cause of the patient's symptoms. With both case-finding and diagnosis, emphasis is placed on the accuracy and predictive value of the identification procedure. Since a given test can often be used with any of these methods of detecting disease, one must be clear about the exact purpose of the test when assessing its value as a detection instrument.

In summary. We have surveyed the challenging problems and potential benefits of the early detection of alcohol abuse. It is premature to judge the value of early detection, but the premise that it will give rise to a better outcome of treatment is attractive, especially with the concern over the increasing costs of health care. However, a comprehensive program of research is needed to provide a rigorous test of this premise. A vital first step in this research is the development of valid procedures for early identification.

Guiding principles for early identification programs

Integrate biomedical and psychosocial factors. There is growing evidence that different indicators are more sensitive to certain stages of alcohol abuse. For example, in a study of predominantly young men referred to a medical officer because of problems related to suspected alcohol abuse, most of the men acknowledged their psychosocial problems, but few had clinical or laboratory manifestations of diseases associated with chronic alcohol abuse.[55] Many clinical disorders, such as cirrhosis of the liver, are evident only in later stages of alcoholism, and an individual may have been drinking excessively for 5 to 10 years before they develop.[56] Furthermore, although over 50% of deaths from cirrhosis of the liver are linked to alcohol misuse, only a small proportion (around 8%) of those who abuse alcohol have this disease at a given time.[56] Thus, despite the fact that data on mortality due to cirrhosis provide a good estimate of the prevalence of alcohol-related health problems in a population, the signs and symptoms of cirrhosis are not helpful for early clinical identification.

Aside from the later onset of many clinical disorders related to alcohol abuse, a number of clinical abnormalities are time-specific. For instance, the levels of serum transaminases may fall to normal with abstinence or a substantial reduction in alcohol consumption.[46,57] On the other hand, psychosocial problems, such as being arrested for driving while intoxicated or being reprimanded for drinking on the job, span a longer time-frame and are not reversible since the event either did or did not occur. In general, research suggests that certain psychosocial items and laboratory findings will be more relevant in younger individuals who began excessive drinking early.[58] More clinical abnormalities in addition to social and psychologic problems could be expected in older individuals.

Fig. 1 depicts a general sequence of disorders in relation to the duration of excessive drinking. The usual focus of medical interest is on the later stages, in which treatment is oriented toward rehabilitation. For these patients diagnosis is often fairly straightforward because of the presence of classic signs of alcohol abuse. As physicians attempt to detect cases at earlier stages of excessive drinking, various disorders are potentially relevant. However, any single item may lack sensitivity to alcohol abuse, since a good proportion of individuals who drink excessively will not have the clinical abnormality or psychosocial problem. On the other hand, a composite picture based on carefully selected items should lead to improvements in early diagnosis. Major challenges for research are to determine which biomedical and psychosocial items are key indicators of alcohol abuse, and to decide how to combine these variables in making diagnostic decisions.

Use composite indices. Whereas no single biomedical or psychosocial item has proven to be highly accurate for early detection, there are indications that carefully chosen composite indices can improve diagnosis. Ryback and collaborators[59] constructed a biochemical profile, including biochemical and hema-

tologic indices from routine laboratory tests, that might alert a physician to the possibility of alcohol abuse in a patient. The blood chemistry tests included sequential multiple analyses (SMA 12 and SMA 6) and the hematologic series (such measures as the mean corpuscular volume). When used together in a multiple discriminant function the 25 laboratory tests correctly classified [illegible] of patients in hospital medical wards because of alcohol abuse, 94% of patients in a treatment program whose condition had deteriorated less and 100% of patients in medical wards for reasons other than alcohol abuse. Although the diagnostic accuracy of this biochemical composite needs to be cross-validated with new samples, this study does illustrate the potential gains from devising composite indices. Another example of this approach in the diagnosis of alcoholism is given by Drum and Jankowski.[28]

An important reason for using composite indices is to capitalize on the different sensitivity of each item to various levels of alcohol abuse. Consider the biochemical test for γ-glutamyl transpeptidase (GGT) and high-density-lipoprotein cholesterol (HDL-C). The serum concentration of GGT is a measure of liver function that appears to reflect heavy drinking (consumption of over 60 g/d of absolute alcohol, or ethanol, in the previous days or weeks).[29] Similarly, the serum HDL-C level appears to show a graded response to moderate consumption of alcohol (40 to 60 g/d of ethanol).[30,31] Although the GGT and HDL-C levels may be raised because of various factors other than alcohol consumption, each test may prove to be differentially sensitive to levels of alcohol consumption, and their composite use may significantly improve diagnostic accuracy. Evidence to support a composite index of GGT and HDL-C has recently been documented.[32] Similarly, Shaw and Lieber[33] found that the combined use of serum levels of GGT and α-aminobutyric acid improved the estimation of level of alcohol consumption.

Composite indices yield a total index score that will, in general, be more reliable than any single item; hence the widespread use of composite scales to assess such measures as the severity of liver disease[34] or the extent of physical dependence on opiates.[35] The MAST reliability estimates were quite low for a single item, moderate for short composites of items and quite high for the total index based on all 24 items.[7]

A basic assumption in our research is that alcohol abuse is likely a steadily increasing (monotonic) function of the number of biomedical and psychosocial abnormalities the patient demonstrates. We are devising an overall index score for identification that may include different items, depending on the patient's age, sex and socioeconomic background. The index score will provide a quantitative estimate of the level of alcohol abuse, consistent with the growing support for a multidimensional diagnostic system that views alcohol abuse as existing in degrees rather than as an all-or-none phenomenon.[36,37,38]

Consider addictive behaviour in general Information on other potentially addictive behaviours, such as smoking, drug use, caffeine consumption, gambling, eating and working, may prove to be key predictors of individuals who are likely to abuse alcohol. Gilbert[39] has argued that the causes of alcohol abuse may be common to other forms of excessive behaviour. Excessive drinking may have as much in common with overeating as it has with social drinking. Certain obese individuals exhibit compulsive eating habits, such as food binges, that are remarkably similar to the actions of episodic or binge drinkers,[40] and there is evidence that alcoholics smoke significantly more than nonalcoholics.[41] Hence, alcohol abuse may be viewed as a particular manifestation of addictive behaviour that may have much in common with other addictions. Thus, as we attempt to advance the level at which we can detect alcohol abuse to more formative stages, consideration should be given to a general model of vulnerability to addictive behaviour.

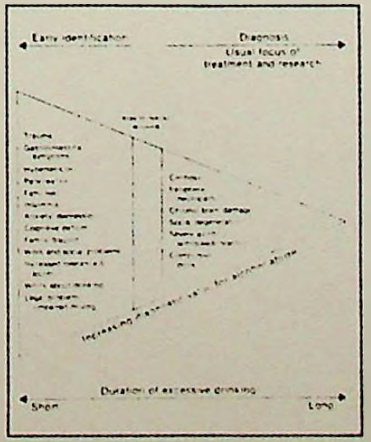

FIG. 1—Temporal sequence of disorders in relation to duration of alcohol abuse.

Psychosocial indicators

Parallel to the diverse profile of physical signs and symptoms associated with excessive drinking (as described in part 2 of this review) a patient's alcohol abuse may be manifested in a wide range of social and psychologic problems. Table II highlights various psychosocial items that surveys of the literature have found to be either classic late signs of alcohol abuse and dependence or early indicators of alcohol misuse. In Table III are listed factors that potentially predispose individuals to drink excessively or increase the likelihood that they will do so, as well as situations that may precipitate patterns of maladaptive drinking, such as stressful life events. The emphasis is on high-risk situations that either trigger the start of excessive drinking or exacerbate an existing pattern of alcohol misuse.

Classic signs

Several of the classic signs listed in Table II have been found to be presumptive evidence of alcoholism.[11] Although the presence of any one sign does not necessarily indicate alcohol dependence in an individual, the probability of alcoholism increases substantially when two or more signs are present. For instance, of the NCA criteria for alcoholism Ringer and coworkers[42] found that only 4 of the 86 (morning drinking, blackout periods, gross tremor and regressive defence

mechanisms) were needed to predict with 90% accuracy whether patients in hospital were alcoholics. Similarly, Costello and Baillargeon[23] found that only two items, morning drinking and blackouts, were needed to achieve good concordance with independent ratings by psychiatrists of alcohol abuse in patients. Morning drinking and attempts to cut down on drinking are [redacted] of the four CAGE* items that [redacted] been used successfully to screen for alcohol abuse.[24] Loss of control after one or two drinks and morning drinking, presumably to relieve or avoid alcohol withdrawal symptoms, have been found to be central factors in alcohol dependence.[25]

Various amounts of alcohol consumed per day have been suggested as defining excessive use. Severe liver damage has been associated with a mean consumption of 12 drinks (Table II) or 160 g or more of ethanol a day. According to Lelbach,[2] histologic examinations revealed the presence of liver cirrhosis in one out of four alcoholics after approximately 12 years of alcohol intake at this level. The Addiction Research Foundation of Ontario has conducted several large-scale surveys within the province. Using a criterion of 120 g of ethanol (nine drinks) a day for alcoholism, Schmidt and de Lint[26] found that approximately 3.5% of the population in Ontario over the legal drinking age drank more than this.

It is well documented that long-term alcohol abuse causes structural and functional impairment of the nervous system.[27] Ryback[28] has suggested that the effect of alcohol on memory is a continuum from specific memory deficits common to cocktail-party drinking, to alcohol amnesia or blackout after an acute prolonged rise in blood alcohol concentration, to the essentially permanent memory deficits of the Wernicke–Korsakoff syndrome. Neuropathologic studies using computer-assisted tomography have revealed cortical atrophy among alcoholics.[29,30] The frontal lobes appear to be damaged more often than other areas of the brain. Psychometric studies have demonstrated that chronic alcohol abuse is particularly detrimental to the patient's ability to solve problems, to manipulate abstract concepts and to perform complex psychomotor tasks.[31] Furthermore, these deficits have been found in subjects whose general intellectual level is average or above average. When the subjects abstained from alcohol some improvement in their performance was demonstrated during the first few weeks.[32]

*An acronym derived from questions that ask if the patient feels a need to *Cut* down on drinking, is *Annoyed* by criticism, feels *Guilty* about drinking and ever has an "*Eye*-opener".

Table II – Psychosocial indicators of alcohol abuse

Classic signs
Very heavy drinking: often nine or more drinks* a day (about 120 g/d of ethanol)
Morning drinking
Blackouts, memory lapses when drinking
Loss of control, craving for more when drinking
Compulsive drinking style, frequent thoughts about drinking
Severe alcohol withdrawal reaction (such as delirium tremens)
Repeated attempts to cut down on drinking have failed
Gross cognitive deficits (such as "alcohol amnesia")
Social degeneration: job loss, family problems, legal convictions related to drinking
Earlier indicators
Heavy drinking, often four or more drinks* a day (about 60 g/d of ethanol)
Increased tolerance to alcohol
Drinking quickly, gulps the first drinks
Eating lightly or skipping meals when drinking
Concern or worry about drinking by self or family or both
Intellectual impairment
Accidents in which alcohol intake is involved
Tardiness or absence from work because of drinking
[redacted]st friends are heavy drinkers
[redacted]st leisure activities involve drinking
Frequent use of alcohol to relieve stress, anxiety, depression
Attempts to cut down on drinking have had limited success

*One drink = 13.6 g of absolute alcohol (ethanol) or approximately 340 ml of Canadian beer, 43 ml of Canadian liquor, 142 ml of wine or 85 ml of sherry, port or vermouth.

Table III—Factors related to alcohol abuse

Predisposing factors
Genetic influences
Family history of alcoholism
Birth order – born later in a large family
Disrupted family background
Early age at onset of regular drinking
Childhood history of minimal brain dysfunction
Personality traits – impulsivity, rebelliousness, low self-esteem
Delinquency
Precipitating or correlated factors
Stressful life events in recent past
Change in peer group
Greater availability of alcohol
Improved financial status
Change to higher-risk occupation
Heavy smoker
Abuse of other drugs

Earlier indicators

One of the most contentious issues in alcohol research is the establishment of a level where "hazardous" alcohol consumption begins. Popham and Schmidt[33] succinctly state: "In short, we do not yet know the limit of safe consumption from a biomedical standpoint." This issue has been further complicated by evidence that moderate alcohol consumption protects against ischemic heart disease.[34,35] However, a level of consumption that may protect against ischemic heart disease may predispose to other medical and psychosocial disorders.[36] Research will give us only probability estimates of threshold values for hazardous levels of consumption, and these estimates will vary for definable subgroups. There is evidence that the hazardous level will vary with age, sex, somatotype,

presence of medical complications, such as liver disease or cardiac problems, pregnancy, drinking style (continual v. intermittent), and neuropsychologic status.[14,15,17,20] This evidence will be considered further in part 2 of this review. Hence, there is no simple answer to the question What is a hazardous level of alcohol consumption?

Guidelines have been suggested, however, for the risk of harmful effects from drinking. On the basis of epidemiologic investigations Thaler[21] has suggested a danger level for damage to the liver of 60 g of ethanol (four drinks) a day for men and 20 g (one and a half drinks) a day for women. Pequignot and collaborators[22] estimated the relative risk of ascitic cirrhosis for different daily levels of alcohol consumption. The likelihood of avoiding ascitic cirrhosis was estimated at 73% for a daily consumption of less than 60 g of ethanol, 83% for less than 40 g and 93% for less than 20 g. Despite the excellent work of Lelbach[23] and Pequignot and collaborators,[22] further research is needed to estimate the relative risk of other medical and psychologic disorders in relation to daily alcohol consumption.

Tolerance to alcohol has been identified as an important factor in the escalation of drinking. In studies on alcoholism, tolerance refers to a decrease in the response to alcohol as a result of frequent drinking; it is established when the effect of a given dose diminishes through frequent drinking, and when an increase in dose will reinstate the effect.[24] However, the scientific evidence is sketchy on whether increased tolerance necessarily alters the probability of further excessive drinking.[25] Cappell and LeBlanc[25] have speculated that tolerance may be most relevant during the formative stages of drinking habits, since individual differences in tolerance may underlie differential adaptation to the noxious effects of alcohol; that is, tolerance may be an important factor in determining which individuals increase their consumption of alcohol to excessive levels.

Another important factor that may signal either incipient or established alcohol abuse is the rate at which alcohol is consumed, especially for the first few drinks. Research has established that the degree of intoxication is greater when there is a rapid peak in the concentration of alcohol in the blood.[24] Impairment is also greater when the concentration is rising than when it is falling, although the actual levels may be identical;[26] this, Mendelson and Mello[26] have argued, "may explain the frequent observation that alcoholics tend to initiate a drinking episode with a large volume of ethanol and also to gulp their drinks". Furthermore, since the gastric emptying rate is a key determinant of the blood alcohol concentration, individuals may eat lightly or skip meals when drinking in order to expedite the rise in blood alcohol level and the development of intoxication.[26]

Perhaps one of the best early indicators of alcohol abuse is a growing concern or worry about personal drinking habits. The recognition of alcohol-related problems by drinkers and their family and friends is a major factor underlying items in the MAST questionnaire.[27] In a study using a different instrument (the MALT) many of the young men who had been referred to a medical officer for suspected alcohol abuse recognized that they had psychosocial problems related to drinking.[28] Indeed, three of the statements on the MALT that best indicate alcohol abuse are: "I think alcohol is destroying my life"; "Since I have started drinking, I have been in worse shape"; and "I think I ought to drink less." These concerns have consistently been identified as significant in studies of methods of assessing alcohol abuse.[29]

Prolonged heavy drinking is related to a number of cognitive deficits; for instance, alcohol impairs the ability to attend to several tasks simultaneously.[30] Also, both alcoholics and nonalcoholics have been found to have considerable cognitive deficits after taking large doses of alcohol.[31] Parker and Noble[31] found that the performance of social drinkers on tests of abstracting and adaptive abilities was negatively associated with the amount of alcohol consumed before the test. Although the relation was strongest for heavy drinkers, deficits were also noted in light and moderate drinkers. In a sample of alcoholic men under the age of 35 years, Lee and associates[32] found that 59% were intellectually impaired, 49% had cerebral atrophy revealed by computer-assisted tomography, but only 19% had cirrhosis of the liver. Since other neurologic complications were considered trivial, Lee's group argued that intellectual impairment may be the earliest complication of chronic alcohol abuse and may arise at an early stage in an individual's excessive drinking. This study also corroborates our finding of a relatively low prevalence of clinical signs and symptoms of alcohol abuse among young problem drinkers.[32]

Alcohol abuse is a factor in various forms of accidents. The NIAAA estimates that half of all traffic fatalities and one third of all traffic injuries are alcohol related; the probability of being involved in a traffic accident increases dramatically as a driver's blood alcohol concentration increases.[33] Furthermore, up to 40% of fatal industrial accidents, 69% of drownings and 83% of fire fatalities are alcohol related.[33] As will be discussed more fully in part 2 of this review, traumatic injuries resulting from accidents are often associated with alcohol abuse. When examining a patient with accidental injuries the physician should routinely consider the possibility that the patient, family members or others involved in the accident were drinking to excess at the time.

Several other items have potential as early indicators of alcohol abuse; for instance, some organizations have found tardiness, absenteeism and lowered productivity among their alcohol-troubled employees.[34] Hence, employment records may provide valuable leads to the presence of drinking problems. An individual's peer group and leisure activities may also be indicative; when most friends are heavy drinkers and most leisure activities involve drinking, the person may be under considerable social pressure to drink heavily. Another

signal of maladaptive drinking that could escalate into chronic alcohol abuse is the frequent use of alcohol to relieve stress, anxiety or depression. As well, if individuals have had only moderate or limited success in trying to alter their drinking habits, their control over their alcohol intake is impaired even though they may be consuming only moderate amounts of alcohol at the [redacted].

Predisposing factors

One of the most challenging areas of research on alcohol abuse is the study of the factors that may predispose individuals to drinking problems or increase the likelihood that such problems will develop (Table III). Recent evidence has indicated that genetic factors are important determinants of the risk of alcohol abuse. Several studies of individuals separated from their biologic relatives soon after birth and raised by unrelated foster parents have found that the risk of alcohol abuse was increased in those with a biologic parent who was alcoholic.[95-107] Goodwin[107] postulated that certain people may have genetically determined physical reactions to alcohol that lower the likelihood that they will be able to drink alcohol in large enough quantities to become alcoholic. Other studies have compared those with a family history of alcohol [redacted] to control subjects and found that such a history influences levels of acetaldehyde in the blood,[108] self-ratings of the degree of alcohol intoxication[109] and the amount of static ataxia or body sway[110] after drinking a given amount.

Aside from potential genetic influences, it is well established that alcohol abuse is much more prevalent in relatives of alcoholics than in relatives of nonalcoholics.[107] In a large sample of alcoholic men, those with a family history of alcohol abuse had more severe symptoms of this abuse, more antisocial behaviour, less social stability and more severe physical disorders than those without alcoholism in their family.[106] Furthermore, the risk of alcohol abuse is increased for those whose families were disrupted during their childhood, perhaps by the separation of their parents or by frequent moves.[111] Also, research has found that children born last in large families have an increased probability of alcohol abuse.[100]

Other research has focused on individual characteristics that are related to the onset of drinking problems. There is some evidence that children with symptoms of minimal brain dysfunction (e.g., hyperactivity, poor control of impulses and poor concentration) begin to consume excessive quantities of alcohol at an early age.[112] Other studies have found that conduct disorders and delinquency in childhood are positively associated with the future development of alcohol abuse.[113] Finally, there has been extensive research on a possible "alcoholic personality".[111] Comparisons of alcoholic patients with those having other disorders have failed, however, to support such a concept. Indeed, research reports have emphasized the heterogeneity of individuals who abuse alcohol, and several distinct subtypes have been identified.[114] On the other hand there is some evidence to support the concept of a prealcoholic personality.[111] Longitudinal studies have suggested that such personality characteristics as impulsivity, poor self-esteem and low ego strength during childhood and early adulthood are related to the subsequent development of alcoholism.[115,116]

Precipitating factors

The final class of variables include factors that can either trigger a pattern of excessive drinking or exacerbate an existing habit (Table III). Research has shown that as the number of recent stressful life events increases — such as a job loss or the death of a spouse — there is a greater likelihood of some physical or mental disorder.[113] The magnitude of the event also tends to increase an individual's general vulnerability to illness. According to Rahkin and Struening,[114] stressful life events do not cause disease but "alter the individual's susceptibility at a particular period of time and thereby serve as a precipitating factor". To date, research has found limited support for a specificity hypothesis — that is, that certain life events (e.g., job changes) are more highly associated with a specific class of disorders (e.g., gastrointestinal disease).[117] Both the number and the magnitude of stressful life events in the recent past may increase the frequency of drinking bouts and the quantity of alcohol consumed.

A related set of variables involve situational changes that increase an individual's access to alcohol. These might include an improvement in financial status, with more money available to spend on alcoholic beverages,[11] a change to a peer group or club that encourages heavy drinking, greater access to alcoholic beverages because of social policy changes,[14,15] and employment in a higher-risk occupation, such as catering.[4]

Alcohol consumption is correlated with smoking[21] and the nonmedical use of drugs.[14] Indeed, the combined use of alcohol and other drugs represents the second most frequent cause of drug-related medical crises.[24] Although no causal relationship is assumed between drinking, smoking and drug abuse, these behaviours are correlated; involvement in one of these habits may predispose to, or exacerbate, other forms of addictive behaviour.

Taken together, Tables II and III describe the classic signs of alcoholism, indicators of the initial stages of excessive drinking, factors that predispose to alcohol abuse in the long run, and factors that may either increase a person's vulnerability to heavy drinking or exacerbate an existing pattern. Consideration of all these factors should provide a fairly comprehensive assessment of a given patient's status with respect to alcohol abuse.

Conclusions

Traditional approaches to the medical management of alcohol-related disorders have met with limited success in reducing the prevalence of alcohol abuse. Evidence suggests that the early detection of problem drinking and the use of low-cost interventions could make significant inroads in preventing the disabilities related to excessive alcohol consumption. Indeed, the po-

tential impact of the use of such an intervention by general practitioners is substantial. To date, the possible cumulative effort by physicians in private practice and in general hospitals has not been fully realized. However, before intervention is possible, physicians must take steps to identify alcohol abuse. Patients will often reveal problems related to drinking when directly asked about their alcohol consumption, but they are not likely to do so spontaneously.[1] On the other hand, there is evidence that certain physicians avoid confronting patients who misuse alcohol and may take action only after the patient requests help.

Instruments for assessing alcohol abuse, such as the MAST,[2] are oriented toward the later stages of alcohol abuse, when there are notable medical and psychosocial disorders. Instruments that are sensitive to the earlier stages are urgently needed. Pending further research, we recommend assessing a patient with the various indicators listed in Tables II and III of this paper and in the tables of part 2 of our review. A working hypothesis could be that the larger the number of abnormalities revealed by such an assessment, the higher the likelihood of alcohol abuse. Current research is aimed at establishing more precise diagnostic strategies.

Members of the medical profession are perhaps the best position to detect alcohol abuse at an early stage, and to intervene with guidance and treatment. However, alcohol consumption and misuse are governed by complex interactions among such variables as socioenvironmental factors, individual differences in constitution and personality, and the availability of alcohol. This diversity challenges health care personnel to be concerned with the social and psychologic as well as the biomedical dimensions of disorders related to excessive drinking.[3] Although physicians generally accept that the medical sequelae of alcohol abuse are within their domain, they are less ready to recognize and deal with the psychosocial elements. However, it is now clear that physicians must look beyond their traditional focus on biophysical factors if medicine is to significantly reduce alcohol-related morbidity and mortality.

We acknowledge valuable suggestions from our colleagues Drs. Mary Jane Ashley, Laurence Blendis, Harvey Haakonson, Bushan Kapur, Joan Marshman, Hector Orrego, Edward Sellers and Barry Sisson, and thank Romilla Chhabra for her assistance in preparing this manuscript.

The research for this paper was supported by the Addiction Research Foundation of Ontario. Stephen Holt acknowledges support from the Carnegie Trust for the Universities of Scotland.

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REVIEW ARTICLE

Early identification of alcohol abuse: 2: Clinical and laboratory indicators*

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Despite awareness of the wide variety of clinical and laboratory abnormalities associated with alcohol abuse, drinking problems often remain undetected in hospital and in general medical practice. The diagnosis of alcohol abuse has been emphasized repeatedly in the literature but far less attention has been paid to indicators that would permit detection of excessive drinking at a stage when intervention might be more effective and less costly. The search for indicators of early alcohol abuse is complicated since many of the medical sequelae of alcoholism are nonspecific and may only be manifested after a number of years of excessive drinking. Part 2 of this two-part series considers various clinical and laboratory features related to alcohol abuse and highlights items that are potentially more sensitive for detecting early stages of problem drinking. Use by physicians of a composite profile of both biomedical and psychosocial indicators of excessive alcohol consumption is recommended for early identification of this problem.

Malgre la connaissance des multiples anomalies cliniques et de laboratoire reliées à l'abus de l'alcool, les problemes d'alcoolisme échappent souvent a la detection, en milieu hospitalier comme en pratique generale. Dans la litterature, on a insiste sur le diagnostic de l'abus de l'alcool, mais on a accordé beaucoup moins d'attention aux indicateurs qui permettraient de deceler l'abus de l'alcool au stade ou une intervention pourrait être plus efficace et moins coûteuse. La recherche des indicateurs du début de l'alcoolisme est compliquée par le fait que plusieurs des sequelles medicales sont non specifiques et qu'elles peuvent ne se manifester qu'apres un certain nombre d'annees de consommation exagerée. Dans la deuxieme partie de cette serie de deux, on envisage diverses caracteristiques cliniques et de laboratoire rattachees a la consommation excessive d'alcool et on met en lumière les items susceptibles d'être plus sensibles pour detecter les premiers stades de l'alcoolisme. Afin de pouvoir identifier tôt ce probleme, on recommande l'emploi par le médecin d'un profil associant des indicateurs biomedicaux et psychosociaux de la consommation excessive d'alcool.

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*Part 1 of this review, which considered critical issues for identifying alcohol abuse and the psychosocial indicators for a composite index, was published in the May 1, 1981 issue of the Journal.

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An increasing awareness that excessive drinking is a major medical problem has prompted research aimed at determining reliable indicators of hazardous alcohol consumption.[1,2] Although many of the alcohol-induced symptoms, clinical signs and abnormal laboratory findings are nonspecific, it is reasonable to assume that the more alcohol-related problems a patient has, the greater the probability that excessive alcohol consumption has been or is present. Despite considerable interest in the medical sequelae of alcohol abuse, there is relatively little information on the value of using alcohol-related diseases as predictors of past or present problem drinking. Few epidemiologic studies have compared the disease patterns of patients who abuse alcohol with the patterns of control subjects. Without adequate prevalence data, the value of a variety of alcohol-associated disorders for detecting problem drinking in the general population cannot be adequately assessed.

Alcohol-related problems are known to be a common cause for medical consultation, and knowledge of the protean manifestations of hazardous alcohol consumption should aid in detecting alcohol abuse. This approach is encouraged by the successful use of a variety of medical factors known or believed to be associated with a high prevalence of alcoholism for the screening of patient populations in order to identify the "hidden alcoholic".[3-9] A recent World Health Organization (WHO) study group indicated that an important objective of future research on alcohol abuse should be the "development of methods for screening and early detection of alcohol-related disabilities, with correlation of questionnaire and laboratory methods".[1]

Although a number of reviews on the detection of alcohol abuse have appeared in recent years,[2,10] few

studies have focused on biomedical factors associated with both early and later clinical manifestations of excessive alcohol consumption. The aim of this paper is to discuss the value of various symptoms, signs and clinical investigations for identifying alcohol abuse. Particular attention is given to describing the morbidity profile at progressive stages in the development of alcohol abuse and on a critical appraisal of the growing number of laboratory tests and biochemical markers of such abuse.

This paper was prepared during the planning of a series of tests for identifying problem drinking. Before this research began at the Clinical Institute of the Addiction Research Foundation of Ontario in Toronto, attempts were made to justify including various biomedical and sociobehavioural items that might form the basis of an identification battery for the detection of alcohol abuse. The major thrust of this research is to determine accurate indicators of excessive alcohol consumption and related problems, and to incorporate these into a brief medical examination and a questionnaire for the patient that may be used in a variety of settings, including hospitals, industrial clinics and private practices.

Disorders related to alcohol abuse

Chronic hazardous drinking results in a wide spectrum of disorders causing a multitude of clinical symptoms and signs, many of which may be useful in detecting alcohol abuse. It is accepted that the functional and morphologic changes in many organs of the body that accompany or result from excessive alcohol consumption can provide reliable evidence of excessive drinking. However, relatively few clear correlations between clinical data and prolonged heavy drinking have been adequately demonstrated. Although alcohol abuse and problem drinking have diverse clinical manifestations, the most specific disorders usually represent advanced and often irreversible effects of alcohol and therefore may not be relevant to the early identification and treatment of these problems. Nevertheless, by accepting the assumption that the larger the number of alcohol-related alterations, the greater the probability that excessive alcohol consumption is present, a physician may compensate for the lack of specificity of some clinical items by using combinations of symptoms and signs.

In his excellent study on the hidden alcoholic in general practice Wilkins[1] drew attention to items in a patient's history that are valuable clues to alcohol abuse. These items, known or believed to be associated with a higher prevalence of alcoholism, were compiled in an "alcoholic at risk" register for detecting individuals whose drinking habits were already advanced. Wilkins recognized that earlier detection of alcohol abuse would improve the chances of arresting this disorder but concentrated his attention mainly on individuals with established alcoholism.

A patient's social history, including marital status, social class, living conditions, ethnic origin and occupation, should be considered since circumstances such as divorce, low social class and poor housing may be associated with a higher prevalence of hazardous alcohol consumption.[4,5] Wilkins emphasized that the general practitioner may be in an almost ideal situation for detecting alcohol abuse, as clues to the presence of alcoholism can be obtained by studying demographic data reported on patients' records even before they are seen in a clinic or an office. Alcoholics have a higher incidence of sickness, absenteeism, accidents, neurologic disorders, pulmonary disease and hypertension than matched controls.[1,6] It has been suggested that these alcohol-related disabilities may follow a recognizable sequence, and that early complications of alcohol abuse may manifest themselves before the patient or clinician is aware that a drinking problem exists.[7] For example, trauma and gastrointestinal disease tend to occur earlier in the course of alcohol abuse than neurologic disease or cirrhosis of the liver. The recognition of certain disorders as earlier indicators of hazardous alcohol consumption could aid in the detection of problem drinking.[7]

This review will concentrate on the principal medical sequelae of alcohol abuse, emphasizing the signs and symptoms related to gastrointestinal disorders, liver disease, neurologic alterations, trauma, and cardiovascular and respiratory disease. Attempts will be made to indicate disorders that are useful for detecting early rather than more advanced stages of alcohol abuse (Table I).

Gastrointestinal disease

Morning retching, nausea, anorexia and vomiting occur frequently in persons who abuse alcohol, and may be associated with hangovers or withdrawal symptoms.[8,9] Alcohol ingestion has been causally associated with a number of inflammatory lesions in the upper gastrointestinal tract. Because alcohol reduces the pressure of the lower esophageal sphincter and interferes with esophageal peristaltis, short-term or long-term alcohol abuse may promote gastroesophageal reflux.[10] Smoking, a common habit in abusers of alcohol,[11] may also reduce sphincter pressure, predisposing the drinker who smokes to esophageal reflux.[12] Portal hypertension induced by alcoholic cirrhosis may lead to esophageal varices and severe gastrointestinal bleeding.[13] Severe vomiting after overindulgence in food and alcohol may tear the mucosa at the gastroesophageal junction and cause hematemesis (Mallory–Weiss syndrome).[14] Erosive gastritis and acute duodenal erosions can develop in heavy drinkers and may lead to gastrointestinal hemorrhage.[15,16] Endoscopic studies have shown an increased prevalence of gastric mucosal disease in alcoholics. Moderate to severe antral gastritis has been found in 46% of alcohol abusers (the proportion was about half in a control group),[17] and abnormal gastric tissue was noted in all the members of a group of 51 patients with chronic alcoholism.[18] However, the evidence that alcohol consumption causes chronic gastritis has been questioned.[19]

Many authors have stressed a high frequency of dyspeptic symp-

toms in alcohol abusers. In one study of absenteeism in office staff it was noted that one third of a group of heavy drinkers were absent from work because of upper gastrointestinal complaints.[30] It would appear that the clinical manifestations of peptic ulceration are twice as common in alcoholic patients as in control subjects.[13,31] Although a number of studies suggest that the incidence of peptic ulcer is higher with alcohol abuse,[14,15,32] further investigation is required to substantiate this apparent relationship.[33]

Alcohol consumption is an important etiologic factor in both acute and chronic pancreatitis. The incidence of alcohol-associated acute pancreatitis in younger age groups seems to be increasing.[32] Chronic pancreatitis, when advanced, can cause an insufficiency of digestive enzymes. This may account for certain abnormalities in nutrient absorption that are associated with chronic alcohol abuse.[34] Motor function of the stomach and small intestines is affected by alcohol.[34-37] Robles and colleagues[35] have shown that impeding peristaltic waves are decreased by alcohol, but that propulsive movement remains unchanged. This altered small bowel motility could increase the rate of transit in the small bowel and may contribute to the diarrhea that is associated with "binge drinking". In addition, chronic, heavy consumption of alcohol may interfere directly with absorption in the small intestine.[38] Recent evidence indicates that caffeine consumed in normal amounts may increase the serosa to mucosa flux of fluid in the intestines, leading to an intraluminal accumulation of fluid and subsequently to diarrhea.[39] Thus, the alcohol abuser who also consumes caffeine may be even more prone to diarrhea.

Table 1—Clinical symptoms and signs of alcohol abuse

Symptoms and signs	Stage of appearance*	Diagnostic value†
General appearance		
Hand tremor	E	+
Excitability, irritability, nervousness	E	
Unkempt appearance	L	
Jaundice	L	+
Alcoholic facies	E	?
Mouth		
Coated tongue	E	
Peridontal disease	L	
Alcoholic fetor by day	E	+
Gastrointestinal tract		
Dyspepsia	E	
Morning nausea and vomiting	E	+
Recurrent diarrhea	E	
Recurrent abdominal pain	L	
Acute and chronic pancreatitis	E, L	+
Hepatomegaly	E	+
Splenomegaly	L	
Ascites	L	
Gastrointestinal bleeding	E, L	
Genitourinary system		
Polyuria	E	
Amenorrhea	L	
Impotence	E	
Face, skin and hands		
Rosacea, seborrheic dermatitis	L	
Parotid swelling	L	
Spider nevi	L	
Finger clubbing	L	
Dupuytren's contracture	L	+
Scars unrelated to surgical procedure	E	+
Cardiovascular and respiratory system		
Palpitations	E	
Cardiomyopathy	L	
Hypertension	E	
Chronic obstructive airways disease	L	
Recurrent chest infection and pneumonia	L	
Central nervous system		
Poor memory for recent events	E	
Blackouts	L	
Seizures	L	
Ataxia	L	
Peripheral neuropathy, myopathy	L	
Insomnia, nightmares	E	
Hallucinations	L	
Delirium tremens	L	
Wernicke Korsakoff syndrome	L	
Miscellaneous		
Trauma	E	+
Random blood alcohol level > 65 mmol/l (300 mg/dl)	E	+
No gross incidents of intoxication with blood alcohol level > 33 mmol/l (150 mg/dl)	E	+

*E = usually early; L = usually late.
†+ = probably a good indicator of alcohol abuse.

Both drinking in binges and chronic alcohol abuse may lead to the malabsorption of a variety of substances, including D-xylose,[40] calcium,[41] B-complex vitamins[40,42] and iron.[43] However, other factors are involved in the nutrient deficiencies of alcoholics. These include an inadequate diet and metabolic disorders secondary to chronic hazardous alcohol consumption.[44] Overt symptoms and signs of malnutrition represent relatively late-stage complications of alcoholism and appear to be more prevalent in the "skid-row" alcoholic.[45]

Liver disease

Liver disease occurs frequently with chronic alcohol abuse, and alcoholic liver disease in adult populations is positively correlated with the overall per capita consumption of alcohol.[46] About 10% of alcohol abusers have cirrhosis, which is more likely to develop in the "continuous imbiber" than in the "spree drinker".[47] The correlation between per capita alcohol consumption and rates of death from cirrhosis is well recognized.[48,49] In one large study the observed death rate from cirrhosis in 6478 alcoholics was about 13 times more frequent than would be expected in the general population.[47] Although

cirrhosis may present a number of characteristic clinical symptoms and signs, it occurs at a relatively late stage in the morbidity sequence of alcoholism.[35] It may take 5 to 10 years of chronic alcohol abuse before cirrhosis appears.[36] Important precursors of cirrhosis including fatty liver, alcoholic hepatitis and fibrosis, may frequently be asymptomatic and have few or even no clinical signs.[36-41] For example, the spectrum of those with alcoholic hepatitis may range from an asymptomatic individual to a patient with fever, jaundice encephalopathy and ascites.[37] Rankin and coworkers[38] drew attention to the problems of diagnosing alcoholic liver disease by demonstrating a lack of correlation between clinical signs and the severity of underlying liver disease as assessed by liver biopsy. The degree of overlap of clinical manifestations of the various liver disorders is such that differentiation on clinical grounds is not possible with any degree of accuracy.[41]

It has been suggested that a composite index, derived by multivariate statistical analyses of various clinical signs and tests of liver function, provides greater diagnostic accuracy than consideration of any single test.[36] An important attempt to overcome some of the problems surrounding the clinical diagnosis of alcoholic liver disease is the development of a composite index to assess severity of alcoholic liver disease.[36] This clinical and laboratory index is based on 11 clinical signs (hepatomegaly, splenomegaly, ascites, encephalopathy, a clinically overt tendency to bleed, spider nevi, palmar erythema, collateral venous circulation on the anterior abdominal wall, circulation, peripheral edema, anorexia and weight loss) and six laboratory findings (levels of glutamic oxaloacetic transaminase [SGOT], γ-glutamyl transpeptidase [GGT], alkaline phosphatase, albumin and bilirubin in the serum, and prothrombin time). The scoring system of this index is based on the concept that the severity of the underlying liver disease is proportional to the number of abnormal clinical and laboratory findings. Certain items, such as encephalopathy, ascites, a raised level of serum bilirubin and a prolonged prothrombin time, are weighted because they are known to be associated with more advanced disease and a poorer prognosis.

Neurologic disease

Both periodic heavy drinking and chronic alcohol abuse produce a variety of complex metabolic and pathophysiologic alterations in the central and peripheral nervous systems. However, the precise cause of many of the neurologic sequelae of alcohol abuse is unknown. Neurologic disorders in the alcoholic have been classified on phenomenologic, etiologic and neuropathologic bases.[42-44] The main neurologic disturbances in alcohol abusers are acute intoxication, withdrawal symptoms (e.g. tremulousness), hallucinations, epilepsy, delirium tremens, Wernicke–Korsakoff syndrome, polyneuropathy, cerebellar degeneration, central pontine myelinolysis, Marchiafava–Bignami disease, neurologic sequelae of chronic hepatic disease (e.g., hepatic encephalopathy), cerebral atrophy with neuropsychologic impairment and alcoholic dementia.[45] In addition to neurologic dysfunction, alcohol abusers may have an uncommon but well defined syndrome of acute alcoholic myopathy, with muscle pain, tenderness, swelling and variable myoglobulinuria.[45-51] A chronic myopathy may occur with an insidious onset of muscle weakness and atrophy.[45-52]

Although symptoms and signs of withdrawal from drinking alcohol may be useful clinical indicators of alcohol abuse, they are extremely variable. It is especially important to be aware of mild withdrawal reactions such as tremor, anxiety, insomnia, hyperreflexia, and a lowered seizure threshold.[53-55] All of these symptoms may appear within a few hours of withdrawal from drinking alcohol and may last for approximately 2 days.[56] Severe withdrawal reactions, manifested by confusion, hallucinations, seizures and full-blown delirium tremens, are often most evident between 2 and 4 days after withdrawal but may persist for up to 10 days.[57]

Although peripheral neuropathy may be present in up to 10% of cases of chronic alcohol abuse,[52,58] it may occur only after a number of years of heavy alcohol use.[52] The onset of symptoms of this disorder is variable but usually extends over weeks or months. The neurologic deficit is frequently bilateral, symmetric and sensorimotor in type. It is important to be aware that subclinical neuropathy is common. Clinical findings include weakness, muscle wasting and tenderness, a loss of reflexes and distal sensory impairment or loss. Patients may complain of burning feet or of trophic skin changes in the lower limbs. The legs alone are affected in approximately 70% of all cases.[52,59] Significant defects in memory and other cognitive processes are often present in alcohol abusers,[60-62] but clinically overt intellectual impairment may be present in a smaller proportion of patients.[63-65] Dementia occurs in about 9% of abusers of alcohol, its incidence increases with age and it is more common in women than men.[66]

Many of these neuropsychologic deficits have been presumed to be related to cerebral atrophy.[67] Recent studies have indicated that both abnormal results of neuropsychologic tests and cerebral atrophy, as measured by computer-assisted transaxial tomography, are present in abusers of alcohol.[68-71] However, these measures may not reflect the duration of heavy drinking, and the neuropsychologic test results do not always correlate with the amount of cerebral atrophy.[72] Although physicians do not generally use neuropsychologic tests, it seems likely that such tests would be effective in detecting early stages of alcohol abuse.

Trauma and accidents

Alcohol abuse plays a major role in accidents, criminal behavior, acts of violence, suicide and other serious events.[1,2] Numerous studies have implicated alcohol as a cause of many traffic, industrial and recreational accidents.[73-75] A direct result of an increased accident rate among abusers of alcohol is a high incidence of traumatic injuries. Past or present traumatic events are among the most common diagnoses in heavy drinkers admitted to treatment units.[76-78] One survey estab-

lished that 36% of regular drinkers had reported at least two accidental injuries in the preceding year, compared with an accident rate of only 8% in nondrinkers.[88] The extent to which alcohol is involved in accidents and emergency admissions to hospitals has been substantiated by a recent study that demonstrated that approximately one third of all patients attending a casualty department in a large general hospital in the evening had a blood alcohol concentration of over 17.4 mmol/l (80 mg/dl).[89] Bone fractures occur commonly in heavy drinkers,[90-92] and since injuries are known to be among the most common causes for medical consultation in inebriated patients,[93] an awareness of the prevalence of such alcohol-related morbidity can aid in detecting alcohol abuse.

In an investigation conducted by the Addiction Research Foundation of Ontario in Toronto, rib or thoracic vertebral fractures or both were found on the routine chest roentgenograms of 28.9% of a group of alcoholic men but in only 1.3% of a matched control group of social drinkers.[94] An increased exposure to trauma was the most likely cause of this 16-fold increase in the number of thoracic fractures. Since trauma occurs early among the problems related to alcohol abuse, evidence of fractures may be an early indicator of past or present hazardous alcohol use.

A number of studies have drawn attention to the association between acute alcohol intoxication and head injury.[95-97] In one prospective study 62% of the men and 20% of the women admitted to hospital with a head injury had detectable levels of alcohol in their blood.[95] Galbraith and associates[97] observed that assaults and falling while under the influence of alcohol were common modes of head injury, whereas road traffic accidents accounted for only 25% of cases. Preliminary data from a study of patients with chronic subdural hematoma admitted to a large district general hospital in Toronto indicate that between 30% and 40% of such patients may be abusers of alcohol (M.S. Jacobs, P.L. Carlen, personal communication, 1979).

Cardiovascular and respiratory disease

A number of investigations have documented an association between alcohol consumption and hypertension.[98-101] In a study of the alcoholic employees of a large company, Pell and D'Alonzo[101] found a two- to threefold greater prevalence of hypertension (a systolic blood pressure higher than 160 mm Hg or a diastolic pressure higher than 95 mm Hg) in alcohol abusers than in matched controls. From a survey of 84 000 people Klatsky and associates[98] reported that the prevalence of hypertension was 11.2% in individuals ingesting six or more drinks a day compared with 4.6% in nondrinkers. Alcohol abusers have an increased risk of premature death from diseases of the cardiovascular system.[98] It seems likely that alcohol consumption, by contributing to hypertension, increases the risk for such disease. However additional investigation is required to define the level of alcohol consumption that is associated with an increased risk of cardiovascular disease and to ascertain the underlying mechanisms.[100]

The relation between alcohol abuse and coronary artery disease requires further clarification, since it appears that the prevalence of coronary artery disease decreases as the quantity of alcohol consumed increases, up to an intake of about 70 ml of ethanol a day.[102] Those who drink more than this amount may have a higher risk of coronary artery disease than nondrinkers.[103] A clear association between alcohol abuse and cardiomyopathy is well recognized, and is usually manifested by congestive cardiac failure in alcohol abusers under the age of 50.[71,104] Alcoholic cardiomyopathy may be acute or chronic, and is often manifested by tachycardia and hypotension.[71] Cardiac arrhythmias may occur in association with cardiomyopathy[105] or with intoxication in patients without other clinical evidence of underlying heart disease.[106-108]

Alcohol abuse is frequently associated with pulmonary disease, and especially with chronic obstructive airways disease, pulmonary fibrosis, tuberculosis and bronchiectasis.[109-113] A predisposition to pulmonary disease may be a direct result of the toxic effect of alcohol on the lung or an indirect result of the fact that alcoholics tend to be malnourished, to suffer from aspiration pneumonia and repeated respiratory tract infections and to be heavy cigarette smokers.[109,114] It seems likely that excessive smoking is a major cause of cardiac and pulmonary disease in alcoholics.[71,109]

Miscellaneous symptoms and signs

Normal sleep patterns are disrupted by alcohol consumption.[115] Sleep problems such as insomnia and frightening dreams are frequently experienced by abusers of alcohol.[116] Detailed studies of the sleep of alcohol abusers and of normal volunteers following alcohol ingestion have shown that both the quality and the quantity of sleep are disturbed by alcohol.[115,117] Complaints of sleep disturbance, especially from young patients, should prompt a clinician to enquire about drinking habits.

Reduced libido and impotence are recognized as being associated with chronic alcohol abuse. Masters and Johnson[118] indicated that secondary impotence in men is frequently caused by the excessive consumption of alcohol. Lemere and Smith[119] reported impotence in 8% of over 17 000 alcoholic men but found little evidence of sexual dysfunction in women who abused alcohol. Most alcoholic patients report that sexual performance has been normal for a number of years, but in the later stages of their heavy drinking sexual ability tends to be reduced.[118] The wide variability in what is considered normal sexual performance and the problem of obtaining accurate accounts of sexual function render sexual activity difficult to study. Important components of impotence that the alcoholic man may experience are a reduction or absence of sexual drive and a failure to achieve erection or ejaculation or both.[71] The causes of sexual dysfunction in the alcoholic are not understood completely but Lemere and Smith[119] have suggested that alcohol may damage the complex of neurologic reflexes subserving erection and that

psychologic factors may often be of secondary importance.

Margolis and Roberts[114] reported that 24% of a population of "chronic drinkers" derived from 500 consecutive admissions to hospital medical wards had significant skin lesions. Metabolic derangements, poor hygiene, inadequate nutrition and trauma may cause cutaneous lesions in alcohol abusers.[115] A wide variety of cutaneous stigmata may provide useful clues for the detection of alcohol abuse.[116-120-121]

The face of the habitual drinker often shows persistent erythema with or without telangiectasia, capillary engorgement of the conjunctiva, edema of the forehead and periorbital region, and grooving and thickening at the eyelid margins. A "whiskey nose" or rhinophyma may be present.[122] Young[123] has usefully divided cutaneous signs of alcohol abuse into three categories describing the sequelae of acute alcoholism, the sequelae of chronic alcoholism and the dermatoses that are influenced by alcohol. Trauma and injury, which occur commonly in alcohol abusers and problem drinkers, may cause bruises and scars in unusual numbers or sites.[119] Uncommon or unusual cutaneous signs, such as pressure sores,[124] widespread insect stings,[125] tattoos,[126] frostbite scars,[127] extensive varicose ulcerations in the lower leg,[127] eruptions caused by drug reactions,[128] cutaneous signs of drug abuse,[127] abnormal sweating,[129-130] and cutaneous infections[126] may all provide a clue to alcohol abuse. A growing dependence on alcohol frequently leads to self neglect.[114] The patient may be unkempt and practise poor skin and oral hygiene. In addition, there appears to be an increased incidence of rosacea, neurodermatitis, dermatophytosis, seborrhea capitis, seborrheic dermatitis, eczematoid dermatitis, aggravated acne and psoriasis in alcoholics.[116-119-122]

When chronic alcohol abuse is associated with underlying liver disease, more specific skin changes appear.[56] Vascular skin lesions such as spider angiomas and wiry telangiectasia[122-131] are among the more specific indicators of underlying liver disease in an alcohol abuser,[61] although they may also occur in healthy people.[132] Other signs of alcoholic liver disease include jaundice, purpura, abdominal varices (caput medusae), "paper money skin", the venous star, Campbell de Morgan's spots (cherry angiomas),[57] sparse axillary and pubic hair, with testicular atrophy,[133] gynecomastia,[134] generalized itchiness with scratch marks,[118] palmar erythema,[124] Dupuytren's contracture,[132] pigmentary changes with pellagroid melanosis of areas exposed to the sun,[122] and a variety of finger-nail changes, such as clubbing,[57] white nails,[124] white-banded nails,[135] opaque nails[136] and thinned nail folds with a wide cuticle.[122]

Painless unilateral or bilateral enlargement of the parotid gland caused by fatty infiltration of acinar tissue is sometimes present in alcoholic patients.[140-141] In one study 41 out of 50 patients with asymptomatic parotid gland enlargement were alcoholic, and 25 of this group had cirrhosis of the liver.[41] Wolfe and collaborators[141] also drew attention to the increased incidence of parotid enlargement in patients with alcoholic liver cirrhosis, but other investigators have noted this condition in patients who did not have cirrhosis and drank only moderate amounts of alcohol.[142] In an analysis of the clinical features of 60 new patients referred to a joint psychiatric and medical outpatient clinic for alcohol abusers, Shaw and Thomson[48] found that 3.3% of the group had parotid enlargement. A compensatory type of enlargement of this gland may also be associated with calcific pancreatitis in nonalcoholic patients,[143] and transient parotid enlargement with excessive salivation has been recorded in an alcoholic man in association with recurrent attacks of acute pancreatitis.[140] The cause of parotid swelling in abusers of alcohol is unknown, but could be related to poor nutrition or to vitamin A deficiency.[144] A reduction of alcohol intake may decrease the amount of enlargement of the parotid gland, as will an improvement in liver function in patients with cirrhosis.[136] The relative size of the parotid gland may be a useful clinical sign for detecting chronic alcohol abuse and perhaps provide an indication of reduced or continued alcohol consumption.

Laboratory tests and biochemical markers

As with many of the clinical signs and symptoms used to diagnose alcohol abuse, laboratory tests used for this purpose are often relatively nonspecific.

The only true indicator of alcohol consumption is the detection of alcohol or one of its metabolites in the patient's body fluids. However, the relatively short half-life of these compounds and the fact that their presence alone does not predict the patient's drinking habits or tolerance of alcohol detract from their usefulness as markers of alcohol abuse. Both the circumstances under which alcohol is detected in body fluids and its concentration may be strong indicators of the presence of hazardous alcohol consumption. The National Council on Alcoholism (NCA) criteria for diagnosing alcohol abuse regard a blood alcohol level of greater than 65 mmol/l (300 mg/dl), recorded at any time, or a level of more than 22 mmol/l (100 mg/dl), recorded during a routine clinical examination, as important indicators of alcoholism.[121] A blood alcohol level of more than 33 mmol/l (150 mg/dl) in a patient who is not obviously intoxicated is evidence of tolerance to alcohol and is also a strong indicator of alcohol abuse.

The laboratory tests that may be used to detect alcohol abuse will be reviewed. A summary of key findings to date is given in Table II.

Alcohol and acetaldehyde

Estimates of the level of alcohol in urine or blood have an established role in the diagnosis of intoxication,[145] and repeating these estimations may provide an indication of chronic hazardous alcohol consumption.[133-134] Clinical signs such as slurred speech, muscular incoordination, alcoholic fetor and erythema of the conjunctiva are often unreliable indicators of intoxication since a medical practitioner may consider a patient sober when blood alcohol concentrations are recorded at a level consistent with marked

inebriation.[156] When difficult management decisions occur because of suspected intoxication, a test that will give an immediate result is required. This has led to the analysis of alcohol in the breath as a diagnostic tool in clinical practice.[156] Inaccuracies in the use of breath alcohol analysers have prompted studies of saliva and sweat as indicators of alcohol abuse[155,157] and these methods seem promising. It may be possible to obtain an objective estimate of the amount of alcohol consumed over a period of time by analysing sweat,[157] but the value of this technique in general clinical practice has not been explored in detail.

Acetaldehyde and acetate, which are products of the oxidation of alcohol in the body, can be measured in blood. However, technical difficulties in their measurement in the laboratory, their low concentrations in blood and their short half-life render it impractical to use them as a diagnostic test, and they offer little advantage over measuring blood alcohol levels.[1]

Multitest approaches

There are many examples of the use of a battery of laboratory tests, especially in patients in hospital, to diagnose alcohol abuse.[158-171] In a large survey of patients attending a multiphasic health screening centre, alcohol consumption, within the range that may be considered normal, was found to affect a number of biochemical and hematologic findings, including the serum levels of GGT, uric acid, triglycerides, aspartate aminotransferase (ASAT) and the mean corpuscular volume (MCV).[167] Age- and sex-related differences were noted in the results of a number of tests, and fewer abnormalities were detected in younger patients. From these data Whitfield and colleagues[167] suggested that these laboratory tests could be used to compare alcohol intake within groups of individuals over a period of time. However, if such tests were used for the early identification of alcohol abuse there might be false-negative and false-positive results. Therefore, it is necessary to interpret laboratory findings in conjunction with other medical or sociobehavioural data.[166]

Blood lipids

Alcohol exerts an effect on the metabolism and transport of lipids, tending to raise serum concentrations of triglycerides and high-density-lipoprotein (HDL) cholesterol.[172-175] Results of the Cooperative Lipoprotein Phenotyping Study indicated that alcohol consumption was correlated with HDL-cholesterol levels in all populations, and that lipid levels appeared to show a graded response even over lower ranges of alcohol consumption.[172] In addition, it was noted that serum triglyceride levels showed a modest positive correlation and low-density-lipoprotein (LDL) cholesterol levels a consistently negative correlation with alcohol consumption.[172] Other studies in large populations have confirmed these observations.[173-175] It has been demonstrated in healthy volunteers that when alcohol is added to a normal diet HDL-cholesterol levels rise, whereas with a cessation of drinking these levels fall.[176,177]

Despite the strong association between changes in blood lipids and alcohol consumption, there have been relatively few studies of HDL-cholesterol levels in alcoholic populations. Johansson and Medhus[178] measured serum HDL-cholesterol levels in 69 alcoholic men who had been on drinking bouts for various periods (5 to 10 days) before admission to hospital. They observed that HDL-cholesterol levels were increased in 60 of the patients but tended to return to normal within 2 weeks after the patients stopped drinking. However, they found no correlation between HDL-cholesterol concentrations and liver damage as assessed from the serum levels of bilirubin, SGOT, glutamic pyruvic transaminase (SGPT) and GGT, and the retention of sulfobromophthalein. In a study of similar design an increase in serum HDL-cholesterol levels was noted in 25 of 39 alcoholic men while they

Table II—Laboratory markers of excessive alcohol, or ethanol, consumption

Marker	Diagnostic value
Serum γ-glutamyl transpeptidase level	Raised in 70% to 80% of alcoholic patients. Responds to ethanol consumption in excess of 40 to 60 g/d. Probably one of the best early indicators except in individuals with nonalcoholic liver disease and those taking other drugs.
Mean corpuscular volume	Raised in 75% to 90% of alcoholic patients. Appears to respond to ethanol consumption in excess of 60 g/d.
Serum aspartate aminotransferase level	Raised in 30% to 75% of alcoholic patients. Primarily indicative of liver disease and probably not responsive to low levels of alcohol consumption.
Serum high-density-lipoprotein cholesterol level	Raised in 50% to 80% of alcoholic patients. Probably sensitive to moderate ethanol consumption (40 to 60 g/d) but not in patients with severe alcoholic liver disease.
Serum glutamate dehydrogenase level	Raised in alcoholic patients with severe liver disease and in patients with fatty liver following excessive alcohol ingestion. Not responsive to consumption of 140 g/d for 4 weeks in normal individuals. Actual prevalence of abnormal values in alcoholic patients not clear.
Serum transferrin level	Raised in 81% of alcoholic patients who consume over 60 g/d of ethanol. Not present in patients with nonalcoholic liver disease and raised levels of serum glutamic oxaloacetic transaminase. Sensitive to low to moderate ethanol consumption. Quantitation and methodologic simplification of test methods should render this determination valuable.
Ratio of α-amino-n-butyric acid to leucine	Raised in some types of alcoholic patients but not in others. Apparently dependent on liver dysfunction and nutritional status.

were intoxicated,[186] but again no correlation was found between HDL-cholesterol levels and other measures of liver function. However, these studies did not provide data on the patients' dietary and nutritional status, the type or amount of alcohol they consumed, the results of liver biopsy or details of prior drug treatment, all of which may modify serum concentrations of HDL-cholesterol.

Since liver disease exerts a major effect on the level of HDL-cholesterol in the serum, using this test as a marker of alcohol consumption may not be useful in patients with alcoholic hepatitis or cirrhosis. To further examine the relationship between serum HDL-cholesterol levels and alcohol consumption in patients with liver disease, liver function and alcohol consumption (assessed objectively by estimating the amount of alcohol in the urine) were measured in 57 alcoholic patients with liver disease.[187] HDL-cholesterol levels were also determined in a control group of 67 hospital employees with no known history of hazardous alcohol consumption. There was no significant difference between the HDL-cholesterol levels of the patients and the controls. However, within the patient group HDL-cholesterol was found to be significantly higher in those who were still drinking (positive urine tests) than in those who had not been drinking. Nevertheless, serum HDL-cholesterol levels were found to be inversely correlated with the severity of liver disease as assessed by laboratory measures of prothrombin time, total bilirubin, albumin and alkaline phosphatase. Although HDL-cholesterol has a tendency to be higher in alcoholic patients who are still drinking it may be within normal limits in patients with liver disease, largely because impaired liver functions will tend to lower HDL-cholesterol levels. Thus, a measure of HDL-cholesterol may not be a reliable test for detecting alcohol abuse in patients with liver disease.[187]

Enzymes

The concentration of a number of enzymes in the blood is known to increase with excessive alcohol consumption. These enzymes include SGOT (ASAT), SGPT (alanine aminotransferable [ALAT]), glutamate dehydrogenase (GDH), lactate dehydrogenase (LDH) and alkaline phosphatase. Skude and Wadstein[188] found that in a group of patients admitted to a Swedish hospital for the treatment of alcohol abuse 77% had raised SGOT values and 50% had raised SGPT values. Rosalki and Rau[189] reported that 32% of a similar group of patients had raised SGOT levels. Orrego and coworkers[4] studied alcoholic patients with mild liver disease and found that 76% had raised SGOT values. Although levels of SGOT and SGPT are nonspecific indicators of liver damage, an SGOT/SGPT ratio greater than 2 is considered highly suggestive of alcoholic hepatitis or cirrhosis or both.[190]

The serum GGT level appears to be a good early indicator of alcoholic consumption and has been shown to be raised in about three quarters of a group of alcoholic patients who had no evidence of hepatomegaly or other clinical signs of liver disease.[191,192] While the serum GGT level is known to be raised in patients with a variety of liver diseases, it may also be raised in alcoholic patients at a time when the SGOT, SGPT and alkaline phosphatase levels are normal.[193] GGT is stored mainly in the liver as a membrane-bound constituent of the microsomal fraction.[191] Since an increase in microsomal mass is one of the earliest results of chronic alcohol consumption, the location of GGT probably accounts for its special sensitivity as an indicator of liver disturbances in heavy drinkers. Although an increased level of GGT may correlate with liver cell necrosis,[193] in many cases it probably reflects microsomal induction. It may also be raised because of drug therapy.[194]

Rollason and associates[195] compared the levels of GGT, SGOT and alkaline phosphatase in groups of subjects with different drinking habits. They were able to demonstrate significant differences in these levels between nondrinkers and heavy drinkers. Whitehead and collaborators[196] found an increased level of GGT in 17% of 2034 men in London, England and suggested that excessive alcohol consumption caused the raised levels of the enzyme in over 60% of cases. Pomerleau and colleagues[196] confirmed this observation by finding a significant correlation between the ethanol consumption reported by a group of subjects seeking treatment for alcohol abuse and the subjects' GGT levels. Although the relation between alcohol consumption and raised serum levels of GGT has been demonstrated by numerous studies,[197-203] its use as a diagnostic marker of alcohol abuse may have certain limitations.[204,205]

Studies of LDH concentrations in blood obtained from chronic abusers of alcohol while they were intoxicated indicated that LDH isoenzyme levels change in most drinkers, with a tendency to increases in the LDH-1, LDH-2 and LDH-5 fractions.[196,197] In a study of 100 alcoholic patients the GDH level was found to be a reliable marker of liver cell necrosis.[196] Raised GDH concentrations in the blood discriminated between patients with hepatic necrosis and those without, as assessed by liver biopsy. Furthermore, GDH measurement was able to detect cases of alcoholic hepatitis that were considered "clinically silent" and seemed to yield few false-positive results. However, data from other studies have not confirmed these findings. Jenkins and associates[206] reported that in alcoholic patients with fatty livers, those who had recently consumed an excessive amount of alcohol also showed increased serum levels of GDH. In addition, the level of this enzyme was not markedly raised in nonalcoholic volunteers consuming 2 g ethanol/kg every day for 4 weeks.[206]

Ratio of plasma α-amino-n-butyric acid to leucine

Recently interest has focused on using the ratio of α-amino-*n*-butyric acid (AANB) to leucine in the plasma as a potential biochemical marker of alcohol abuse. Shaw and Lieber[210] have shown that chronic alcohol abuse produces an increase in the level of AANB in the plasma. However, this increase does not

result from a single bout of heavy drinking, which suggests that AANB might be a useful indicator of long-term alcohol consumption. Since the level of AANB is known to be depressed by a deficiency of dietary protein, and since the diet of alcoholics is frequently deficient in protein,[208] it has been proposed that a more accurate marker of alcoholism might be obtained by expressing the level of AANB relative to that of leucine.[211] Expression of the ratio to leucine was chosen because the concentration of leucine in the blood is depressed by protein malnutrition.

Conflicting data have been reported on the use of the AANB:leucine ratio for diagnosing alcohol abuse. Shaw and coworkers[211] reported an increased ratio in well nourished baboons fed alcohol and in both ambulatory and hospitalized alcoholic patients. Furthermore, these investigators found a significant positive correlation between the AANB:leucine ratio and the degree of alcohol abuse as assessed by separate criteria (e.g., NCA criteria and reports of average daily ethanol intake) in a sample of alcoholic and nonalcoholic patients taking methadone. These findings were confirmed in two subsequent studies.[212,213] Using the AANB:leucine ratio in combination with GGT levels was reported to detect alcohol abuse in 26 out of 33 heavy drinkers, with a false-positive rate of only 2%.[213] Recently Shaw and collaborators[214] reported that the level of AANB decreased during withdrawal from alcohol and during abstinence.

Other investigators have disagreed with the findings of Shaw's group. Morgan and colleagues[215] concluded that the AANB:leucine ratio provides an indication of hepatic dysfunction rather than long-term alcohol abuse. Data from Dienstag and associates[216] showed that this ratio increased nonspecifically in humans with liver disease unrelated to alcohol and in animals with liver cell injury. These findings indicate that a raised AANB:leucine ratio is not necessarily characteristic of chronic alcohol abuse. Ellingboe and coworkers[216] confirmed these findings and concluded that this ratio cannot be used as an empirical biochemical marker of long-term alcohol abuse. Finally, Hilderbrand and collaborators[217] found no significant correlation between the AANB:leucine ratio and either reported drinking or GGT levels.

Thus, the potential use of the AANB:leucine ratio to screen for alcohol abuse is questionable. Ellingboe and coworkers[216] suggested that these conflicting results might be explained by the fact that hepatocellular disease in general, rather than alcohol consumption alone, increases this ratio. It is likely that a considerable number of patients studied had liver disease, which could account for the raised AANB:leucine ratios. Even with unanimous evidence to support its value, this ratio is unlikely to be used routinely since AANB measures are expensive, requiring the use of an amino-acid analyser.

Hematologic tests

Excessive alcohol consumption may produce a variety of changes in the hematopoietic system, including anemia with suppression of erythropoiesis,[47] cytoplasmic and nuclear vacuolation of early myeloid and erythroid precursors in the bone marrow,[218-225] altered folate status and its hematologic consequences,[47] transient hemolysis with hyperlipidemia,[221,222] a reversible type of sideroblastic erythropoiesis,[22] leukopenic and leukocytopenic responses to bacterial infections,[221,223] thrombocytopenia,[224-227] hemostatic defects[228-230] and a variety of erythrocytic abnormalities, including macrocytosis,[226,231] acanthocytosis[232,233] and stomatocytosis.[234]

The most frequent hematologic findings a clinician may observe in an alcoholic patient are a normal hemoglobin concentration, a normoblastic marrow, normal serum B_{12} and folate concentrations and a raised MCV.[47] Several studies have indicated that an increased MCV indicates heavy alcohol consumption, and that estimation of the MCV may be important in the detection of alcohol abuse.[169,210,216,235] Unger and Johnson[235] found that 3% of the 8000 employees of a large insurance company had macrocytosis (MCV more than 96 fl for men and more than 100 fl for women); a large proportion of the individuals with a high MCV were considered to be consuming excessive amounts of alcohol. Wu and collaborators[236] determined the MCV in 63 alcoholic inpatients of a general hospital and found that 89% had macrocytosis, generally associated with anemia. In this study megaloblastic marrow samples were found in only one third of the patients. Wu and collaborators[237] were able to demonstrate that macrocytosis resolved with alcohol withdrawal but persisted if alcohol intake continued, despite folate supplementation. They concluded that macrocytosis was due to a direct action of alcohol on developing erythroblasts, an opinion held by other workers.[238-240]

In a study of risk factors for cardiovascular disease the hematologic profiles of healthy men aged 48 to 54 years were examined with reference to both their alcohol consumption and their smoking habits.[239] It was found that the correlation of alcohol consumption with erythrocyte count and MCV was more marked in smokers. In contrast, the correlation between alcohol consumption and leukocyte count was more marked in nonsmokers. These findings underscore the need to consider smoking habits with alcohol consumption when interpreting changes, especially in view of the strong association between alcohol abuse and cigarette smoking.

Miscellaneous tests

Changes in the level of uric acid in the serum have been observed in association with heavy drinking.[166] In one large study it was demonstrated that heavy drinkers tended to have higher serum uric acid levels than light drinkers, but this difference was apparent only in men.[167] Changes in serum electrolyte levels, with a decrease in chloride and an increase in lactate concentrations, resulting in acidosis and a decreased excretion of uric acid, may be found in heavy drinkers.[241,242] It has been suggested that if a patient's serum uric acid level is raised on admission to hospital but returns to normal after a few days, then alcohol involvement can be suspected.[4]

Disturbances in acid–base bal-

ance, with ketosis,[79] alterations in porphyrin metabolism[141] and disturbances in carbohydrate metabolism,[84] may occur with chronic alcohol abuse. Alcohol intake may result in an increased excretion of porphobilinogen, aminolevulinic acid and coproporphyrins. In some circumstances there may be a relation between changes in porphyrin excretion and the amount of alcohol consumed.[139,140] Alcohol exerts a complex effect on carbohydrate metabolism. Following heavy alcohol consumption individuals can have low blood glucose levels while fasting,[79] or can exhibit hyperglycemia or glycosuria, especially if they have liver disease.[80,139]

Alcoholic patients have been found to excrete in the urine an increased amount of D-glucaric acid while drinking; this amount decreases after they stop drinking.[83] Urinary glucaric acid excretion increases after the administration of certain drugs and has been used as a marker of microsomal enzyme induction.[142,143] While investigating the role of D-glucaric acid as a marker of alcohol abuse, Spencer-Peet and colleagues[144] found no correlation between serum GGT and urinary glucaric acid levels. This may be due to the fact that an increased GGT level in alcoholics reflects both enzyme induction and structural damage within the liver. If a persistent increase in the amount of D-glucaric acid excreted is directly related to continuous drinking, measurement of the urinary excretion of this substance may be of value in the detection of problem drinking.

Serum levels of bile acids have been found to be abnormally raised in a large number of patients with alcoholic liver disease.[145,146] In a study cited by Overby[86] the serum levels of cholylglycine were found to be raised in 97% of 144 patients with alcoholic liver disease. Milstein and associates[147] detected marginally increased or normal total bile acid levels in the serum of six patients with alcohol-related fatty liver, and significantly raised levels in 93% of a group of 58 patients with more advanced alcoholic liver disease. These findings suggest that an increased concentration of serum bile acids may be a more sensitive indicator of alcoholic liver disease than the results of a number of more standard serologic tests.[148]

A number of studies have drawn attention to abnormalities of mineral and vitamin metabolism in alcoholics. The excretion of magnesium, zinc and calcium is increased by alcohol consumption, so that serum magnesium and zinc concentrations may be low in alcoholic patients.[87] Vitamin and nutrient deficiencies are common in patients who drink heavily. Their cause is determined by a complex interaction of such factors as poor diet and malabsorption, the specific interference of ethanol with the metabolism of vitamins and the presence of liver disease. In a study to detect vitamin B and C deficiency in patients with alcohol-related illnesses Baines[149] found the prevalence of riboflavin deficiency to be 23%, that of thiamin deficiency, assessed by the pyruvate tolerance test, 55% and that of ascorbic acid deficiency 91%. Baines also reported a poor correlation between vitamin deficiency and GGT activity in the serum. In view of the many influences on vitamin status in alcoholic patients, the use of serum or urinary vitamin levels to diagnose alcohol abuse may not be very rewarding.

New biochemical markers

Two possible markers of alcohol abuse have recently been reported: plasma transferrin and salsolinol. Studies by Stibler and coworkers[87] have shown that electrofocusing of plasma proteins and further characterization by immunofixation reveals an abnormal transferrin band with a pH of 5.7 in 81% of alcoholic patients admitting to consumption of more than 60 g of ethanol a day in the previous week. The percentages were 75% and 25% for those reporting a consumption of 20 to 60 g/d and 7 to 20/d respectively, 8% for those reporting abstinence and 1% for control subjects. When ethanol was given to eight controls at a daily dose of 0.6 g/kg an abnormal transferrin appeared after 5 days in one subject at 7 days in a second and at 11 days in a third; the SGOT level was normal in all three. Furthermore, an abnormal transferrin could not be detected in 22 patients with nonalcoholic liver disease, although 84% had abnormally high SGOT levels. The transferrin abnormalities apparently disappeared after the patients stopped drinking alcohol for 10 days.[85,86]

These data indicate that transferrin could be a good indicator of alcohol abuse since it appears not to reflect liver disease and may detect low to moderate alcohol consumption. Unfortunately, the electrofocusing-plus-immunofixation technique is too complex for routine laboratory analysis. Another promising marker appears to be salsolinol, the product of condensation between acetaldehyde and dopamine. (Salsolinol has been found to be 20 times more concentrated in the urine of alcoholic patients admitted for detoxification than in that of controls.[88]) The concentration of salsolinol was reduced to baseline levels following 4 days of withdrawal of alcohol. As with transferrin, the current method for determining salsolinol levels — high-performance liquid chromatography plus mass spectrum analysis — is too elaborate for routine analysis.

Discussion

Excessive alcohol consumption produces measurable physiologic, pathologic, biochemical and morphologic changes in many organs of the body. However, these changes may also be produced by a variety of diseases not related to alcohol abuse. Even the most specific indicators of alcohol abuse may only measure the events resulting from alcohol or its metabolites in a relatively small proportion of alcoholic individuals. Detecting alcohol or a product of its metabolism in body fluids is the only direct way of ascertaining alcohol abuse, but these compounds have a relatively short half-life and cannot provide a measure of alcohol tolerance or of the duration and extent of previous alcohol abuse. The circumstances under which alcohol is detected is important. For example, the presence of alcohol in blood specimens obtained in the morning[150] or following an accident[151] may be a strong indicator that an individual has an alcohol problem.

The severity of a number of medical sequelae of alcoholism, such as liver disease and ascites, is related to total lifetime consumption of alcohol.[36,38,44] If one accepts the concept that the severity of liver disease is proportional to the number of abnormal clinical and laboratory findings indicative of this condition,[8,36] then many of the clinical signs and symptoms of liver disease are sensitive only at later stages of alcohol abuse. This situation probably applies to a number of medical concomitants of alcohol abuse that may take 5 to 10 years to develop.[36] Thus, signs of liver disease or neurologic disorders will be present in only a small proportion of younger drinkers or those whose drinking problem is of recent onset.[7]

Clinical and laboratory indicators of alcohol abuse may provide objective information to complement interview data. This is of importance, since reports by the patient and interview data are subject to distortion or denial.[45,46] However, there are problems with the application of biomedical data for the detection of alcohol abuse. Some of the medical and laboratory indicators, such as hepatomegaly and raised serum transaminase levels, are reversible if individuals reduce their alcohol consumption or abstain from drinking. In addition, symptoms and signs and biochemical alterations only represent pathologic responses to excessive alcohol consumption and are relatively nonspecific. There are no specific clinical markers of alcoholism, and "true" biochemical markers of alcohol abuse await development.

Incomplete knowledge of the sensitivity and specificity of many of the laboratory tests used in the detection of chronic excessive alcohol consumption prevents a firm recommendation for the selection of available diagnostic laboratory tests. The diagnostic value of the tests most widely investigated or used is summarized in Table II. Measurement of the MCV and the serum GGT and SGOT levels is likely, taking costs and benefits into consideration, the most appropriate collection of laboratory tests, for increased levels of these variables should provide a strong indication of recent consumption of more than 60 g of ethanol a day.

In conclusion, we are impressed both with the diversity of biomedical and psychosocial disorders related to excessive drinking, and with the real difficulties in isolating specific indicators of the early stages of alcohol abuse. This creates a dilemma. Available evidence suggests that a favourable prognosis is contingent on early identification and treatment. Clearly, a concerted program of research with a multidisciplinary focus is needed if we are to make significant gains in our understanding of the development of alcohol abuse.

We acknowledge the helpful suggestions of our colleagues Drs. Mary Jane Ashley, Laurence Blendis, Harvey Haakonson, Bushan Kapur, Joan Marshman, Hector Orrego, Edward Seliers and Barry Sisson. Also we thank Mary-Jane McNally and Romilla Chhabra for their assistance in preparing this manuscript.

The research for this paper was supported by the Addiction Research Foundation of Ontario. Stephen Holt acknowledges support from the Carnegie Trust for the Universities of Scotland.

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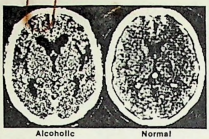

The role of drugs in chronic alcoholism

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Summary

Drugs are certainly not the therapy of first choice in chronic alcoholism. But they can play a significant role in getting patients with anxiety and endogenous depression involved in treatment and keeping them there. Moreover, the threat of a disulfiram reaction, in a well-motivated patient taking that drug regularly, amounts to enforced sobriety, a vital prerequisite to any treatment or rehabilitation program.

Estimates of the number of people in the United States with physical, mental, and/or behavioral problems related to excessive consumption of alcohol vary between 6 and 12 million. Approximately 3% of the work force has drinking problems severe enough to impair job performance; days lost from work because of alcoholism waste millions of dollars annually in wages and productivity. Moreover, since the annual average per capita alcohol consumption is rising, so too is the number of people with such problems.

Patients with alcohol-related problems can be helped by treatment primarily directed at correcting the etiology of the need for alcohol and the secondary consequences of its long-term abuse. Drugs offer an effective therapeutic adjunct, however, helping patients to accept and participate in therapy and maintain their gains once achieved.

Diagnosis of chronic alcoholism is best begun by reviewing the patient's disease history, occupation, employment record, and social interactions. Characteristic clues are sought, such as changes in the individual's health or disruption in family and social life (Table). No single observation during the interview or physical examination is proof of alcoholism, but historic clues and physical signs will often help.[1,2] A

Table: Clues on history for recognition of potential alcohol problems

Alcohol use and related problems	Consumption of >80 gm ethanol/day (12 bottles of beer, 9 oz of spirits, 26 oz of wine); frequent intoxication; blood alcohol >150 mg/dl; withdrawal symptoms; blackouts; physical violence
Family disharmony	Behavioral and/or school problems in children; battered children; psychosomatic complaints, depression, or anxiety in spouse; physical injury to spouse; divorce or separation
Employment record	Fluctuating work pace; loss of sense of responsibility; absenteeism; illness on fixed days; requests for medical certification of illness; drinking on the job; frequent job change
Medical problems caused by alcohol abuse	Cirrhosis; congestive failure (cardiomyopathy); pneumonia; malnutrition; neurologic disorders; pancreatitis; peptic ulcer; recent onset of seizures; physical trauma
Mental problems	Anxiety; depression; drug overdose; suicide attempt
Social changes	Loss of friends; change to drinking friends; loss of recreational interests
Financial problems	Heavy debts; collection agency problems; degenerating living conditions
Accidents and legal involvement	Frequent accidents; impaired driving convictions or charges; motor vehicle accidents; drunkenness charges

full assessment of the alcoholic patient should include extensive and careful interviews with the patient's spouse, children, employer, and anyone else who might be closely involved with him or her. Including these various people in the plans for therapy is also an essential part of the overall treatment program for alcoholic patients.

TREATMENT PLAN

When a person who is physically dependent on alcohol stops drinking, the self-limiting withdrawal reaction seldom lasts longer than 2 weeks.[2] Then the therapy is aimed at correcting the causes that led to the patient's excess intake, reducing his dependency on alcohol, and facilitating his reintegration

into a stable social framework, preferably one including regular employment. Since the causes of chronic alcoholism are many and complex, the therapeutic strategies are also varied and must be adapted to the individual patient.

Pharmacotherapy

Drugs can be invaluable in two major aspects of alcoholic therapy: in alleviation of emotional problems, which are involved in both cause and effect of excessive alcohol consumption, and in the alteration of "chronic alcoholic" behavior.[3] Among the more commonly cited emotional or personality problems said to lead to excessive alcohol consumption are anxiety, depression, low frustration tolerance, latent homosexuality, and immaturity in assessment of long-term consequences. For most of these problems, the treatment, if any, involves various forms of psychotherapy. Drugs are used primarily to alleviate anxiety or depression. They cannot, of course, substitute for psychotherapy, which is aimed at replacing alcohol consumption with more effective problem-solving methods. But short-term (6 to 8 weeks) use of minor tranquilizers and antidepressants can, for example, help inpatients with primary affective disorders to participate in psychotherapeutic and rehabilitative programs.

Anxiolytic drugs. The benzodiazepines—chlordiazepoxide (Libritabs, Menrium), oxazepam (Serax), or diazepam (Valium)—are reportedly superior to placebo with respect to subjective improvement of anxiety or to global rating of patient response over 6 to 8 weeks. Affective changes are minimal, however, and not all studies indicate even short-term effects.

Antidepressants. The three classes of tricyclic antidepressant drugs—the iminodibenzyls, such as imipramine and desipramine (Norpramin, Pertofrane); the dibenzocycloheptenes, which include amitriptyline, nortriptyline (Aventyl), and protriptyline (Vivactil); and the dibenzoxepines, such as doxepin (Adapin, Sinequan)—are effective in the treatment of endogenous depression if given in adequate dosage. (With amitriptyline, for example, patients should receive 50 to 100 mg/day for the first week. Since there is considerable variability in absorption of the drug, the dosage is tailored according to individual needs. The usual daily dose after the first 2 weeks is 75 to 100 mg.) The need for hospitalization, however, is determined by the severity of the endogenous depression, regardless of dosage requirements.

All tricyclics have similar antidepressant activity and would be expected to provide the same symptomatic improvement in chronic alcoholics with endogenous depression—this is the only indication for use of tricyclics in alcoholics. These drugs produce a wide variety of central and autonomic nervous system and cardiac effects, which are particularly important and prevalent in older patients. Among these are the anticholinergic reactions—including dry mouth, urinary retention, constipation, confusion, delirium, hallucinations, bradycardia—and cardiac arrhythmias that have been known to produce sudden death. Thus, dosage must be titrated to achieve relief of depressive symptoms with the fewest pharmacologic side effects.

Neuroleptics. The group of drugs often referred to as major tranquilizers include chlorpromazine (Thorazine), trifluoperazine (Stelazine), thioridazine (Mellaril), and haloperidol (Haldol). They have not proved useful in the rehabilitation of the chronic alcoholic. Occasionally, patients may require these drugs for concurrent primary mental illness or extreme agitation.

Disulfiram. Drinking is discouraged by use of disulfiram (Antabuse), which causes an aversive reaction when alcohol is consumed, thus directly altering the alcohol-dependent patient's behavior. Disulfiram blocks the oxidation of alcohol at acetaldehyde by inhibiting aldehyde-NAD oxidoreductase, an enzyme located in liver mitochondria that mediates the biotransformation of acetaldehyde to acetate. Thus, the concen-

tration of acetaldehyde in the blood may be five to ten times higher than it would be in the normal metabolism of alcohol. Within 5 to 15 minutes of ingesting 0.5 oz or more of spirits, the patient feels warm and a flush begins to develop over the upper chest, neck, and malar region. Pulse pressure and heart rate increase. The reaction may progress to nausea and vomiting, hypotension, severe anxiety, and dyspnea. It may last several hours and has occasionally been fatal.

The alcoholic is encouraged to take disulfiram precisely because of the threat of this reaction. The drug must be taken daily by a willing patient, however, since it loses its effect within several days after being discontinued. Although controlled studies of the drug's action and disposition have not been done, it seems to be effective in reducing the incidence of relapse in some patients. When taken under direct supervision and combined with counseling, maintenance on 250 mg/day may result in better clinic attendance.[4]

The efficacy of disulfiram therapy seems to depend on patient motivation and on the amount and kind of coercion imposed by legal obligation, family members, or employer on the patient to continue taking the drug.

Subcutaneous implantation of disulfiram would seem a reasonable way of ensuring patient compliance. Since it is questionable in some patients whether daily oral dosages of 250 to 500 mg of disulfiram produce an adverse reaction after alcohol, it is puzzling why implants of 500 to 1,000 mg should be effective for up to a month. The pharmacologic actions appear to be minimal; the benefits, mostly psychologic.[4,5]

The most commonly reported side effects of disulfiram—fatigue, morning sleepiness, and drowsiness—are all easily managed by having the patient take the medication immediately before going to bed. Some patients also report apathy, dizziness, impotence, and headaches. Assessment of the drug's adverse effects is difficult, since many of these effects (including peripheral neuropathy, reduced sexual potency, optic neuritis, hepatitis, congestive failure, and myocardial ischemia) may be caused by alcohol or the nutritional deficiencies associated with alcoholism.

Disulfiram also inhibits the biotransformation of warfarin, phenytoin, diazepam, and chlordiazepoxide, potentially causing toxicity from ordinary dosage of these agents. And since it is likely that the biotransformation of other drugs can also be inhibited, patients taking disulfiram should be carefully followed. It is contraindicated in patients with hepatitis, congestive failure, angina, or coronary artery disease. And, if possible, it should not be used in patients with impaired renal or hepatic function, hypertension, primary affective disorders, and a history of drug overdose, as well as those who are known to comply poorly with prescribed instructions.

Citrated calcium carbimide can produce a physiologic interaction with ethanol similar to that of disulfiram, but the reaction is milder, of more rapid onset, and of only half the duration. There are no controlled studies of its efficacy compared to placebo or to disulfiram.

IN CONCLUSION

Drugs play a vital, but secondary, role in the treatment and rehabilitation of chronic alcoholic patients. Informed prescription of appropriate antianxiety and antidepressant agents plus disulfiram can help the well-motivated alcoholic face, and eventually solve, his problem. ∎

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Propranolol-Associated Confused States during Alcohol Withdrawal

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PROPRANOLOL has been used for over a decade as an antiarrhythmic and antianginal agent. Serious central nervous system side effects, in particular, hallucinations, are not common and are usually associated with high doses and extended durations of administration.[1] Greenblatt and Koch-Weser[2] reported 25 adverse reactions in 268 hospitalized medical patients treated with propranolol for angina, hypertension, arrhythmias, and thyrotoxicosis. Eight were life threatening and related to cardiac depression consequent to β-adrenergic blocking activity. In four patients (1.5%), adverse effects involved the central nervous system and included drowsiness, fatigue, lightheadedness, dizziness, headache, nausea, and blurring of vision. No patients experienced hallucinations or other toxic psychotic manifestations. The frequency of delirium tremens is 0.1% in nonmedicated chronic alcoholic patients.[3]

During a randomized double-blind clinical trial to compare the efficacy of propranolol, chlordiazepoxide, and placebo in the treatment of cardiac arrhythmias in alcohol withdrawal (referred to as the arrhythmia study),[4] we noted an exceedingly high incidence (27% of those receiving propranolol alone) of psychotoxic reactions related to relatively small doses of propranolol. One additional patient receiving chlordiazepoxide, 25 mg, four times a day orally, in combination with propranolol also had hallucinations. In the arrhythmia study[4] the four treatment failures in the placebo group and two treatment failures in the chlordiazepoxide group were not associated with delirium or hallucinations.

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Emergency management of alcohol withdrawal

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Since the mean annual per capita consumption of alcohol is increasing in all industrialized countries, physicians can expect to encounter more patients in alcohol withdrawal. The severity of the alcohol withdrawal reaction depends on both the intensity and duration of alcohol consumption. Generally, this withdrawal is mild and usually requires little medical treatment. However, even mild withdrawal may progress to the major withdrawal syndrome of delirium tremens, the mortality of which may be as high as 15%. Morbidity in withdrawal is highest when diagnosis of the syndrome is delayed and when it occurs in patients with other medical or surgical problems.

The proper management of withdrawal reactions depends largely on full assessment and early treatment. Full assessment is intended to detect factors that increase the morbidity of withdrawal (Figure 1), and early treatment is intended to prevent symptoms and signs from progressing to a major reaction (Figure 2). Some complications may be overlooked, while others, such as subdural hematoma, are difficult to diagnose conclusively during withdrawal. The medical and paramedical personnel caring for the alcohol-abusing patient in the emergency department should be sympathetic and noncritical to facilitate full assessment and optimal management.

Clinical profile of alcoholic withdrawal

In the large doses taken by alcoholics, ethanol has a depressant action on the central nervous system. When alcohol ingestion is abruptly decreased or discontinued, it is the compensatory increase in neuronal excitability that produces most of the signs and symptoms characteristic of the alcohol withdrawal reaction.

Primary dependence (alcohol withdrawal syndrome)

There is considerable individual variation in the clinical signs and symptoms of alcohol withdrawal (Figure 2). In mild reactions, the chief symptoms are hyperacuity, hyperactivity of reflexes, tremor, anxiety, insomnia, and reduction of seizure threshold, all of which appear within a few hours after drinking is stopped and last approximately 48 hours. Seizures during withdrawal are typically grand mal, nonfocal, one or two in number, and are most likely to occur between 12 and 48 hours after cessation of drinking. In severe reactions, tremulousness, seizures, auditory and/or visual hallucinations, and global confusion (delirium) are most evident between 48 and 60 hours after withdrawal, but may rarely persist up to 10 days. Low-grade fever (< 38.5 C) is occasionally found in severe withdrawal reactions without apparent

cause; nevertheless meticulous clinical and laboratory assessment must always be made to exclude infection, regardless of the absolute level of temperature.

Secondary metabolic effects

Chronic ingestion of ethanol produces a constellation of predictable secondary metabolic changes (Table 1, column 1). Since most of these changes are alcohol induced, they do not usually require treatment other than supportive care and stopping the consumption of alcohol. The lactic acidemia, hyperuricemia, hypertriglyceridemia, and ketosis are all attributed to oxidation of excess nicotinamide adenine nucleotide dehydrogenase (NADH), which is produced during the conversion of alcohol to acetaldehyde by alcohol dehydrogenase. Insulin therapy is not required for hyperglycemia unless there is a clinically important osmotic diuresis, ketoacidosis, or the patient has a history of diabetes mellitus. On the other hand, severe electrolyte abnormalities should be routinely treated since they may affect the prognosis. If hypoglycemia is suspected, a blood-sugar determination should be made, and 50% dextrose is given (50 ml over 60 seconds). Alcohol-induced ketoacidosis with accumulation of β-hydroxybutyrate, acetoacetate, and lactate develops after several days of heavy drinking, with little or no food, and associated vomiting. When these anions account for the metabolic acidosis, the anion gap ($[Na^+]-([Cl^-]+CO_2)$) will be greater than 15. Arterial blood-gas determinations are necessary to assess the nature and severity of such acid-base abnormalities. Bicarbonate is usually not indicated if the pH is > 7.1 and the actual bicarbonate value is > 15 meq/liter. These patients respond well to solutions of 1N saline and glucose that restore hydration and liver glycogen.

Alcohol-induced disease

After prolonged drinking, the direct toxic effects of alcohol produce various organic disorders (Table 1, column 2). A careful history, obtained from either the patient or a relative, should include the amount and type of alcoholic beverage consumed daily and the duration of its excessive use. An average daily intake above 80 g ethanol is associated with an increased risk of cirrhosis. It is difficult to assess the patient accurately during withdrawal if there is coexistent liver disease (with or without portal encephalopathy), dementia, or other neurologic problems. Excess sedation may precipitate portal encephalopathy, as may the common complications associated with withdrawal—dehydration, electrolyte imbalance, hypoxia associated with pneumonia, infections, and gastrointestinal hemorrhage.

Ascites may be aggravated if saline is aggressively administered to the dehydrated alcoholic with a history or clinical evidence

Figure 1: Etiology of organic diseases in the alcoholic patient

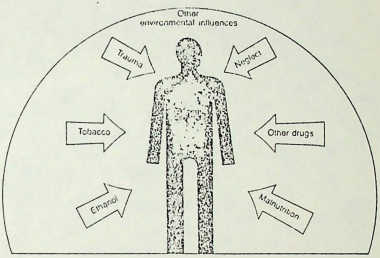
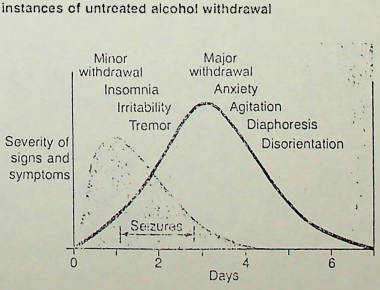


Figure 2: The time course of two typical clinical instances of untreated alcohol withdrawal



of portal hypertension. In these patients, 10% dextran (molecular weight 40,000) in 5% dextrose in water may temporarily correct serious hypovolemia.

Chest pain, arrhythmias, cardiomegaly, congestive heart failure, or combinations of these symptoms may indicate cardiomyopathy. Results of careful examination of the cardiovascular system, including a chest x-ray to assess cardiomegaly and congestive heart failure, an electrocardiogram, and cardiac monitoring, will help determine whether this complication is present.

Alcohol-associated disorders

Many concurrent clinical problems are related to the "life-style" of the alcoholic (Table 1, column 3). For example, hypothermia, which may occur in the alcoholic exposed to cold, can be missed because most clinical thermometers only register 35°C and above. (Electronic thermometers with expandable scales and flexible probe can be invaluable in the hypothermic restless patient.)

Wernicke's encephalopathy (ataxia, ocular palsies, nystagmus) often improves rapidly with parenteral thiamine. Trauma (fractures, visceral injury, head injury, and subdural hematoma) may easily be missed in the patient who is already confused, drowsy, and/or hallucinating. Acute bronchitis, aspiration, or pneumonia is often a more difficult problem to manage in alcoholics who smoke heavily.

Concurrent unrelated disease

Chronic alcoholics may have coincidental diseases that are etiologically unrelated to alcohol consumption and the withdrawal syndrome (Table 1, column 4). Problems arising from the concurrent use of sedatives, tranquilizers, and alcohol often coexist. The alcoholic with diabetes mellitus may have hypoglycemia, hyperglycemia, or diabetic ketoacidosis. Alcoholics with epilepsy may discontinue anticonvulsant therapy and develop status epilepticus. Systolic and diastolic hypertension may subside to normal or mildly elevated values after withdrawal has been accomplished.

Laboratory investigation

Table 2 lists important tests that should

Table 1: Clinical profiles of chronic alcoholic withdrawal

Secondary metabolic effects	Primary alcohol dependence (withdrawal syndrome): Alcohol-induced diseases	Primary alcohol dependence (withdrawal syndrome): Alcohol-associated disorders	Concurrent problems not alcohol related
Electrolyte imbalances	Cirrhosis	Dehydration	Use of sedatives, tranquilizers
Hypokalemia	Ascites ± Encephalopathy ± Varices ± Hepatorenal syndrome	Anemia	Diabetes mellitus
Hypomagnesemia ±	Hepatitis	Malnutrition	Epilepsy
Hypocalcemia	Peptic ulcer	Hypothermia	Hypertension
Metabolic ketoacidosis	GI hemorrhage	Wernicke's encephalopathy	Myocardial infarction
Lacticacidemia	Pancreatitis	Trauma	Other systemic diseases
Overhydration	Cerebellar degeneration	Sepsis	
Hyperglycemia	Dementia	Carcinoma	
Hypoglycemia	Peripheral myopathy	Oropharyngeal	
Hypertriglyceridemia	Cardiomyopathy	Laryngeal	
Hyperuricemia		Bronchial	
		Chronic obstructive lung disease	
		Aspiration pneumonia	
		Tuberculosis	
		Dysrhythmias	
		Subdural hematoma	

Table 2: Clinical workup for assessing patients in alcohol withdrawal

Primary		Additional tests
Barbiturate screening[1]	Urine	Blood may be drawn and stored for later measurement of:
Urine	Analysis	Calcium
	Microscopy	Magnesium
Stool	Occult blood	Amylase
Hematology	CBC and differential	Serum glutamic oxaloacetic transaminase (SGOT)
Biochemistry	Glucose	Serum bilirubin
	Urea	Alkaline phosphatase
	Electrolytes (Na^+, K^+, Cl^-)	Total protein albumin globulin
	Carbon dioxide	Prothrombin time
	Anion gap[2]	Creatinine phosphokinase (CPK)
Arterial	Blood gases	Lactic dehydrogenase (LDH)
X-ray	Skull	Hydroxybutyric dehydrogenase (HBD)
	Bones	Lactate
	Chest	β-hydroxybutyrate
Electrocardiogram		Acetoacetate

1. Other qualitative or quantitative screening of urine or blood is ordered on the basis of clinical assessment (eg, blood ethanol, benzodiazepines, salicylates), provided that facilities are available.
2. Calculated by physician ($[Na^+] - ([Cl^-] + CO_2)$); if CO_2 content or anion gap is abnormal, arterial blood gases should be evaluated.

usually be considered when studying a patient in alcohol withdrawal. Additional helpful diagnostic tests are also cited. The extent to which these patients can be assessed will depend upon the available laboratory facilities, the severity of the withdrawal, and the presence of associated and unrelated clinical problems.

Treatment

Many patients with mild-to-moderate withdrawal reactions can be treated initially in the emergency room and then safely managed at home. Patients with severe uncontrolled withdrawal reactions or complicating problems require hospitalization. General emergency management includes reassurance in surroundings that are well lighted and quiet, monitoring of vital signs as frequently as clinically indicated (eg, cardiac monitoring if there are arrhythmias), hydration, correction of electrolyte abnormalities, and administration of thiamine, 100 mg parenterally.

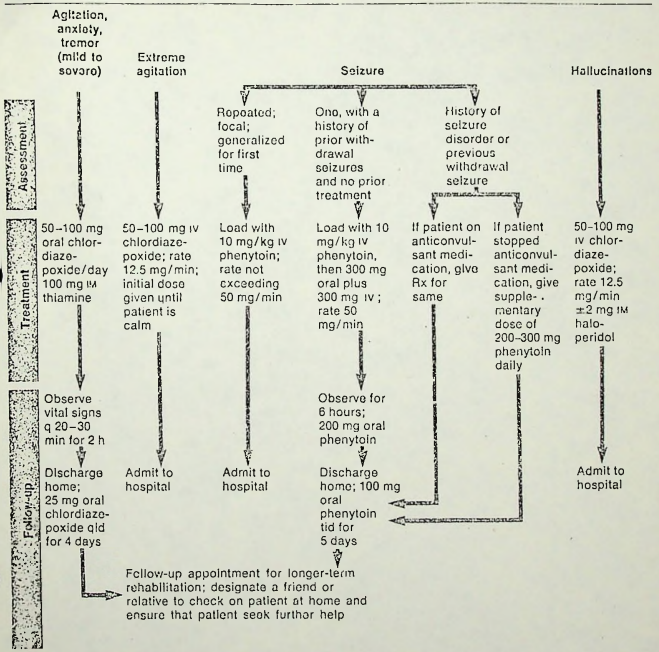
Pharmacotherapeutic objectives

Drug therapy for alcohol-withdrawal reactions is intended to relieve symptoms, prevent or treat more serious complications (eg, seizures, arrhythmias), and prepare the patient for long-term rehabilitation without introducing new drug-dependence problems or therapy-related toxicity.

Various drugs are more effective than placebo for accomplishing these objectives. However, the benzodiazepines have replaced most of the older drugs because of their wide margin of safety. Chlordiazepoxide (Libritabs, Menrium) is the most frequently studied benzodiazepine (although there is no evidence that any one of the benzodiazepines is therapeutically superior to any other). Chlordiazepoxide effectively prevents the reactions from becoming more severe by decreasing anxiety, restlessness, tremor, and the frequency of seizures.

Benzodiazepines are superior to phenothiazines in preventing seizures during withdrawal. (Guidelines for managing seizures are given in Table 3.) There is no direct evidence that the potent antihallucinatory activity of major tranquilizers in schizophrenia has such a specific effect in alcohol withdrawal or on alcoholic halluci-

Table 3: Guidelines for the emergency management of alcohol withdrawal (complete history, physical, laboratory assessment)



nations. Phenothiazines lower the seizure threshold and cause neuroendocrine, dermatologic, and hematologic side effects. Butyrophenones (eg, haloperidol [Haldol]) cause less sedation or hypotension than chlorpromazine and can be reasonably tried for the control of hallucinations, particularly after the risk of seizures has passed. The clinical efficacy of haloperidol can be quite dramatic. Concurrent use of a benzodiazepine will decrease the risk of haloperidol-induced seizures. Acute dystonic reactions, such as oculogyric crisis, may be treated with benztropine mesylate (Cogentin), 2 mg intravenously, followed by 1–2 mg bid orally or parenterally. All patients with hallucinations should be admitted to the hospital.

In general, indications for hospitalization of patients in withdrawal include:

- The presence of a medical or surgical condition requiring treatment (hepatic decompensation, infection, dehydration, mal-

nutrition, cardiovascular collapse, cardiac arrhythmias, trauma)

- Hallucinations, tachycardia > 100 beats/minute, severe tremor, extreme agitation, or a history of severe withdrawal symptoms
- Fever > 38.5 C
- Wernicke's encephalopathy (confusion, ataxia, nystagmus, and ophthalmoplegia)
- Confusion or delirium
- Seizures: Generalized seizure occurring for the first time in the withdrawal state, focal seizures, status epilepticus, seizures in patients withdrawing from a combination of alcohol and other drugs
- Recent history of head injury with loss of consciousness
- Social isolation

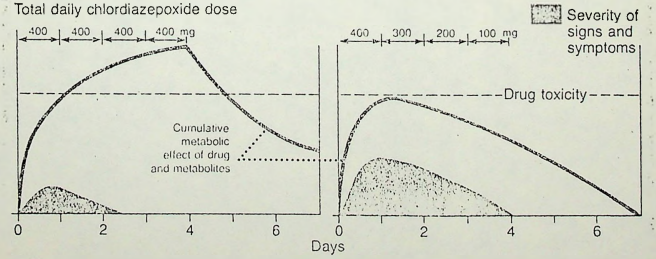
Patients in status epilepticus should be treated initially with 5–10 mg diazepam (Valium) IV as needed, at a rate of 2.5 mg/minute, until seizures are controlled. Equipment for maintenance of an airway and for mechanical support of ventilation must be immediately available. Subsequent doses of 5–10 mg every 20–30 minutes IV as needed, may be given if seizures recur. Appropriate maintenance therapy with other agents should be instituted promptly. Seizures require treatment if they are repeated, continuous, or life threatening. However, there is uncertainty about the therapeutic and prophylactic value of phenytoin in alcohol-withdrawal seizures. Phenytoin should be given orally or intravenously, since it is poorly absorbed from intramuscular injection sites.

Intravenous phenytoin is infused directly. The loading dose is 10 mg/kg, and the oral maintenance dosages are 100 mg tid (Table 3). Phenytoin need not be continued past the withdrawal period except in patients with a pre-existing seizure disorder. Patients withdrawing from a combination of alcohol and other drugs, particularly barbiturates and nonbarbiturate hypnotics, should also be hospitalized, since withdrawal seizures from more than one drug can be more serious and difficult to manage elsewhere.

Drug accumulation

Since chlordiazepoxide and diazepam are both long-acting drugs with pharmacologically active metabolites, repeated daily dosages allow either the drug and/or its metabolite to accumulate, and desired therapeutic (or unwanted toxic) effects may not appear until several days of continuous therapy (Figure 3). Some drowsiness may be of therapeutic benefit, but if dos-

Figure 3: Effect of chlordiazepoxide on clinical course of withdrawal reaction



Left panel indicates the slow cumulative pharmacologic effect of chlordiazepoxide and its active metabolite, desmethylchlordiazepoxide, during repeated daily administration of the same dose. Maximum sedative effects may only be seen after the withdrawal period.

Right panel shows chlordiazepoxide dose to avoid excessive sedation.

ages are not titrated against the clinical state of the individual patient, excessive drowsiness, lethargy, ataxia, diplopia, confusion, respiratory depression, and increased risk of aspiration may follow. To circumvent the consequences of drug cumulation, doses should usually be reduced progressively (Figure 3, right panel). On the first day of treatment, large doses of chlordiazepoxide in the range of 100–100 mg should be given. (Occasionally, doses as high as 1600 mg may be required; this situation is usually associated with delayed treatment.) Thereafter, smaller doses, approximately 25% less than the initial dose, are given daily if required. There

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cannot be a "standard" or "routine" dosage schedule because of the variability in the severity of withdrawal symptoms, the metabolic fate of the drugs, and the presence of other diseases.

Chlordiazepoxide and diazepam are absorbed slowly and incompletely from intramuscular injection sites. When a rapid and predictable clinical effect is required, the oral or intravenous route is preferred. Smaller doses should be given to patients with severe liver disease and/or low serum albumin, since the concentrations of free active chlordiazepoxide and diazepam will be higher, and diazepam is metabolized more slowly in cirrhosis.

In conclusion

Considerable clinical skill and attention are required for the management of alcohol withdrawal. Optimal management of the alcohol-abusing patient includes an adequate opportunity for long-term rehabilitation. ■

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0271-0749/82/0204-0274$02.00/0
Journal of Clinical Psychopharmacology

Vol. 2, No. 4
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TABLE 1. Contributions of individual score components to aggregate score of 66 selected severity assessments in five patients*

Score component	Rank	Total for component	Occasions observed	Observations in which symptom seen (%)	Contribution to aggregate score (%)
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*Total scores of individual subjects (aggregate = 738): 1 (240), 2 (61.5), 3 (135.5), 4 (143), 5 (158).

TABLE 2. Characteristics of the five chronic alcoholic patients

	Patients					Mean
	1	2	3	4	5	
Age	29	40	45	47	51	42.4
Weight (kg)	56.5	67.0	55.0	80.0	76.4	67.9
Years of alcohol abuse	10	20	23	20	30	20.6
Duration of this binge (days)	10	7	21	42	84	32.8
Daily consumption (g/day)	338	170	170	230	230	228.0
Equivalent spirits (oz/day)[a]	30	15	15	20	20	20.0
Admission withdrawal score[b]	35	22	28	23	30	27.6
Admission blood alcohol (mg/liter)[c]	150	730	230	300	300	342

[a] 40% ethanol, volume for volume, minimum concentration in Canadian spiritous liquor.

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Results

The individual data for withdrawal severity and the plasma concentrations of the drug in each subject are given in Figure 1; the mean withdrawal scores across subjects are given in Figure 2. As shown, a slight but statistically nonsignificant decrease in withdrawal severity occurred from the initial assessment until that obtained at the start of the placebo infusion. By contrast, however, the withdrawal score dropped significantly over an equivalent period of time during the saline infusion phase from 23.3 to 8.9. A further but smaller drop in the withdrawal score was observed after the infusion was changed to chlormethiazole; however, by the time the drug was begun, the intensity of withdrawal had abated substantially.

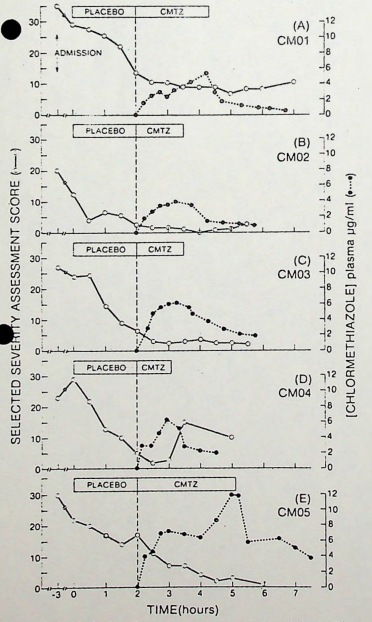

FIG. 1. Individual selected severity assessment scores and plasma chlormethiazole (CMTZ) levels for all five subjects. Horizontal bars indicate duration of intravenous infusions.

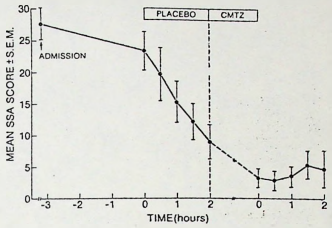

FIG. 2. Mean selected severity assessment (SSA) scores for five subjects. Horizontal bars indicate duration of intravenous infusions. CMTZ = chlormethiazole.

As shown in Figure 1, there was a rapid rise in plasma drug concentration to a peak between 57 and 180 min. Four of five (CM01 to CM04) subjects experienced moderate drowsiness within 2 hours (they could still be aroused), which quickly dissipated as the plasma concentration of the drug rapidly fell (half-life, 0.67 hours; apparent terminal half-life, 1.80 hours).

Discussion

In this investigation, the development and application of the modified scoring system provided an important tool for selecting a homogeneous group of patients according to withdrawal severity and for assessing, repetitively, the course of withdrawal severity in a controlled clinical trial. Such an instrument played an important role in this study because it assured that only patients suitable for the study were entered into the infusion phase of the investigation. Many of the potentially suitable patients who were initially assessed as being in severe withdrawal were found to have low withdrawal scores just prior to the saline infusion and therefore were rejected. In addition, it has been possible to adapt the alcohol withdrawal scoring system for the accurate and reliable monitoring of the sedative withdrawal syndrome.[11]

An unexpected but exceedingly important result of this investigation was the demonstration of the precipitous decrease in withdrawal severity unaided by active pharmacotherapy. Such an observation has important implications for alcohol withdrawal treatment. In the first place, it established that, in selected patients, supportive care and nonpharmacological treatment may be entirely sufficient for the management of acute alcohol withdrawal. While admittedly the influence of supportive care cannot be dissociated from the saline placebo treatment

in this study, other investigators[12] have stressed the impact of supportive care and reality orientation in the detoxification of alcoholics. Secondly, the observation of the potent influence of nonpharmacological measures underlines the considerable lability of the withdrawal syndrome in clinical trials and should warn investigators of the potential problems that may occur in the interpretation of results if adequate considerations are not taken of the experimental design. In our own case it was felt that the reduction of withdrawal symptomatology was sufficiently impressive so as to preclude continuation with a fully blended counterbalanced design using drug therapy first. For the future, however, this study has laid the basis for the conduct of controlled investigations into the efficacy of supportive care in the treatment of unselected withdrawal patients. While the aim of this investigation was to provide an ideal experimental situation for the elucidation of the influence of drug therapy in withdrawal, it was found that, in doing so, a select population of alcoholics was perhaps isolated. The success of nonpharmacological treatment by supportive care of such individuals provides an exciting counterpoint to conventional treatment and establishes a sound basis for the ethical use of such techniques in certain settings. Such supportive care management is, however, very intensive and may not prove to be cost-effective in some centers where drug treatment may be better applied. What is important, however, is that individualized concern and care are important for withdrawing alcoholics, and while it may somewhat confound the ability to quantify the exact value of a current therapy, the benefit to the patient is apparent.

Acknowledgments

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0271-0749/82/0204-0274$02.00/0
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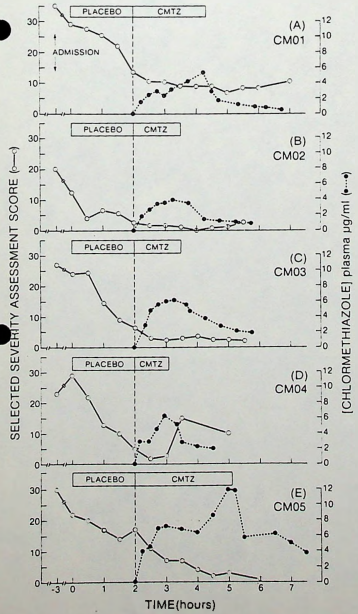

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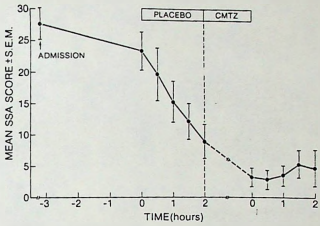

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The authors would like to thank the nursing staff of the Clinical Research Unit and Dr. P. Iversen and staff of the Emergency Department, Clinical Institute, Addiction Research Foundation; Dr. E. Monkman and staff of the Emergency Department, Toronto Western Hospital; Dr. G. Hastie and staff of the Emergency Department, Wellesley Hospital, for their professional assistance in the study; and Ms. Cathy Van Der Giessen for her typing assistance.

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0271-0749/82/0204-0277$02.00/0
Journal of Clinical Psychopharmacology

Printed in U.S.A.

questions of how they were understood by their readers and what effects they might have had. As far as I am aware, no attempt has been made to look at these fundamental issues. In view of the ambiguous and often discouraging results of previous drug education programmes, the Government would be ill-advised to proceed on the assumption that any factually correct information is necessarily going to help matters. How will we know if these leaflets have helped or not? Would it not have been useful to pilot them on selected target groups and to monitor their influence?

It is perhaps this failure of planning that represents one of the weaknesses of the leaflets. Like it or not, drug problems are going to be with us for many years to come. It is laudable that the Government has correctly identified the matter as worth serious attention. It would be a political failure of some magnitude if they now failed to act as if it were a serious problem. It is unfortunate that the national response should have to be implemented in an unplanned, last-minute rush. Let us give two cheers for the leaflets. But let us also hope that the Government will put its money where its political mouth is and fund the research necessary for an effective response.

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Britsh Journal of Addiction 80 (1985), 227–232

Medical Review Series 5
Alcohol and Fits

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Summary

This review indicates the way in which epilepsy and excessive ethanol intake are related and the frequency of each in relation to the other. There are many ways that fits may be precipitated, both during intoxication and during recovery from ethanol excess. Continuing anticonvulsant therapy is not usually indicated. The importance of abstinence is emphasised if fits occur.

Introduction

Fits are only one of many neurological disorders which may occur in relation to alcoholism [1, 2]. They are frequent in alcoholics and proper management depends not only on controlling the fit but also upon recognising the contribution of excess ethanol intake and managing that problem too. The relationship is one which has been appreciated for centuries. William Buchan, for example, published a treatise 'Domestic Medicine' in 1783 [3]. This popular book ran to 17 editions in the next 17 years. He wrote:

> 'Epilepsy is sometimes hereditary. It may likewise proceed from blows, bruises or wounds on the head, a collection of blood, or serious humours in the brain, tumours or concretions within the skull. *Excessive drinking*, contagion received into the body as by the infection of the smallpox, measles etc, hysterical affections . . .' [3]

A medical student in 1983 might well satisfy his examiners if he produced a modern version of this list of differential diagnoses, but he would not be expected to include Buchan's other causes of epilepsy: intense study, excessive sexual activity and suppression of customary evacuations . . .! In spite of such popular accounts, Sir William Gowers, the eminent London neurologist, writing at the end of the 19th century, was much less definite about the relationship. In a two volume treatise 'Diseases of the Nervous System' he wrote one line on the matter, commenting that epilepsy is occasionally an effect of alcohol excess, usually accompanying delirium tremens [4].

In a small group of patients, alcoholism may be a direct result of epilepsy. The latter disorder may cause conflicts and personality disorders which lead to drinking ethanol in excess (in this review the terms 'ethanol' and 'alcohol' are used synonymously). This possibility is obviously difficult to assess in practice and I shall not discuss it further.

Fits may occur in those with primary epilepsy who drink excessively, and in alcoholics without other evidence of epilepsy, either during a drinking bout or in the period of withdrawal and recovery from the excess alcohol. A flow chart showing the causes of fits in relation to drinking alcohol is given in Figure 1.

Primary epilepsy and ethanol excess

What is the frequency of excess ethanol intake in epileptics? Our own observations in Wellington answer this question and our findings are very similar to those reported from other countries. We have recently carried out a study of patients who have had a fit and been collected by the ambulance service in the Wellington region in a year. One hundred consecutive patients were investigated by means of a questionnaire and personal interview. Patients were asked whether they drank ethanol and, if so, how much,

This paper is based on an Invited Lecture given to the Annual Conference of The New Zealand Medical Society on Alcohol and Alcoholism

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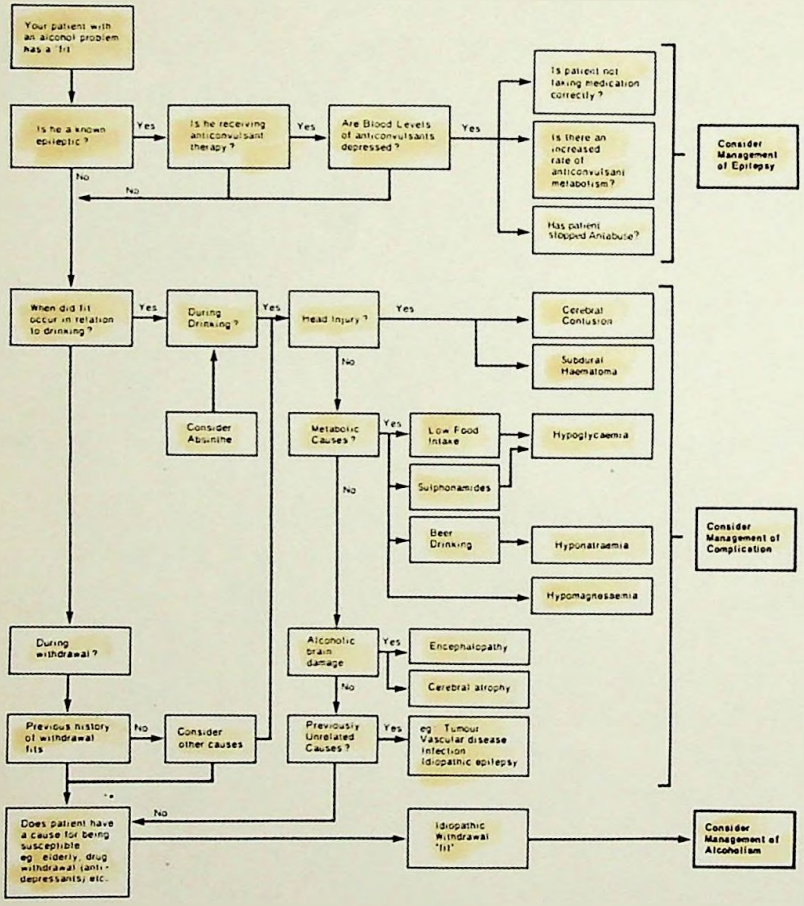

with what frequency, and whether they had drunk ethanol within the 12 hours prior to their seizure. Those subjects with a history suggestive of alcoholism were excluded so that as far as possible the subjects surveyed had primary epilepsy. They were also questioned on the nature of any advice they had received about drinking ethanol in relation to either their fits or their medication, if any, and what they did about taking their medication when they had been drinking. The answers to the latter questions will be considered later. In the study we found that 20 per cent of the epileptics interviewed drank regularly at least 80g of ethanol in the course of a day. Fourteen per cent of the epileptics did so once weekly or more, an extent which rendered effective control of the seizures by drug therapy unlikely. Twelve per cent of seizures were probably precipitated by ethanol in patients who could not be said to have a major drinking problem. In a national survey of New Zealanders aged 14–65 years 6 per cent of the total population were estimated, on the basis of their self report, to consume 80g or more of absolute alcohol per day [5].

Other surveys have also demonstrated a high percentage of epileptics whose frequency of attacks is likely to be influenced by ethanol. The range in published reports is very wide. One found 8 per cent of epileptics were drinking excessively, another reported as many as 35 per cent [6, 7]. In Tasmania, a study of epileptics applying for a driving licence revealed 8 per cent alcoholism as the result of self admission or through police reports [8]. In a survey carried out in Finland, of 560 consecutive seizure patients brought to an emergency department of a hospital in Helsinki during the course of the year, 277 were found to have ethanol intoxication in their immediate history [9]. In this group, ethanol provoked seizures occurred mainly on Sunday and Monday following the weekend pattern of alcohol consumption. It preceded delirium tremens in 21 patients. This series also highlighted the importance of head trauma as a precipitating feature of seizures in alcoholics — a point to which I shall return.

Why do fits occur in epileptics with ethanol excess?

Failure to take medication. Heavy drinkers who are also epileptics on anticonvulsants, frequently do not take their medication on the evening they drink heavily, or on the morning after. Some are forgetful; memory difficulties are common problems with alcoholics both during intoxication and between drinking periods, and others may decide not to do so. In our Wellington series of 100 patients, six of those who drank ethanol in excess replied that they had deliberately stopped taking their drugs when they knew that they would be drinking. One reason may have been the way they had been given advice. Their doctor may have said — 'when you are on anticonvulsant drugs you are not to drink'. The doctor will not have said the patient could drink ethanol if he stopped taking the drugs, but the patient may have interpreted his words in that way.

Depression of blood concentrations of anticonvulsants by ethanol. Ethanol may increase the clearance of anticonvulsants from the blood [10, 11]. Ethanol induces microsomal enzyme activity in the liver leading to shorter half lives of many drugs in the body, together with lower blood levels. The daily intake of ethanol required to stimulate liver enzymes is large, but it is within the range consumed by many who regularly imbibe. About two-thirds of those consuming alcohol in the range of 70–100g daily have the potential of increased clearance rates of drugs and therefore, if they are on anticonvulsants, blood levels of these drugs will fall and seizure activity may be unmasked. [12]. There is evidence that this also occurs in the withdrawal period [13].

Disulfiram (Antabuse), used to strengthen the resolve of the alcoholic by producing a violently unpleasant reaction when it is taken in association with ethanol, has an additional advantage in epileptic alcoholics also taking anticonvulsants. The drug is a non-specific inhibitor of microsomal drug metabolism in the liver. It therefore decreases anticonvulsant clearance and counteracts the effect of ethanol [14]. If, however, the patient taking Antabuse is epileptic and is on anticonvulsants, his blood levels will be maintained by the drug. Should he stop taking it abruptly, worse still should he also start drinking again, his anticonvulsant blood levels will fall dramatically and he is at risk of unmasking his epilepsy.

Precipitating factors during withdrawal. A number of features may be involved in precipitating a seizure in known epileptics. Why seizure threshold is depressed during withdrawal is discussed in the next section.

Alcoholics without previous epilepsy who develop seizures.

What is the frequency of seizures in alcoholics?
A study of 566 patients admitted with acute or subacute alcohol toxicity to a detoxification unit in the U.S.A. showed that, in those who had not been previously admitted, a third had a seizure as one of the presenting symptoms of the admission. Among those who had been admitted previously, a similar proportion had a seizure when they were admitted during the study period, even though many of these had been put on anticonvulsants [15]. These findings may overstate the usual proportion. They were from a population of mixed ethnic background which was predominantly from a low socioeconomic area: in addition, alcoholics admitted to a detoxification unit are likely to be severe alcoholics. A current review of several surveys has suggested that 5 per cent of all alcoholics will have a fit at some stage, depending upon the severity of the alcoholism, the duration of the disease, and the type of beverage consumed [16].

Why do fits occur?

Effect of head injury. Some alcoholics are at risk of developing fits because they have suffered injury to the brain, either acutely during the relevant period of intoxication, or due to previous injury. In over one third of a group of patients in Helsinki who developed fits provoked by alcohol intoxication and were taken to hospital, a contributory cause of the fits was head injury. In many this had been while they were intoxicated, but in some the history of head injury had been earlier and a few of these were found to have a subdural haematoma [8]. Subdural collections of blood should always be considered in this group of patients.

Due to alcoholic brain disease. Epilepsy may occur as evidence of severe ethanol brain damage, including encephalopathy. This is particularly likely in patients who have terminal lever cirrhosis. A further organic cause for the development of epileptic attacks in alcoholics is brain atrophy, which is frequently found in patients with a long history of alcoholism. As a result of the atrophy there may be structural changes which may induce epileptic seizures. In a computerised tomography study of alcoholics, 41 per cent of those with chronic epilepsy had cerebral atrophy compared with 22 per cent of those who only had seizures associated with ethanol withdrawal. Another theory of seizure development without obvious cause, in alcoholics, is that 'kindling' occurs: subliminal repeated stimulation by ethanol abuse eventually resulting in a convulsion [17].

Due to metabolic complications. Hypoglycaemia may develop during prolonged periods of ethanol intake in patients who do not have adequate caloric intake. It may unmask latent epilepsy. Hypoglycaemia may also be precipitated when ethanol is taken in association with some drugs, for example sulphonamides.

Low blood magnesium concentrations [17, 18] may occur during intoxication or withdrawal. Hypomagnesaemia has an excitory effect on the central nervous system and could influence fit development in susceptible individuals. Hyponatraemia, resulting from the excessive water intake of some beer drinkers, may have similar effects.

Due to ingestion of a convulsant substance with ethanol. Absinthe drinkers may develop convulsions due to a convulsant substance, Thujone, normally present in the drink.

As a result of withdrawal from ethanol. Such fits used to be called 'Rum fits' but are now normally known as withdrawal fits. The initial seizure usually occurs after the age of 40 when the individual has been suffering from alcoholism for at least five years. The elderly are particularly prone to withdrawal seizures [19]. This may be partly because the rate of ethanol metabolism decreases with age and the aged are therefore at risk of developing very high ethanol blood levels for a long period. Seizures and delirium tremens may be diagnosed as complications of psychosis, encephalitis, subarachnoid haemorrhage or ischaemic stroke in elderly patients. Withdrawal fits are common. In a series which we have studied in Wellington of patients during withdrawal from ethanol dependence, out of 27 treated symptomatically, but without anticonvulsants, five patients had seizures, being just under 20 per cent [20]. Twelve per cent of patients admitted to hospital for detoxification developed seizures in another series [21]. Seizures are particularly likely to occur in the preliminary stage of delirium tremens.

During ethanol withdrawal, the majority of seizures occur from 12–36 hours after the cessation of ethanol intake [22]. Fits which occur are usually grand mal seizures without any focal symptoms, unless there is a specific localised cerebral lesion, as already discussed. Tetany-like spasms have been described in alcoholics during withdrawal and may be confused with focal epilepsy [23]. About 25 per cent of patients who progress to status epilepticus have excessive ethanol intake as a predisposing factor [24]. There has been argument about whether fits occurring only during ethanol withdrawal should be considered true epilepsy. Some alcoholics only have fits then, and never have epilepsy at any other time in their lives. Although prophylactic anticonvulsants frequently abolish the development of seizures, it is now not usual to consider that such patients are true epileptics or that they require continuing management of fits. The random electroencephalogram (EEG) is usually normal in those patients who suffer from withdrawal seizures, whereas fits that occur during the period of actual drinking are more likely to be related to evidence of epileptic activity, either because the epilepsy predated the development of the alcoholism or because it resulted from a problem such as head trauma. Further evidence that seizures occurring during withdrawal are not true epilepsy is the observation that sleep deprivation rarely provokes focal abnormalities or paroxysms in the EEGs of this group of patients. However, in those with predisposing features for epilepsy, EEG abnormalities are much more frequent, particularly after sleep deprivation [25].

The underlying cause of withdrawal fits is sometimes definable but, in many cases, there is no obvious cause and a number of mechanisms have been proposed, which are noted below:

Withdrawal of other depressants. If a patient is on a depressant drug such as a benzodiazepine, then he may fail to take it during and after a period of ethanol excess, and a seizure may occur as a rebound phenomenon. [26, 27].

GABA depression in the brain. Ethanol may cause a fall in gamma-aminobutyric acid (GABA) levels within the brain. This has been observed in experimental animals. It may explain the effectiveness of sodium valproate (Epilim) treatment of withdrawal seizures as it is a drug which elevates brain concentrations of GABA [28].

Fluid retention. There is increased blood vasopressin during alcohol withdrawal [29]. This may result in an increase in fluid retention and eventually in cerebral oedema: the 'wet-brain' syndrome in which fits are a common symptom.

Alteration of electrical activity in the brain. There may be depression of normal rapid eye movement (REM) sleep and, in a few patients, sleep deprivation: both may trigger epileptic seizure activity. The possibility of 'kindling' of epilepsy has already been discussed.

Altered dopaminergic activity. Impaired central dopaminergic activity has been implicated in photosensitive epilepsy. Dopaminergic neurotransmission is also impaired in the alcohol withdrawal period and this may therefore contribute to the occasional occurrence of television induced seizures as a manifestation of alcohol withdrawal [30].

Management

Alcoholics frequently deny their pathological drinking. If they then develop fits during withdrawal they may run the risk of being labelled as epileptics, with treatment being addressed to the management of epilepsy rather than to that of their alcoholism.

As long as the results of investigations for organic causes of epilepsy are negative, therapy for fits in association with alcoholism should be directed first towards the alcoholism, and complete abstinence should be achieved if at all possible. Non-abstinent patients have a high record of non-compliance in taking drugs, so that long-term administration of anticonvulsant drugs is not likely to be fully effective. A common therapeutic technique is to give anticonvulsants during withdrawal periods for, say, up to a week after cessation of drinking. Among the drugs which have been used is sodium valproate [19, 31], although this should be avoided if there is evidence of liver damage as it may aggravate this. Phenytoin and carbamazine have also been used [24, 32, 33]. When an epileptic is found to require a large dose of anticonvulsant to achieve therapeutic drug levels this may be a clue to unreported ethanol consumption affecting the metabolism of the drug, as already discussed. The value of anticonvulsants is still debated. Some consider that anticonvulsants are not necessary as adequate treatment with drugs used for the treatment of withdrawal [34] such as chlordiazepoxide (Librium) or other depressant drugs should alleviate fit frequency [35]. Further studies are still required.

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British Journal of Addiction 80 (1985), 233–243

Morbid Cravings: The Emergence of Addiction

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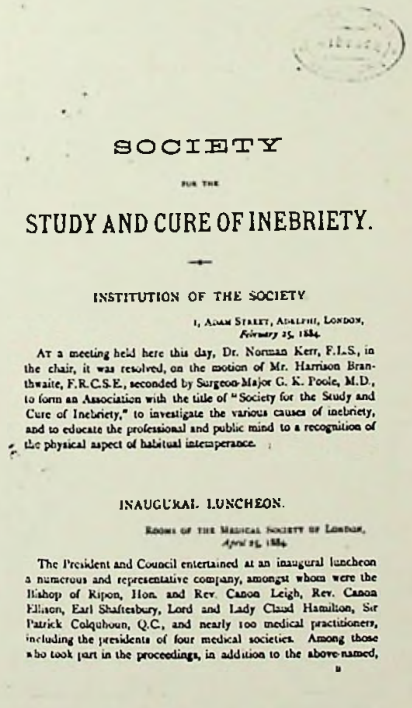
Few readers of the *British Journal of Addiction* can now be unaware that 1984 was the centenary year of the Society for the Study of Addiction. A centennial symposium was held at the Royal Society in October 1984; the centenary issue of the *Journal* appeared and an historical conference on alcohol and alcoholism was held in November. The centenary was also marked in a visual way by an exhibition held at the Wellcome Institute for the History of Medicine. Arising out of an approach by Professor Malcolm Lader, President of the Society, to the Wellcome Institute, the exhibition, entitled 'Morbid Cravings: The Emergence of Addiction' was organised by a small group representing both the Society and the Institute. William Schupbach, curator of the Wellcome Iconographic Collections, Julia Sheppard, archivist of the Contemporary Medical Archives Centre, came from the latter, Malcolm Lader and myself from the Society, Robin Price, Deputy Librarian of the Wellcome Institute both co-ordinated and actively participated in the group.

The exhibition had a certain timeliness in another respect in that it coincided with an upsurge of media interest in the 'heroin problem' and political recognition of public concern through the injection of funds into regional drug teams. However, the 'dangerous drugs' were by no means the only addictive substances covered in the exhibition. Addiction to tobacco, opium, cocaine, medically prescribed drugs such as chloral hydrate, the barbiturates and amphetamines, the 'gentle poisons' of tea, coffee and chocolate, and historically and currently, the chief 'problem drug' of them all, alcohol — were all covered. Other organisations and institutions generously provided items. The Pharmaceutical Society, for example, lent a selection of chlorodyne advertisements and the University of London Library provided a copy of the 1920 Dangerous Drugs Act. But the bulk of the material on display came either from the library of the Wellcome Institute itself, or from the vast collection of medically related items initiated by Sir Henry Wellcome, originally housed in a medical museum in the Euston Road building, but now on permanent loan to the Science



SOCIETY

FOR THE

STUDY AND CURE OF INEBRIETY.

INSTITUTION OF THE SOCIETY

1, Adam Street, Adelphi, London, *February 25, 1884.*

At a meeting held here this day, Dr. Norman Kerr, F.L.S., in the chair, it was resolved, on the motion of Mr. Harrison Branthwaite, F.R.C.S.E., seconded by Surgeon-Major G. K. Poole, M.D., to form an Association with the title of "Society for the Study and Cure of Inebriety," to investigate the various causes of inebriety, and to educate the professional and public mind to a recognition of the physical aspect of habitual intemperance.

INAUGURAL LUNCHEON.

Rooms of the Medical Society of London, *April 25, 1884.*

The President and Council entertained at an inaugural luncheon a numerous and representative company, amongst whom were the Bishop of Ripon, Hon. and Rev. Canon Leigh, Rev. Canon Ellison, Earl Shaftesbury, Lord and Lady Claud Hamilton, Sir Patrick Colquhoun, Q.C., and nearly 100 medical practitioners, including the presidents of four medical societies. Among those who took part in the proceedings, in addition to the above-named,

Figure 1. Proceedings of the Society for the Study and Cure of Inebriety. 1884, 1(1) p. 1. Institution of the Society.

Museum. The exhibition organisers were embarrassed for choice. Julia Sheppard, dealing with tobacco, had particular problems in choosing from the vast Science

used as one filter in deriving short-form-ADD, best separated 'alcoholics' and 'regular drinkers' with items tapping cognitive and to a lesser extent behavioural events, rather than by items associated with more severe dependence such as withdrawal phenomena. A Guttman analysis of short-form-ADD would be useful to test for a hierarchical relationship between cognitive, behavioural and physiological questions.

Short-form alcohol dependence data questionnaire (SADD)

The following questions cover a wide range of topics to do with drinking. Please read each question carefully but do not think too much about its exact meaning. Think about your MOST RECENT drinking habits and answer each question by placing a tick (√) under the MOST APPROPRIATE heading. If you have any difficulties ASK FOR HELP.

	Never	Sometimes	Often	Nearly always
1 Do you find difficulty in getting the thought of drink out of your mind?	...	...	...	...
2 Is getting drunk more important than your next meal?	...	...	...	...
3 Do you plan your day around when and where you can drink?	...	...	...	...
4 Do you drink in the morning, afternoon and evening?	...	...	...	...
5 Do you drink for the effect of alcohol without caring what the drink is?	...	...	...	...
6 Do you drink as much as you want irrespective of what you are doing the next day?	...	...	...	...
7 Given that many problems might be caused by alcohol do you still drink too much?	...	...	...	...
8 Do you know that you won't be able to stop drinking once you start?	...	...	...	...
9 Do you try to control your drinking by giving it up completely for days or weeks at a time?	...	...	...	...
10 The morning after a heavy drinking session do you need your first drink to get yourself going?	...	...	...	...
11 The morning after a heavy drinking session do you wake up with a definite shakiness of your hands?	...	...	...	...
12 After a heavy drinking session do you wake up and retch or vomit?	...	...	...	...
13 The morning after a heavy drinking session do you go out of your way to avoid people?	...	...	...	...
14 After a heavy drinking session do you see frightening things that later you realize were imaginary?	...	...	...	...
15 Do you go drinking and next day find you have fogotten what happened the night before?	...	...	...	...

Acknowledgements

Special thanks are given to Prof. Griffith Edwards and Dr Bram Oppenheim who have given advice, criticism and support throughout this project. Thanks are also due to John Bartle for his help in selecting the final ADD questions, and to the Addiction Unit nursing staff for their conscientious collection and scoring of questionnaires through several phases of development. Finally an appreciation to Margrit Salter for her careful secretarial work on the paper.

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alcoholics in a setting different from that in which it was developed. Based on the data reported here, we feel that subjects with scores ranging from 1–9 should be classified as non-dependent or low dependent instead of only low dependent, as proposed by Raistrick *et al.* [6].

Our alcoholic population fell into the medium or high dependent categories, and provides further support for Hodgson *et al.*'s [13] assertion that not all people admitted to alcohol treatment units are severely dependent, although they share a range of complaints, problems and disabilities caused by drink.

From the present study we were unable to analyse whether alcoholics attending an out-patient service differed in severity from those being treated in an in-patient unit, or whether social class was related to severity of alcohol dependence. We hope to address these questions in a further study.

Appendix 1. Short-form alcohol dependence data questionnaire (SADD) as described by Raistrick, *et al.* [6]

The following questions cover a wide range of topics to do with drinking. Please read each question carefully but do not think too much about its exact meaning. Think about your MOST RECENT drinking habits and answer each question by placing a tick (✓) under the MOST APPROPRIATE heading. If you have any difficulties ASK FOR HELP.

	Never	Sometimes	Often	Nearly Always
1. Do you find difficulty in getting the thought of drink out of your mind?	....	....	....	....
2. Is getting drunk more important than your next meal?	....	....	....	....
3. Do you plan your day around when and where you can drink?	....	....	....	....
4. Do you drink in the morning, afternoon and evening?	....	....	....	....
5. Do you drink for the effect of alcohol without caring what the drink is?	....	....	....	....
6. Do you drink as much as you want irrespective of what you are doing the next day?	....	....	....	....
7. Given that many problems might be caused by alcohol do you still drink too much?	....	....	....	....
8. Do you know that you won't be able to stop drinking once you start?	....	....	....	....
9. Do you try to control your drinking by giving it up completely for days or weeks at a time?	....	....	....	....
10. The morning after a heavy drinking session do you need your first drink to get yourself going?	....	....	....	....
11. The morning after a heavy drinking session do you wake up with a definite shakiness of your hands?	....	....	....	....
12. After a heavy drinking session do you wake up and retch or vomit?	....	....	....	....
13. The morning after a heavy drinking session do you go out of your way to avoid people?	....	....	....	....
14. After a heavy drinking session do you see frightening things that later you realize were imaginary?	....	....	....	....
15. Do you go drinking and next day find you have forgotten what happened the night before?	....	....	....	....

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The CAGE Questionnaire: Validation of a New Alcoholism Screening Instrument

BY DEMMIE MAYFIELD, M.D., GAIL MCLEOD, M.S.W., AND PATRICIA HALL, M.S.W.

The CAGE questionnaire, a new brief alcoholism screening test, was administered to all patients (N = 366; 39 percent alcoholic) admitted to a psychiatric service over a one-year period. The authors indicate that the CAGE questionnaire is not a sensitive alcoholism detector if a four-item positive response is the criterion; however, if a two- or three-item criterion is used, it becomes a viable rapid alcoholism screening technique for large groups.

A NUMBER of investigators have attempted to devise a viable screening questionnaire for the detection of alcoholism (1, 2). None of these questionnaires meets all the requirements of brevity, ease of administration, sensitivity, and validity desired in such an instrument. Ewing and Rouse (3) have developed a questionnaire that promises to correct many of the shortcomings of previous alcoholism screening instruments. The CAGE questionnaire, consisting of four questions of a nonincriminating nature, appears in preliminary studies to be a sensitive indicator of covert problem drinking. We conducted the following study in order to further evaluate the usefulness of the CAGE questionnaire.

At the time this work was done, the authors were with the Veterans Administration Hospital, Durham, N.C., where Dr. Mayfield was Assistant Chief, Psychiatry Service, and Mses McLeod and Hall are staff social workers. Dr. Mayfield is now Chief, Psychiatry Service, Veterans Administration Hospital, Providence, R.I. 02908, and Professor of Psychiatry, Brown University Program in Medicine, Providence.

METHOD

The study was conducted on the Psychiatric Service of the Veterans Administration Hospital, Durham, N.C., a 500-bed university affiliated general hospital. The Psychiatric Service is an 80-bed acute inpatient facility with no specifically designated program for alcoholics, but alcoholic patients are regularly treated on an individual basis intermixed with the general psychiatric population.

After admission, patients receive diagnostic evaluation by a team of staff and resident psychiatrists, social workers, psychologists, nursing personnel, and personnel from a variety of other disciplines, plus medical and social work students and psychology trainees. Social workers routinely collect information from family or other informant sources and incorporate this information into the work-up and treatment plan. Length of stay varies from one week to six months, averaging approximately six weeks.

Each patient admitted to the Psychiatric Service over a one-year period was interviewed by a psychiatric research technician one to seven days following admission. The interview consisted of a standardized introduction and a series of 16 questions of a benign and indifferent nature (education, marital status, etc.), with the CAGE questions included. The CAGE questions are: "Have you ever felt you should *cut* down on your drinking?" "Have people *annoyed* you by criticizing your drinking?" "Have you ever felt bad or *guilty* about your drinking?" "Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hang-over (*eye-opener*)?" Two

other alcohol-related questions, asked before the CAGE questions, were "Do you ever drink?" "Does your wife ever drink?"

Following discharge, each patient's name was placed on a list maintained by the social worker (G.M., P.H.) covering that patient's ward area. The social worker categorized the patient as alcoholic or nonalcoholic on the basis of diagnostic formulations by the multidisciplinary team from the information collected from the patient and from informant sources. The alcoholic/nonalcoholic categorization was subsequently correlated with the CAGE responses. The alcoholism designation was correlated with the response to each of the CAGE questions and with the questionnaire as a one-, two-, three-, or four-item instrument. The statistical analysis was accomplished by using the phi coefficient of correlation for correlation of a true dichotomy with a dichotomized variable (4).

RESULTS

Three hundred sixty-six patients were evaluated over the one-year period. The patients were predominantly male (99 percent), white (77 percent), and middle-aged (63 percent between 35 and 55 years), ranging in age from 19 to 75 years. Lower socioeconomic classes (5) were overrepresented (classes IV and V = 73 percent), and upper classes were underrepresented (classes I and II = 7 percent). Sixty percent of the subjects were married, 16 percent separated or divorced, 2 percent widowed, and 22 percent single.

Of the 366 patients, 79 percent were alcohol users and 21 percent were abstainers, while 33 percent of their spouses were alcohol users and 67 percent were abstainers. Of the 366, 39 percent were categorized as alcoholics and 61 percent as nonalcoholics. Table 1 shows the responses of the alcoholic and nonalcoholic patients to the CAGE questionnaire. Used as a complete four-item questionnaire, there were no false positives, but only 37 percent of the alcoholics were appropriately identified, and the correlation was rather unimpressive (r = .65). Using two or three positive responses as criteria, however, yielded a rather impressive correlation coefficient (r = .89).

The question "Have people annoyed you by criticizing your drinking?" had substantially lower power as a predictive criterion than did the other three CAGE questions, primarily because 50 percent of the alcoholics failed to answer it affirmatively.

We carefully examined the individual records of those patients who were most clearly misidentified by their CAGE responses (alcoholics who scored zero or one positive response and nonalcoholics who scored three positive responses). Fourteen patients categorized as alcoholics gave negative responses to all CAGE questions. Of these, seven were psychotic—four with functional psychoses (three schizophrenic and one manic patient) and three with organic brain syndromes. The other seven patients manifested competent mental status.

TABLE 1
Comparison of Positive CAGE Responses of Alcoholic and Nonalcoholic Patients, in Percents

CAGE Questions	Alcoholic Patients (N = 142)	Nonalcoholic Patients (N = 224)	Correlation Coefficient
Number of positive responses			
4	37	0	.65
3	30 (67*)	2	.59
2	14 (81*)	9 (11*)	.59
1	9 (90*)	10 (21*)	.55
0	10	79	.55
Type of question			
Cut down	87	16	.88
Annoy	50	8	.60
Guilty	76	7	.59
Eye-opener	63	4	.83

*Cumulative percent.

Twelve patients categorized as alcoholics gave positive responses to only one CAGE question. Of these, six were psychotic (two schizophrenic and two manic patients and two patients with organic brain syndromes). The mental status of the remaining six alcoholics was not remarkable.

Five nonalcoholic patients gave positive responses to three CAGE questions. All of these patients were clearly incompetent to give valid responses because of psychotic states at the time of the interview. (Four were schizophrenic, and one had an organic brain syndrome.)

COMMENT

The CAGE questionnaire is brief and easy to administer, comparing quite favorably in this regard with the Michigan Alcoholism Screening Test (MAST) (1) and a shortened version of the MAST (2). The CAGE questionnaire also appears to be less intimidating to the respondent than either version of the MAST.

Our work indicates that the CAGE questionnaire is not a sensitive detector of alcoholism if a complete, four-item response is the sole criterion. Its sensitivity is impressive, however, if a two- or three-item positive response is accepted as the criterion. Elimination of those subjects who are clearly incompetent to give a valid response (schizophrenic and manic patients and those with organic brain syndromes) would further strengthen the sensitivity and validity of the CAGE as an indicator of alcoholism. Development of a weighted scoring system would probably also improve the validity of the questionnaire.

Our delineation of alcoholism, based on a comprehensive multidisciplinary evaluation of the patient over a period of time and using available informant sources, seems to be a practical criterion against which to validate an instrument for the detection of alcoholism or problem drinking. The questionnaire needs further evaluation in different populations and different settings, but it appears to be a very promising technique for rapid screening of alcoholism in large groups.

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To Drink or Not To Drink: An Experimental Analysis of Group Drinking Decisions by Low Alcoholics

BY MARK S. GOLDMAN, PH.D.

The author reports on a study that examined the effects of social influence on the initiation, maintenance, and termination of drinking in a group of four men who were chronic alcoholics. These men made decisions in a group setting about alcohol and cigarette use for which they earned reinforcement points redeemable for drinks and cigarettes during the study or for money at its end. Decision making delayed the initiation of drinking and tended to lower overall alcohol consumption during a period of prolonged drinking but was not effective in inducing early termination of the drinking episode. Mood disturbances, physical symptoms, and psychopathology became more pronounced when the amount of alcohol consumed increased. The extent of decision making changed during times of high motivation for drinks or cigarettes; these decisions were mainly a function of individual leadership. In the light of these findings, the author discusses the possible utility of the group decision-making model as a tool for the treatment of alcoholism.

THE PRESUMPTION that social factors play a major role in the etiology and maintenance of chronic alcoholism is now well supported in the literature on alcoholism (1–3). However, most efforts to clarify the role of social factors in alcohol addiction to date have depended on field observations in natural settings. Recently, two groups of laboratory-based experimenters have begun to examine the effects of social behavior on consequent drinking behavior, but these have not yet focused on the reciprocal relationships between drinking and interpersonal behavior (4–7).

The extensive experimental literature on small-group dynamics offers methods by which reciprocal relationships between drinking behavior and small-group phenomena can be explored. In particular, the work of Wallach and associates (8, 9), which explored the effects of group decision making on willingness to come to risky decisions, and that of Schachter (10), which studied communication patterns between regular and deviant group members during decision making, seem to offer appropriate models for this purpose.

The study on which this paper reports examined the behavior of four male chronic alcoholics who lived together in a laboratory environment for 25 days. At certain times during the study, their drinking and smoking behavior depended on group decisions, drinking and smoking at other times were a function of individual decisions. The study was undertaken to explore reciprocal relationships between group behavior and the social, affective, and drinking behavior of the four chronic alcoholics who participated in the study. Because group decisions to initiate, maintain, and terminate a period of prolonged drinking were required, group behavior during an entire episode of drinking could be subjected to detailed examination.

Based in part on a dissertation submitted to Rutgers University, New Brunswick, N.J., in partial fulfillment of the requirements for the Ph.D degree in psychology.

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This study was supported by research grant MH-18850 from the National Institute on Alcohol Abuse and Alcoholism to Peter E. Nathan, Ph.D.

The International Journal of the Addictions, 16(2), 283-303, 1981

Acute and Chronic Drug Abuse Emergencies in Metropolitan Toronto*

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Abstract

From 3,548 drug overdose or abuse cases presenting at 21 Metropolitan Toronto hospitals' Emergency departments, data con-

*Presented in part at the Canadian Royal College of Physicians meeting, Quebec City, January 1976; the Canadian Conference on Pharmaceutical Research, Saskatoon, May 1976; and the annual meeting of the National Council on Alcoholism, Washington, D.C., May 1976.

cerning demographic and medical characteristics, investigative and management procedures, drug analysis services, and disposition of patients were collected. Of the 3,548 cases, 2,723 (77%) were acute overdose and 816 (23%) were drug abuse. Drug overdose was more common than drug abuse for both sexes, but was more characteristic of females. The drugs most frequently alleged ingested were benzodiazepines (34%), ethanol (32%), salicylates (16%), and barbiturates (14%). The frequency with which particular classes of drugs are alleged in overdose corresponds closely to the frequency of prescribing these drugs in Ontario.

INTRODUCTION

Recent studies of the epidemiology and treatment of acute drug ingestion in adults have highlighted the social impact of acute drug overdose. This impact includes the incidence of such overdoses (Alderson, 1974; Jensen, 1977; Kessel, 1965; Smith, 1972), the nature of the drugs associated with morbidity and mortality in overdose (Ghodse, 1977; Lawson and Mitchell, 1972; Proudfoot and Park, 1978; Stewart et al., 1974), the cost to the health care system associated with drug overdose (Smith, 1972), and the possibility of identifying a "high-risk" population (Gethin Morgan et al., 1975; Hindmarch, 1972; Kennedy et al., 1974; Kessel, 1965).

There have been four previous studies of drug abuse emergencies in Canada, but because of different methodologies, the results are difficult to compare. Ruedy (1973), studying all overdose cases presenting at the Montreal General Hospital in 1972, found benzodiazepines and nonbarbiturate sedatives to be the most frequently ingested drug classes, with 43% of patients taking several drugs (other than ethanol, which was alleged in 23% of all cases). Rangno (1975), studying just the patients admitted to intensive care from the same population, found very similar results. In both of these studies, the drug classifications were a combination of patients' allegations and laboratory confirmations. In a study of all Hamilton, Ontario, hospitals for 15 months in 1966 and 1967, Sims et al. (1973) found sedatives (mostly barbiturates) more frequently implicated than minor tranquilizers, with fewer than 10% of the cases involving multiple drugs other than ethanol. This report did not state how the responsible drugs were identified. Sellers (1975) considered only the analyses of samples sent to a central Toronto laboratory in 1972 and 1973, when an adequate benzodiazepine assay was not available. Among the drugs de-

tectable, barbiturates were much more common than nonbarbiturate sedatives, and in 25% of the samples more than one drug was detected, not including ethanol. The four studies agree in finding much less involvement of "street drugs" (primarily amphetamines and hallucinogens) than of prescription drugs in leading to acute poisoning.

The present study was designed to determine the following: the patient characteristics and alleged choice of drugs associated with drug abuse emergencies; the accuracy of reporting by patients, their friends and relatives, and emergency care workers of what drugs have been ingested; the role of drug analysis in the clinical management of such patients; the nature of the morbidity, the extent of the mortality, and the estimated costs of caring for these patients; whether a high-risk overdose population can be identified; and patterns of follow-up of patients who have ingested overdoses of psychoactive drugs. Such information is needed for evaluating and modifying medical education, government regulation, and hospital administration.

This study includes 3,548 Emergency visits for acute adult drug ingestion or drug abuse during a 6-month period. All Metropolitan Toronto general hospitals with Emergency Departments are included; therefore, this is the first total urban catchment survey of this problem.

This first paper describes the methodology and general results of the study. Subsequent papers will focus on particular drugs, such as ethanol; on patient care strategies, such as the usefulness of serum analyses; on demographic determinants of drug abuse; and on the social policy and health care implications of our findings.

METHODS

The Patient Population

This study is based on the records of all patients with "acute drug overdose" or "drug abuse," presenting for Emergency services at 21 Metropolitan Toronto general hospitals between January 1 and June 30, 1975. While patients under the age of 14 were not specifically excluded from the study, most such patients in Metropolitan Toronto are referred to a special children's hospital which was not included in the study. There are eight "core" hospitals, seven of which are university-affiliated teaching hospitals. These teaching hospitals treated 99.4% of the core patients. The 13 "suburban" hospitals treated 34.2% of all patients, and included three teaching hospitals, which treated 44.6% of the suburban patients.

Problem Classification

Overdose, or acute drug ingestion, is defined as "A single ingestion of a quantity of drug greater than an individual's usual habit or greater than the maximum recommended prescribed dose." This category also includes the 2% of our sample who ingested substances other than drugs, topical agents, and household chemicals. *Drug abuse* is defined as "The chronic ineestion or injection of a quantity of psychoactive or analgesic drug which is greater than the recommended usual dose." This category includes most "street drugs" for which there is usually no medically recommended dose. From another viewpoint, the difference between overdose and abuse is usually that between intentionally self-destructive drug taking and the accidental consequences of experimental drug use. Patients whose sole recorded problem was excessive consumption of beverage alcohol ("has been drinking") were not included in the study.

Data Collection

Nurse monitors reviewed all Emergency presentations at the 21 hospitals to collect these data:

1. Demographic characteristics: sex, age, admission problem (overdose or drug abuse), persons or agencies accompanying the patient.
2. Alleged drugs ingested: name, quantity, route of administration, legal classification (prescription, o.t.c., or illicit), source of drug, and alleged concomitant ethanol ingestion.
3. Clinical characteristics, if recorded: level of consciousness (alert, drowsy, unconscious), psychic effects (confusion, delusion, hallucinations), blood pressure, pulse rate, respiratory rate, rectal temperature, reflexes.
4. Laboratory tests, if performed: blood gases, hemoglobin, hematocrit, electrolytes, BUN, creatinine, blood sugar, x-rays.
5. Treatment in emergency: medications given, other treatments (forced diuresis, forced emesis, intubation, lavage, etc.), duration of stay in Emergency, type of discharge (admitted to inpatient treatment, expired, psychiatric referral, community service agency, clinic, etc.)

Other than the hospital records, these data were available to us:

1. Serum and urine analyses were frequently performed to determine the correspondence between the drugs alleged and those that could be confirmed, as well as to determine the overall pattern of drugs confirmable

in laboratory tests. Analytical toxicological facilities vary widely among the hospitals in Metropolitan Toronto, but they all have access to a common laboratory at the Addiction Research Foundation Clinical Institute. All decisions concerning the selection and use of these analytical services rested with the attending physicians, and considerable care was taken during the study not to interfere with the usual patterns of patient management.

2. Coroner's office records of deaths from drug overdose during the time of this study were made available to us, so that we could compare the patterns of drug-related deaths outside of Emergency Departments to the drug use of our sample.

Statistical Processing of the Data

In addition to the usual computer software, general-purpose languages such as FORTRAN and application packages such as SPSS, we needed to develop two special programs for the data-collection phase of this project. The first of these programs uses a computer-driven plotter to draw complex coding forms on which the nurse monitors entered data at the hospitals and from which the data could be keypunched directly. The second program facilitates data validation by reporting improbable or illegal values in the various data fields, and subsequently combines the differing numbers of cards from each patient (depending on the types and numbers of tests performed) into master-file records of a common length and format. (More complete details of the data collection procedure, including the recording forms, drug codes, and analysis procedures, are available from the authors.)

The code numbers assigned to the drugs were chosen to facilitate subsequent analysis. Each drug is assigned a four-digit code, of which the first two digits signify a pharmacologic category and the last two digits specify a drug within that category. For example, the drugs chlordiazepoxide, diazepam, and flurazepam are all given numbers in the 1700 series, benzodiazepines. Some of the drug categories, such as benzodiazepines, barbiturates, salicylates, and bromides, are quite homogeneous. Unfortunately, it was also necessary to create some less homogeneous categories, such as "other analgesics" (not salicylates or opiates), "hallucinogens," and "household and industrial products" (which included mercury, waxes, shampoo, and lye). However, almost all of the drugs alleged were from the pharmacologically reasonable categories. All of the data reported in this paper concern these categories of drugs, not specific drugs within the categories.

In all of our drug category statistics, unless we specifically state otherwise, a patient may be classified into several overlapping categories, corresponding to the multiple drugs ingested, whether they are dispensed as components of a combination product or as separate products. For example, the class of patients who have taken both ethanol and salicylates does not exclude patients who have added barbiturates to that combination. For that reason, there are many tables in which patients appear more than once, and in which percentages total more than 100%.

RESULTS

During the 6 months of this study, 3,548 visits were recorded at the Emergency Departments of the 21 Metropolitan Toronto hospitals. Of these, 816 (23.1%) were drug abuse cases and 2,723 (76.9%) were drug overdose cases. Of the visits, 21.4% were by the 9.7% of the patients seen at least twice during the 6-month period, including some patients who were transferred between two hospitals' Emergency Departments for the same incident.

Metropolitan Toronto had, in 1975, a population estimated at 2,177,400.* Based on this statistic, we estimate the incidence of drug abuse and drug overdose Emergency visits to be 75 and 250 per 100,000 population per year. Of the 3,548 patients, only three died in Emergency as a result of the incident. They were a 21-year-old male who had taken cyanide, a 33-year-old female who had taken ethanol and an unspecified drug, and a 26-year-old female who had alleged "sleeping pills."

Patient Characteristics

Table 1 shows the total patient population, separated into drug abuse and overdose categories, tabulated against sex, age, and hospital location. Compared with the general population of Metropolitan Toronto in 1975, the patient population has a higher proportion of females and of persons under the age of 35. The relationship between sex and problem was statis-

*Except for Figs. 1 and 2, values for the total Metropolitan population and the core:suburban division are 1:4 interpolations between the 1971 and 1976 census data. The age and sex proportions are from the 1971 census, excluding persons under the age of 10 years. In Figs. 1 and 2, the age distribution graphed is based on the 1971 census distributions, with the population for each age cohort multiplied by 4 years' growth in the total Metropolitan Toronto population, the age bounds for each cohort advanced by 3 years, and the resulting distribution smoothed.

Table 1
Profile of Total Patient Population (N = *3,548*)[a]

Type of problem	Total patient population		Sex[b,c]				Age[b,c]								Hospital location[b,c]			
			Male		Female		< 21		21-35		36-50		< 50		Core		Suburban	
	N	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)
Drug overdose	2723	(76.9)	930	(34.2)	1791	(65.8)	606	(22.9)	1267	(47.8)	562	(21.2)	213	(8.0)	806	(29.6)	1917	(70.4)
Drug abuse	816	(23.1)	555	(68.0)	261	(32.0)	347	(44.2)	335	(42.7)	75	(9.6)	28	(3.6)	503	(61.6)	313	(38.4)
Total	3539	(100.0)	1485	(42.0)	2052	(58.0)	953	(27.8)	1602	(46.7)	637	(18.6)	241	(7.0)	1309	(37.0)	2230	(63.0)
Metropolitan Toronto				(48.8)		(51.2)		(22.5)		(29.4)		(22.7)		(25.5)		(30.7)		(69.3)

[a]Throughout the paper the percent values have been calculated after the removal of the cases with missing data (e.g., for the overdose-abuse division, there were nine cases whose problem was not recorded).
[b]The percentages in this subtable are row percents, summing to 100% across the demographic categories, not down the drug problem categories.
[c]Statistically significant difference ($p < .01$) in proportion of drug overdose between sexes, age groups and hospital location.

tically significant ($p < .01$); while females were only 57.9% of the overall patient sample, they were 65.8% of the overdose patients.

Table 2 summarizes the characteristics of the patient population in the core and suburban hospitals. The geographic distribution of patients (63.0% suburban, 37.0% core) is very similar to that of the general population (64.9% suburban, 35.1% core). While the patient population for each of the two groups of hospitals is not restricted to residents of the hospital area, patients and ambulances tend to use nearby hospitals rather than those more distant, especially in emergencies. Most drug abuse (61.6%) was treated at the core hospitals, while most overdose (70.4%) was treated in the suburbs ($p < .01$). The suburban patient population includes a significantly higher proportion of females (63.8%) than does the core patient p;pulation (48.0%) ($p < .01$) or the general Metropolitan Toronto population (51.2%). There is also a significant relationship between location and age ($p < .01$); the 21–35 group represents 50.9% of the core patients but only 44.2% of the suburban patients. In general, this age group is overrepresented in the patient population (46.6%) compared to its representation in Metropolitan Toronto (28.9%). Figure 1 displays, in greater detail, the age distribution of the Metropolitan Toronto population at the time of the survey, the patient population, and the portion of the patient population who alleged one of the commonly mentioned classes of drugs, barbiturates. This class is alleged frequently in both overdose and drug abuse presentations. Figure 2 shows the Metropolitan and patient distributions by age and sex, and shows all allegations of hallucinogens other than cannabis, a class seen almost exclusively in drug abuse presentations.

Use of Laboratory Services

Most Metropolitan Toronto hospitals can detect only three classes of drugs in serum: barbiturates, salicylates, and bromides. One core hospital can screen for a somewhat larger spectrum, but only the common laboratory can routinely detect alcohols, solvents, benzodiazepines, other nonbarbiturate depressants, neuroleptics, antidepressants, amphetamines, and hallucinogens, in serum or urine as appropriate. Table 3 shows the use of both local and common analysis facilitates by core and suburban physicians. Overall, drug abuse patients are tested less often than overdose patients, even though drug abuse is largely concentrated into the core area where the common laboratory could perform the necessary screen. Among overdose patients, those in suburban hospitals have a lower overall rate for screening than do core patients, even though the rate of screening in

Table 2

Characteristics of Patient Population of Core and Suburban Hospitals (N = *3,548*)

Hospital location	Total patient population		Sex[a,b]				Age[a]							
			Male		Female		< 21		21–35		36–50		> 50	
	N	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)
Core	1313	(37.0)	682	(52.0)	630	(48.0)	337	(27.2)	631	(50.9)	201	(16.2)	71	(5.7)
Suburban	2235	(63.0)	808	(36.2)	1426	(63.8)	619	(28.1)	973	(44.2)	408	(19.9)	171	(7.8)
Total	3548	(100.0)	1490	(42.0)	2056	(58.0)	956	(27.8)	1604	(46.6)	639	(18.6)	242	(7.0)

[a]The percentages in this subtable are row percents summing to 100% across the demographic categories, not down the location category.
[b]Statistically significant differences ($p < .01$) in proportion of core and suburban patients between sexes.

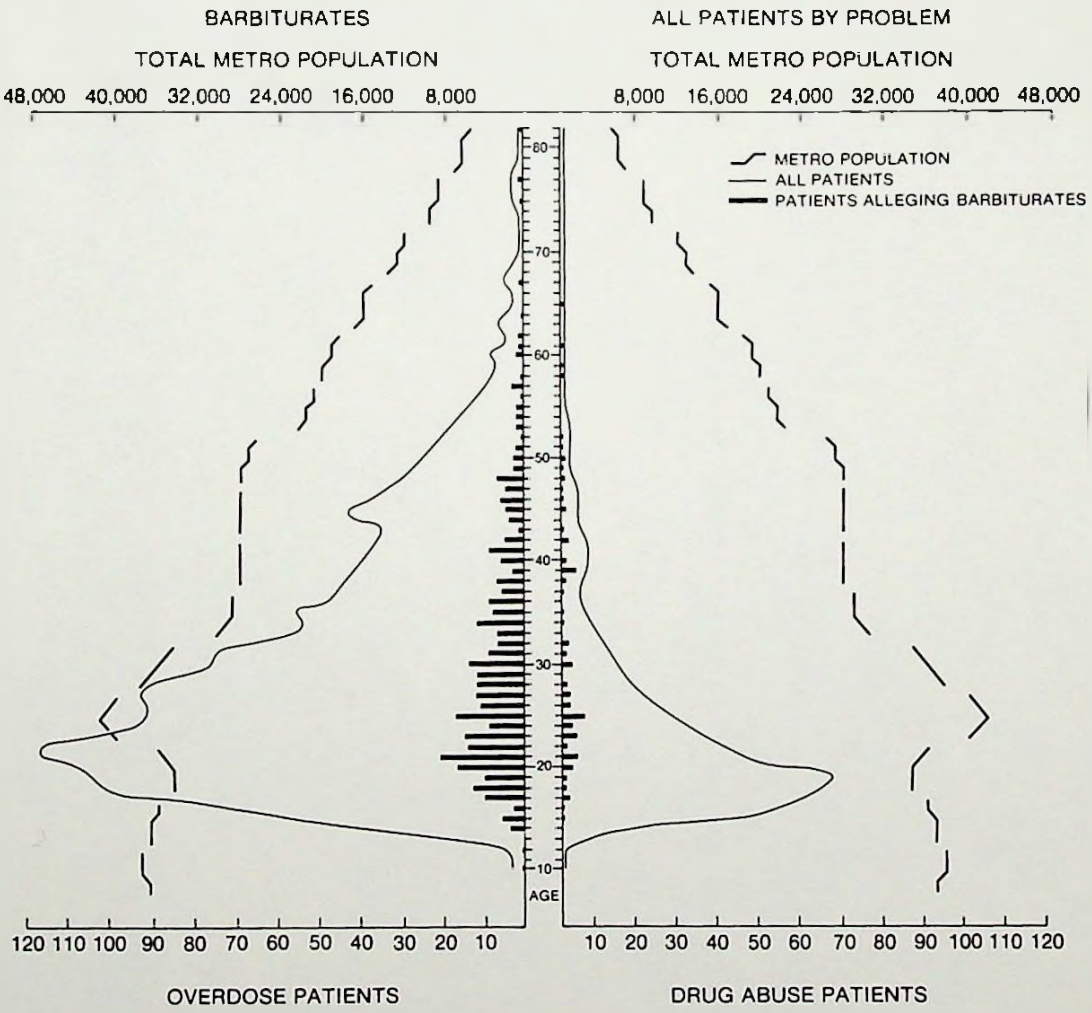

Fig. 1. Age distribution for Metropolitan Toronto in 1975 for patients requiring emergency treatment for acute overdose or drug abuse in the first half of 1975, and for the 504 patients (14%) alleging barbiturates among the drugs responsible for their admission.

the hospital of admission is twice as high. That is, almost all of the suburban patients' screens are done at the home hospital, with little use of the central, common laboratory. In the core area, twice as many screens are done at the common laboratory than at the hospital of admission.

Drug Classes Ingested

Table 4 summarizes the drug classes most frequently alleged by the total patient population. By "alleged," we mean whatever drugs were listed in the hospital Emergency record, whether the drugs were reported by the

Table 3

Use of Toxicological Laboratory Services as a Function of Hospital Location[a]

Type of problem	Hospital location	No use		Primary care laboratory		Common laboratory		Both laboratories		Any screen	
		N	(%)	*N*	(%)	*N*	(%)	*N*	(%)	*N*	(%)
Overdose	Core, $N = 806$	240	(29.8)	171	(21.2)	347	(43.1)	48	(6.0)	566	(70.2)
	Suburban, $N = 1{,}917$	852	(44.4)	827	(43.1)	176	(9.2)	62	(3.2)	1065	(55.6)
	Total, $N = 2{,}723$	1092	(40.1)	998	(36.7)	523	(19.2)	110	(4.0)	1631	(59.9)
Abuse	Core, $N = 503$	257	(51.1)	19	(3.8)	220	(43.7)	7	(1.4)	246	(48.9)
	Suburban, $N = 313$	221	(70.6)	58	(18.5)	29	(9.3)	5	(1.6)	92	(29.4)
	Total, $N = 816$	478	(58.6)	77	(9.4)	249	(30.5)	12	(1.5)	338	(41.4)

[a]In each row of this table, the first four percentages sum to 100%. The last column is the sum of the preceding three columns.

Table 4
Drugs Most Frequently Alleged Ingested

Drugs or drug group	Allegation of use in total patient population[a]	
	N	(%)
Benzodiazepines	1,201	33.9
Ethanol	1,125	31.7
Salicylates	559	15.8
Barbiturates	504	14.2
Nonbarbiturate hypnotics	388	10.9
Narcotic analgesics	295	8.3
Tricyclic antidepressants	247	7.0
Hallucinogens	219	6.2
Major tranquilizers	217	6.1
Other analgesics	214	6.0
Amphetamines	180	5.1
Cannabis	135	3.8
Solvents	45	1.3
Bromides	32	0.9
Cocaine	24	0.7

[a]When drugs of two or more groups were allegedly ingested by a given patient, the patient was included in each of the relevant groups.

patient or persons accompanying the patient, listed on bottle or pill labels, or suggested by physicians' clinical judgment. Benzodiazepines and ethanol were each alleged by approximately one-third of the total patients, followed by salicylates and their combinations, barbiturates, and other hypnotics. The "streeet drugs," hallucinogens and cannabis (9.3%) and amphetamines (5.1%) were alleged by only a small proportion of the patients, and by very few patients in the overdose category.

The users of each drug category are clasified by sex and problem in Table 5. The drugs in this table can be divided into two categories. "Street" drugs form one category, including cannabis, other hallucinogens, amphetamines, solvents, and cocaine. These drugs are characteristic of males and of drug abuse. The other category consists of drugs of legitimate medical uses, including analgesics, antidepressants, and depressants, along with ethanol, which is pharmacologically similar to the prescription depressants. These drugs are characteristic of overdose patients, and many of them are characteristic of female patients.

Multiple Drug Use

Approximately half (51.6%) of all patients had alleged more than one drug, including ethanol, prior to their presentation at Emergency. "Mul-

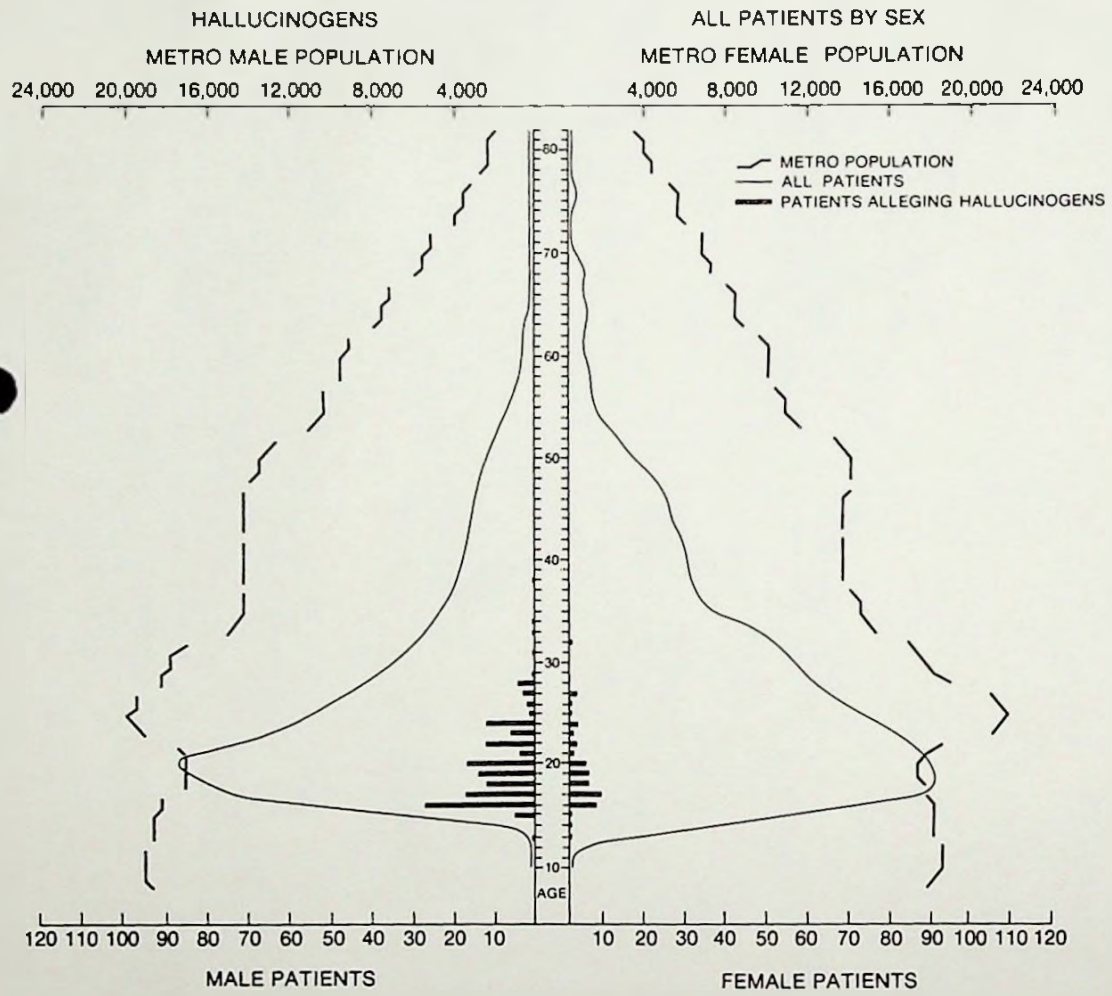

Fig. 2. Age distribution by sex for Metropolitan Toronto in 1975 for patients requiring emergency treatment for acute overdose for drug abuse in the first half of 1975, and for the 209 patients (6%) alleging hallucinogens other than cannabis among the drugs responsible for their admission.

tiple drugs" includes single formulations with several active ingredients of different pharmacological classes (such as over-the-counter combinations of ASA, caffeine, and codeine), but not combinations of separate products from the same category (such as amobarbital and secobarbital). Excluding ethanol, 33.5% of the patients alleged multiple drugs. The distribution of the number of drug categories alleged is shown in Table 6. No difference in multiple drug use is found between males and females, but the incidence of multiple use is slightly higher in overdose than in drug abuse (Fig. 3). The two categories most frequently encountered in multiple use are ethanol and benzodiazepines (17.4% of patients alleging multiple drugs, including ethanol). In 9.2% of the patients, both ethanol and ben-

Table 5
Characteristics of Alleged User Population for Specific Drug Groups

Drug or drug group	*N*	% of all patients	% of overdose patients	Overdose Abuse	Female Male
All patients	3,548	100.0	100.0	3.31[a]	1.38[c]
Benzodiazepines	1,201	33.8	39.3	8.38[a,b]	1.88[c,d]
Ethanol	1,125	31.7	31.1	3.06[a]	1.00[d]
Salicylates	559	15.8	19.0	12.56[a,b]	2.25[c,d]
Barbiturates	504	14.2	15.2	4.69[a,b]	1.19
Nonbarbiturate hypnotics	387	10.9	12.4	6.88[a,b]	1.25
Narcotic analgesics	295	8.3	8.2	3.13[a]	1.44[c]
Tricyclic antidepressants	247	7.0	8.8	39.81[a,b]	2.44[c,d]
Hallucinogens	219	6.2	0.7	0.06[a,b]	0.31[c,d]
Major tranquilizers	217	6.1	7.4	14.38[a,b]	1.75[c]
Other analgesics	214	6.0	7.3	12.38[a,b]	2.56[c,d]
Amphetamines	180	5.1	1.0	0.19[a,b]	0.56[c,d]
Cannabis	135	3.8	0.7	0.13[a,b]	0.44[c,d]
Solvents	45	1.3	0.5	0.38[a,b]	0.19[c,d]
Cocaine	24	0.7	0.1	0.06[a,b]	0.50[d]

[a]Significantly different from 1.00.
[b]Significantly different from the overdose:abuse ratio in patients not alleging this category.
[c]Significantly different from the female:male ratio among the Toronto population.
[d]Significantly different from the female:male ratio among patients not alleging this category.

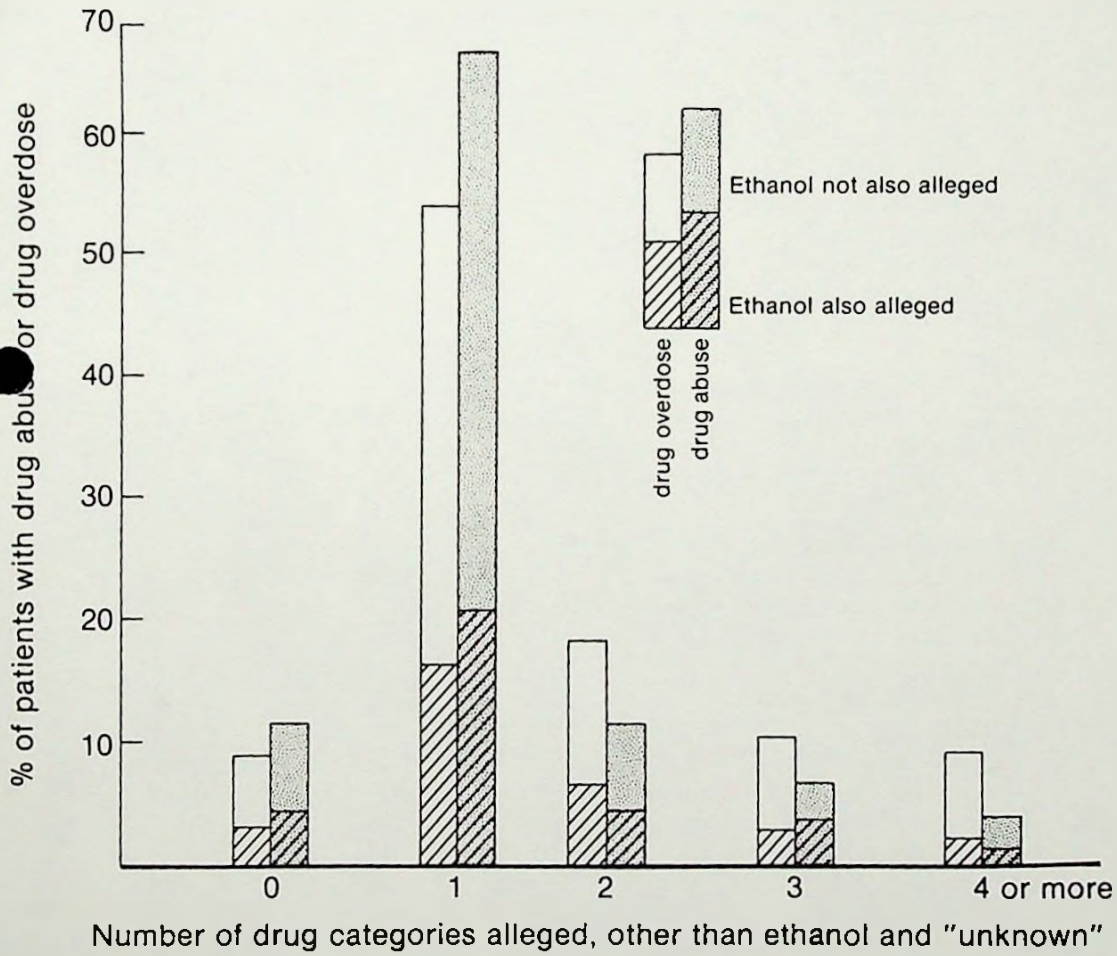

Fig. 3. Number of drug categories alleged ingested, other than ethanol and "unknown," for patients in the overdose and drug abuse categories (overdose $n = 2{,}723$, drug abuse $n = 816$).

zodiazepines were alleged. Table 6 shows the exact combinations of drug categories that were most frequently alleged. In this table, unlike Tables 4 and 5, no patient appears more than once.

Disposition of Patients

Overall, 1,263 (35.6%) of the patients presenting at Emergency were subsequently admitted for inpatient treatment. The overdose cases were admitted more frequently (41.1%) than the drug abuse cases (17.2%, $p < .01$). The suburban hospitals, which had a disproportionate share of the overdose cases, had an overall admission rate twice that of the core hospi-

Table 6
Frequencies of Most Commonly Alleged Multiple Drug Combinations (N = 3,548)

Drug combination	Frequency	
	N	*(%)*
Ethanol		
+ Benzodiazepines	255	(7.2)
+ Barbiturates	35	(1.0)
+ Opiates + salicylates + caffeine[b]	22	(0.6)
+ Nonbarbiturate sedatives	14	(0.4)
+ Other categories[a]	130	(5.1)
+ Barbiturates	98	(2.8)
+ Hallucinogens (including cannabis)	74	(2.1)
+ Nonbarbiturate sedatives	40	(1.1)
+ Antidepressants	35	(1.0)
+ Nonsalicylate analgesics	24	(0.7)
+ Amphetamines	20	(0.6)
+ Other single categories	74	(2.0)
+ Opiates + salicylates + caffeine[b]	22	(0.6)
+ Other categories[a] without benzodiazepines	315	(8.9)
Benzodiazepines		
+ Salicylates	40	(1.1)
+ Nonbarbiturate hypnotics	37	(1.0)
+ Barbiturates	33	(0.9)
+ Opiates + salicylates + caffeine[b]	26	(0.7)
+ Other categories[a] without ethanol	247	(7.0)
Opiates + salicylates + caffeine[b]	63	(1.8)
Other combinations[a]	595	(16.8)
Single drug categories	1349	(38.0)

[a]Including the category "unknown" when the patient was suspected of having taken one or more drugs in addition to any that could be identified.
[b]In Canada, this combination is a common analgesic product in various strengths, available without written prescription for the smaller quantities of codeine.

tals (44.0% compared to 21.3%, $p < .01$), and the higher suburban admission rate remains true for patients presenting with all levels of consciousness. The total of 1,263 inpatient admissions during this study represents only 0.6% of the total of 190,000 admissions to these hospitals for all causes in the first half of 1975. Of the patients not admitted, about half were referred to their own physicians or to medical or community agencies for further treatment.

DISCUSSION

The results of this study of Metropolitan Toronto indicate that acute and chronic drug abuse are common, resulting in 325 Emergency visits

and 116 inpatient admissions per 100,000 population per year. Many of the results of the study coincide well with general experience. For example, mortality is low, chronic drug abuse is more common in the male and younger age groups, frequently more than one drug is alleged, and many patients have repeated incidents. Such observations assure us that the study methodology was adequate.

The division of patients into (acute) overdose and (chronic) drug abuse groups is much less objective than the classification of patients by age or sex. It is sensible to speak of a patient's "sex role" or "age group identification," self-concepts that may well influence the way in which drugs are used. It is doubtful that many patients have well-defined "abuse roles," continuing identifications with the class of acute or chronic substance abusers. Therefore, we see our division of patients into these two categories as only a summary judgment of the circumstances of drug ingestion, which might correlate with or predict more objectively classifiable events, but cannot really be said to cause them. Only the self-perception of "attempted suicide," an extreme type of overdose which we could not differentiate from other overdose, might alter the patient's choice of substances, quantities, and circumstances in drug abuse.

Successful drug-induced suicide is not as rare as the three deaths in this study might imply. The Metropolitan Toronto Coroner (1975) reports 103 cases of drug suicide during 1975. While those data do not differentiate among deaths at home, in ambulances, in Emergency, or afterwards, clinical experience suggests that few overdose patients pass through Emergency only to die soon afterwards as inpatients. As death rates are generally low, we must look for differences among lesser consequences of these drugs' ingestion, such as admission rates and duration of hospital stay.

The greater suburban admission rate cannot be explained by the fact that more patients do present in critical condition: the greater admission rate holds for all levels of consciousness. The reasons more patients are admitted to suburban hospitals may relate to different emergency staff levels, availability of hospital beds, or lack of alternate community facilities. From the point of view of controllling health care costs, the doubled suburban hospitals' admission rate for drug overdose patients deserves careful scrutiny.

The most striking pattern of results concerns which drugs are chosen by patients taking an intentional overdose (Table 5). Among prescription drugs, the ranking is benzodiazepines (39.3%), barbiturates (15.2%), nonbarbiturate sedatives (12.4%), tricyclic antidepressants (8.8%), and neuroleptics (7.4%). The same rank ordering, with almost the same proportions of use (39.6, 16.4, 11.1, 10.9, and 6.2%), is found for these five classes of

drugs in the 1973 Parcost survey of Ontario prescription patterns (Cooperstock, 1976a). The figure of 16.4% for barbiturates excludes phenobarbital, which is not usually prescribed to the general population for the same sedative uses as other barbiturates, but is usually restricted to the treatment of epilepsy. Drugs easily obtainable without a prescription, ethanol, salicylates, and (in Canada, in small doses per tablet) codeine, are also high on the list of agents chosen for overdose. These data are consistent with the hypothesis that the patient who wishes to take an overdose will simply ingest whatever psychoactive or analgesic drugs are available. The sex ratio in overdose, roughly 1.9:1 female, supports this availability hypothesis, as Cooperstock (1976b) found that about 1.7 times as many females as males receive prescriptions for psychoactive drugs. Our data imply that females are only slightly more liable to drug overdose (in the sense of mental predisposition) than are males; twice as many females simply have the opportunity to act upon the impulse to take an overdose.

It is interesting that no drug classes' users are strongly overrepresented among the overdose population. That is, patients with antidepressants available for overdose, patients who were presumably "depressed" before drug therapy, do not develop the predisposition to overdose any more often than patients who were in pain or in need of tranquility. This is consistent with a very successful titration of therapy to the needs of the patient population. While patients taking antidepressants may have entered therapy with more "depression" (a state that might predispose a patient to self-destructive behavior), the patients actually taking such drugs show no more tendency to engage in such behavior than do the patients taking other classes of drugs. This could be viewed as evidence that the physicians who dispense psychoactive drugs are having remarkable success in reducing or raising all patients to the same psychologic state.

Another hypothesis can also account for the close correspondence between drugs available to the population and drugs used in overdose, despite the available drugs' being directed at quite different mental states. According to this alternate hypothesis, the conditions that lead a physician to prescribe a particular psychoactive drug are simply unrelated to the conditions that lead to overdose, and the various drugs have no differential effect on that predisposition. In other words, if different psychoactive drugs that had equal effects (or no effect) on self-destructive predispositions were to be dispensed at random to patients with psychological problems, including self-destructive tendencies, we would see exactly the pattern of drug overdose as is seen in this study. Such drugs may be affecting other components of psychological adjustment, but there may be no differences in their rates of eliciting or preventing self-destructive drug-taking behavior.

One of the remarkable results of this study is that it is rare for a drug overdose case that reaches hospital to result in death in Emergency. This may simply mean that if a drug is going to do serious damage, then it is going to do such damage immediately, rather than slowly. There is not much that we can say about the relative safety of different drugs, based on the results of this study, as we have neither accurate control nor measurement of the amount of drug consumed by the patients.

While it is clear that drug-related parasuicide is a major problem, it is not clear what steps the medical community should be taking to reduce the problem. The close correspondence between drug availability and parasuicidal drug use suggests that these acts are largely impulsive, making use of whatever substances are at hand when the impulse strikes. However, this study provides no data from which we can determine how many patients use other alternatives when no drugs are available. In other words, if we could reduce the availability of drugs to these patients, we might simply be converting parasuicide into successful suicide, as patients opt for other available and potentially more dangerous (para)-suicidal behavior. It is overly simplistic to call drug-related parasuicide a "medical" problem, just because physicians prescribe the drugs that are used. If the drugs being prescribed were effective agents of suicide, then perhaps we could blame physicians for releasing them to the public. However, this study suggests that if we construct a "social interaction ratio" for forms of attempted suicide, the probability that the act will gain needed attention compared to the probability that it will kill the actor, drugs must rate as safe, compared to guns, carbon monoxide, or lye.

We must also consider another view of these emergencies. Perhaps they are not impulsive acts, unrelated to drug therapy, which simply use drugs as the handiest alternative form of self-damage. Instead, they might be logical extensions of the same drug-taking behavior that the physicians prescribe. A patient consumes drugs for the course of several weeks or months, obtaining reinforcement for every pill-taking act or, more dangerously, for almost every pill-taking act. This latter situation is known as partial reinforcement, and generates very stable and persistent behavior patterns. If, at some point, the situation for which the drugs are consumed becomes worse, or tolerance sets in and the drugs become less effective, the act of taking a pill fails to produce its expected effect. By the usual laws of operant conditioning, this situation should lead to an immediate increase in the rate of taking the pills. Only after a long history of no observable effect from taking the drug will the response extinguish, and by that time the patient has already consumed an overdose. If this is what happens in overdose, in some if not all cases, then we can no longer hold the physicians as blameless as if the parasuicides were impulses unrelated

to therapy. Instead, we would need to hold the physicians primarily to blame, as the agents who first taught the patients, individually or as a society, to use drugs as partially and temporarily effective solutions to their emotional problems. Clearly the data from this study cannot be used to evaluate such a detailed behavioral hypothesis, and clearly the act of taking an overdose is more complex than a simple increase in the rate of emitting an operant behavior at the beginning of extinction. However, such a possibility must be considered to counteract the view that physicians are blameless in the incidence of parasuicide, as stated above.

An epidemiological study such as this one can measure the scope and costs of some publicly-observable form of behavior, such as taking an overdose of drugs. As such, it can tell us whether the problem is of major or minor significance in the population. It cannot tell us much about the individual psychology of overdose. Without such information, we cannot tell whether to encourage physicians to prescribe drugs, because of their high "social interaction ratio," or discourage them, because of the danger of conditioning the patients to consume drugs. In making the usual call for further research, we would strongly suggest that it be directed toward intensive study of individuals rather than a survey of large groups. Educational programs that develop out of such studies should realistically combine social policy considerations with training in the identification and management of certain high-risk patients, those who are likely to resort to dangerous attention-getting behaviors.

ACKNOWLEDGMENTS

We are indebted to the Medical Directors, physicians, Medical Records staffs, Emergency Department Head Nurses, and nursing staff of each hospital that participated in this study. Without their cooperation, advice, and considerable work, the study would have been impossible. This study was supported in part by the Non-Medical Use of Drug Directorate, Health Protection Branch, National Department of Health and Welfare, RODA Grant No. 1212-5-144.

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Alcoholic Liver Disease: Information in Search of Knowledge?

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Anyone surveying the literature on alcoholic liver disease is struck by the dissociation between the vast information that has accumulated and the relative lack of its integration into coherent knowledge of the pathogenesis of alcoholic liver disease. It is our impression that we lack the framework to allow us to differentiate between what is central to alcoholic liver disease and what are epiphenomena. The reader in this field cannot escape the feeling that we may have engaged too actively in pursuit of peripheral areas which are concomitants or consequences of liver dysfunction but not necessarily the causes of alcoholic liver disease in man. In general, research by clinical and basic investigators has been slanted toward explaining mechanisms of obvious morphological abnormalities that often accompany liver disease. This has occurred despite the fact that some morphological abnormalities are not present in all patients with clinical manifestations of alcoholic liver disease, while other morphological abnormalities that follow chronic alcohol consumption occur without clinical liver disease.

Furthermore, the morphological approach has resulted in categorization of stages, such as fatty liver, alcoholic hepatitis, and cirrhosis, that not so long ago were believed to correspond to a sequence of events in this disease (1). A more fertile approach may be to address the problem from the extreme end of the pyramid, represented by death from alcoholic liver disease. The two main reasons leading to death in alcoholic liver disease are hepatocellular dysfunction and hemodynamic alterations resulting from portal hypertension.

ALCOHOLIC LIVER DISEASE: A CONSTANT CONCOMITANT OF HEAVY ALCOHOL CONSUMPTION?

It is only in the last decade that this problem has been subjected to critical epidemiological analysis. Mortality due to cirrhosis in different countries closely correlates with the amount of alcohol consumed and is not related to the type of beverage ingested (2-4). Although alcohol is the major etiological factor, it is not evident whether some individuals are protected against the development of cirrhosis. At a given time, in a population of alcoholics, only 8 to 15% have cirrhosis on liver biopsy (5). This has been taken as evidence that only such a percentage of the population is at risk (6). Lelbach (2) analyzed this aspect with regard to levels of alcohol consumption and the time required to develop alcoholic cirrhosis. His studies indicate that there is a combined, cumulative risk factor for development of cirrhosis given by the average daily consumption of alcohol multiplied by the time of consumption at that level. These studies have shown that, given a constant daily amount of alcohol consumption equivalent to the maximum rate of alcohol metabolism, the *probability* of developing cirrhosis (confirmed by biopsy) increases linearly with time.

According to Lelbach's studies (2), the probability of developing cirrhosis, for an individual consuming about 210 gm of ethanol daily for 22 years, is 50% and increases to 80% after 33 years of similar daily consumption. These data suggest that virtually every alcoholic can develop cirrhosis and that what primarily differentiates individuals is the time of heavy consumption required for cirrhosis to develop. Several studies suggest that women develop cirrhosis following shorter periods of heavy drinking (7-9).

From a biological point of view, it is difficult to understand a single process that takes 10 or more years to develop despite constant and maximal self-titrating levels of the injurious agent. The half-life of total liver proteins in man is approximately 10 days (10). Thus, unless the biological process which is altered by ethanol has a half-life markedly longer than that of total proteins, at constant levels of ethanol ingestion, a new steady state should occur in a short time. To reconcile these facts, at least two alternatives are conceivable (11): (a) alcoholic liver damage involves alteration of several processes in series, each with a long half-life, or (b) liver damage is produced by simultaneous combination of a primary effect produced by ethanol in a short time and a precipitating factor which occurs at random, independent of alcohol consumption. A simile of the second model is

[redacted] was supported by a grant from the Provincial Health Research Programme of the Ministry of Health for Ontario.

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[redacted] individual who continuously bets (drinks continuously and produces a primary effect) the same number at a roulette table. The probability of his having his number called (precipitating factor) increases linearly with time.

From the above, it is unlikely that the universal changes occurring after alcohol consumption for a very short time, such as fatty liver (12, 13) or mitochondrial enlargement (12, 13), can be the *single* cause leading to cirrhosis.

Due to the morphological criteria that dominate most studies, researchers have used cirrhosis as the final landmark of alcoholic liver injury. Since we feel that the final landmark of alcoholic liver disease should be death, due to complications resulting from portal hypertension or liver failure, we will emphasize these latter aspects. The expression of functional abnormalities in alcoholic liver disease has a notoriously bad correlation with the classical morphological categorization of alcoholic liver disease (14, 15). While correlation analyses in populations find severe alcoholic liver disease in groups with cirrhosis (14), it is well known that cirrhosis can be subclinical, the individual may be asymptomatic (8, 14, 16-20), and death and serious liver disease can occur in individuals with fatty liver or alcoholic hepatitis (21-23) without cirrhosis.

Full-blown histological cirrhosis is found in liver biopsies of 10 to 20% of alcoholics without clinical or laboratory manifestations of liver dysfunction (14, 16). (If we consider that there are several million alcoholics in the world, this means that a sizable number have asymptomatic cirrhosis.) The consequence of this outlook is that cirrhosis is not necessarily accompanied by hepatic dysfunction and that other factors, which frequently occur in cirrhotics, account for the clinical abnormalities leading to death. One concludes that there are fundamental and important unknown factors, other than cirrhosis, which must determine the degree of hepatic dysfunction in alcoholic liver disease.

The striking observation that mortality attributed to cirrhosis was reduced in France by 60% in a single year following wine rationing (24), is in agreement with a dissociation between function and classical morphology. It is unlikely that this decline in a short time could have resulted from reduction in the prevalence of established cirrhosis.

SPECIFIC MORPHOLOGICAL AND FUNCTIONAL ALTERATIONS FOLLOWING CHRONIC ALCOHOL CONSUMPTION: THEIR POSSIBLE RELEVANCE IN ALCOHOLIC LIVER DISEASE

Many excellent recent reviews have dealt with mechanisms that appear to be responsible for histological alterations in the liver of patients with alcoholic liver disease, and in the liver of rats fed alcohol for different periods (25-27). This review will focus on factors which, on the basis of present knowledge, may lead to liver dysfunction and production of portal hypertension. Due to lack of appropriate markers of liver cellular damage, it is difficult to define the alterations which are ultimately responsible for the clinical manifestation of liver failure. It is necessary to *postulate* that cell necrosis is the end stage of a continuum that starts with mild hepatocellular damage. We will assume that alterations that lead to cell death also lead to intermediate stages of cell damage, which can be better characterized biochemically rather than morphologically. Thus, necrosis of a small percentage of the hepatocyte population would not be a necessary condition for severe liver dysfunction. Necrosis could be the tip of the iceberg of underlying generalized cell dysfunction.

MITOCHONDRIAL ALTERATIONS

Abnormal mitochondrial forms are frequently seen in alcoholic liver disease. In general mitochondria are enlarged, crystae appear deformed and contain crystalline or paracrystalline inclusions (28–32). Enlarged mitochondria with bizarre forms occur in rats (33–35) and primates (36) fed alcohol chronically for short periods.

The functional significance of these changes, however, is not clear. Studies have shown either no changes in the activity of isolated mitochondria (37) or an impaired mitochondrial function (38–41); however, most studies suggest abnormal mitochondrial function as measured *in vitro*. The deleterious effects of ethanol on mitochondrial function depend on the composition of the diet that the animals receive concurrently with chronic administration of ethanol. Chronic ethanol administration with a high-protein-low-fat diet does not alter the rate of mitochondrial respiration *in vitro*, whereas, under identical experimental conditions, chronic ethanol administration with a high-fat-adequate-protein diet decreases state-3 and state-4 respiration (42). Are these mitochondrial alterations manifested *in vivo* (43)? Mitochondria in the intact cell normally operate at respiratory rates well below the maximal ones elicited *in vitro* in the presence of excess adenosine diphosphate (ADP). Evidence suggesting that the abnormalities observed in isolated mitochondria from chronically alcohol-fed animals are not functionally important comes from numerous studies showing that tissue slices (44), perfused livers (11, 45), and isolated hepatocytes (46, 47) of these animals do not respire at rates below controls. In fact, some studies show increases in tissue respirations following chronic ethanol treatment (11, 44, 45). Furthermore, in isolated hepatocytes from these animals, which showed no differences in respiration when compared to controls, oxygen consumption was markedly increased by a mitochondrial uncoupler (47).

Jenkins and Peters (48) presented data indicating that liver mitochondria of alcoholics without cirrhosis or hepatitis have normal or increased activity of a number of mitochondrial enzymes including succinate, glutamate and malate dehydrogenases, and cytochrome oxidase. These authors propose that the increases in enzymatic activities represent an adaptive response to ethanol exposure. Similar views were presented by Kiessling (49) for animals fed alcohol chronically. Thompson and Reitz (50) reported increased rates of choline oxidation in mitochondria of alcohol-fed rats. Mitochondrial shuttle systems for the transfer of reducing equivalents have also been reported to be normal following ethanol consumption (51). Similarly, ADP/O ratios are unaltered by chronic ethanol administration (39, 40, 43).

Most of these studies have been performed *in vitro* in

the absence of ethanol or acetaldehyde. Mitochondria prepared from ethanol-fed rats appear to be more sensitive to the inhibitory effects of ethanol and acetaldehyde *in vitro* (52). These compounds inhibit the incorporation of ^{14}C-leucine into rat liver mitochondria *in vitro* (53-55).

Another aspect to be considered is the specificity of the mitochondrial abnormalities observed in histological studies and its relationship to ethanol and acetaldehyde. Mitochondrial enlargement, disrupted crystae and crystalline, and paracrystalline inclusions occur in many pathological states in which ethanol or acetaldehyde involvement can be discounted (56). These include non-alcoholic hepatitis (57), halothane hepatotoxicity (58), Wilson's disease (59), systemic sclerosis (60), cells in the vicinity of hepatocellular carcinomas (61), old age (62), Gilbert's disease (63), hyperlipoproteinemia (64), chronic passive congestion of the liver (65), and hyperthyroidism (66). In the latter condition, mitochondrial respiration is increased rather than decreased, which suggests that it is difficult to draw conclusions as to whether these changes in mitochondria are pathogenic or represent compensatory changes.

Mallory's Hyalin and the Role of Immunology

Excellent reviews on the structure, nature and pathogenesis of Mallory's hyalin have appeared in the first issue of *Hepatology* (67, 68) and thus will not be analyzed here. To our knowledge, the importance claimed for Mallory bodies in the production of alcoholic liver disease relates mainly to their possible role in the immunological abnormalities which occur in this condition. There is abundant evidence of immunological hyperactivity of T- and B-lymphocytes in alcoholic liver disease (69-72), which may be the mechanism through which some patients, despite complete (self-reported) abstinence, continue to show hepatocellular necrosis and progression to cirrhosis (73-75).

The following findings support involvement of Mallory's hyalin in the immunological response seen in alcoholic liver disease: (a) Mallory bodies appear to be leukotactic since cells that contain this material are often surrounded by polymorphonuclear leukocytes (76). (b) The lymphocytic infiltrates in the liver (77) [sometimes in close apposition to abnormal hepatocytes (78)] and the number of T-lymphocytes (79-81) within the inflammatory infiltrate in alcoholic hepatitis [and the corresponding depression in peripheral blood T-cells (81)] correlate with the presence of Mallory's hyalin in the liver (82). (c) Mallory bodies *in vitro* stimulate transformation of lymphocytes (83) from patients with alcoholic hepatitis, and also stimulate production of migration-inhibitory factor (84), fibrogenic factor (85), and transfer factor (86). (d) There is a correlation between cytotoxicity to isolated autologous or heterologous liver cells of lymphocytes derived from patients with alcoholic hepatitis and the presence of Mallory's hyalin in the lymphocyte donor (87-89). (e) The cytotoxicity of T-lymphocytes preincubated with isolated Mallory bodies or acetaldehyde increases significantly (87, 90). (f) Mallory's hyalin antigen has been detected in the serum of patients at the onset of alcoholic hepatitis and evokes an antibody response. Circulating antibodies persist for some time after Mallory bodies disappear from the liver (91-93). (g) Mallory's hyalin binds IgA. (h) Liver and kidney from patients with advanced alcoholic hepatitis or active alcoholic cirrhosis have immune complexes which contain anti-Mallory IgG and IgA immunoglobulins (91), although others have not been able to identify the exact nature of the antigen (94, 95).

The hypothesis of an immunological response playing a role in the production or autoperpetuation of alcoholic liver disease rests on the concept that lymphocytes are sensitized to antigenic components in the liver. Mallory's hyalin may be one of several neoantigens which activate a humoral and cellular immune reaction (96, 97).

Nevertheless, the role of Mallory's hyalin and, for that matter, of immunology, as the initial cause of liver damage is not conclusive. Separation of cause and effect has not been attained. Thus, the immunological findings in alcoholic liver disease might represent an epiphenomenon resulting from cellular abnormalities induced by ethanol and may have no significance in the production of damage. Immunologic mechanisms may be limited to perpetuation of liver damage in a group of alcoholics with liver damage and abnormal immunologic responses (72).

Other factors make the role of Mallory's hyalin uncertain. Among these is the problem of ascertaining the purity and specificity of the Mallory material with which antibodies have been prepared (88). It should also be noted that the presence of Mallory's hyaline not only is not specific for alcoholic liver disease (98) but, as observed in some studies, it does not appear to be related to the severity or to the prognosis of the disease (15, 99-101). Baboons treated with alcohol-containing diets do not manifest Mallory's hyalin (102) but develop lymphocytic cytotoxicity to autologous liver cells (103).

Acetaldehyde may play a direct role in immunological reactivity in alcoholic liver disease (104, 105). Acetaldehyde (and to a lesser extent ethanol) stimulates lymphocyte transformation (90) and production of lymphokines (87, 104, 105). The significance of these findings is controversial. Because of its small molecular size, acetaldehyde is unlikely to be an antigen. Activation of lymphocytes by acetaldehyde seems to be nonspecific as it also occurs in patients with chronic active hepatitis of non-alcoholic origin (90). Nevertheless, acetaldehyde is a highly reactive molecule which reacts with thiol groups of many enzymes and proteins and can covalently bind to normal or abnormal liver proteins (106). Acetaldehyde affects the plasma membrane by depolymerizing proteins and producing altered surface antigens (88). Antibodies against liver cell membrane antigens occur in patients with alcoholic liver disease (107), and a "liver specific lipoprotein (LSP)" has been described as a purified plasma membrane macrolipoprotein (108, 109). LSP has been used as a tool to determine specific lymphocytic cytotoxicity against liver cell membranes in patients with alcoholic hepatitis. Patients with alcoholic hepatitis frequently have circulating lymphocytes that are cytotoxic against autologous liver cells or isolated rabbit hepatocytes *in vitro*. This cytotoxicity is antibody-mediated and cell-dependent and is blocked by addition of LSP (74, 88, 110). This suggests that the hepatocyte damage may be caused by an immunological reaction directed specifically

at this antigen. Lymphocyte-mediated, LSP-inhibited cytotoxicity *in vitro* appears to correlate with necrosis in alcoholic hepatitis *in vivo*, and disappears following clinical and biochemical recovery (73). As proposed for Mallory bodies, it is also postulated that inability to suppress an autoimmune response to LSP after withdrawal of alcohol in predisposed individuals may explain the occasional progression to cirrhosis despite abstinence (73). The finding of an increased prevalence of HLA-B8 in patients with alcoholic hepatitis (72) suggests that, in [illegible] patients, there may be an abnormality in the control of immune responses. Interpretation of these data is hampered by controversy surrounding the purity and specificity of LSP. LSP is a complex system of antigens and may not be organ-specific, although it may contain very labile liver-specific components (111-116). Antisera to purified LSP have reactivity against antigens in human serum, kidney, and liver (111-113). The importance of LSP in immune reactions in alcoholic hepatitis will not be clarified until the purity and specificity of the antigen are established (114).

Contrary to what is observed in chronic active hepatitis, in alcoholic liver disease it is uncommon to find non-organ-specific autoantibodies against nuclei, smooth muscle, or mitochondria (117). Hyperglobulinemia is common and may involve an increase in all Ig classes, but it is particularly marked for IgA (118-120) and is attributed to decreased extraction of antigens which are normally phagocytized by the Kupffer cells due to intra- and extra-hepatic vascular shunts (118, 121). This abnormality also occurs in alcoholics without liver disease (119).

Alcoholic Metabolism and Metabolic Tolerance

Ethanol is metabolized in the liver primarily by the action of alcohol dehydrogenase (ADH), an enzyme present in the cell cytosol, and ethanol is converted to acetaldehyde, and NAD^+ is converted to NADH (122). The rate-limiting factor under normal conditions appears to be the availability of NAD^+ to the enzyme and thus the rate of NADH reoxidation which is a mitochondrial activity (45, 123-127). ADH may become rate-limiting in protein starvation when ADH levels are markedly depressed (128). In some rat strains, ADH levels are depressed by testosterone and explain the greater rate of metabolism in female than in male rats (129, 130). Under normal conditions in the human, ADH levels appear not to be rate-limiting in ethanol metabolism. Individuals with the "atypical" (fastatic) variant of ADH, which at physiological pH has 4-fold the activity of typical ADH, do not metabolize ethanol at rates significantly higher [illegible] to the rest of the population (131, 132). Electrophoretic studies reveal at least six different ADH forms (133). The relevance of these forms to metabolism of ethanol *in vivo* is not clear (122).

A special variant of ADH has been described which, unlike the other forms, is not inhibited by pyrazole and has a high K_m (6 to 10 mM as opposed to 0.5 to 2 mM for other forms) (134). This enzyme may become active at high ethanol concentrations in blood. However, its participation in ethanol metabolism *in vivo* is also unknown.

Lieber and coworkers described a microsomal (MEO[illegible]) system that could also become active at high ethan[illegible] concentrations (135, 136). This system is linked to cytochrome P450 and requires participation of NADPH [illegible] oxygen to transform ethanol into acetaldehyde. The [illegible] is 8 to 9 mM ethanol (137). This system appears to [illegible] different from alcohol dehydrogenase and catalase; however, the extent of its participation is uncertain (122, 1[illegible] 138). An important aspect of the interaction of alcoh[illegible] with MEOS is that the microsomal mass increases follo[illegible]ing chronic alcohol treatment (35, 135, 139). Since t[illegible] cytochrome P450 system metabolizes many compoun[illegible] into both inert and toxic compounds (140), the latter m[illegible] be important in the interaction of endogenous and e[illegible]ogenous compounds in the alcoholic (141, 142).

Chronic ethanol ingestion leads to metabolic toleran[illegible] with an increased rate of ethanol metabolism (137, 14[illegible] 143-148). In humans and monkeys, increases up to 100[illegible] have been reported (143-148) while, in rats, the increas[illegible] range from 30 to 70% (45, 123, 146). Lieber and cowerke[illegible] (137) suggest that such increases in ethanol metabolis[illegible] are due to an increase in microsomal mass and MEO[illegible] activity. Our studies in Toronto (123, 124) and those b[illegible] Thurman in Philadelphia (45, 125) suggest that an i[illegible] crease in ethanol metabolism follows an increased ox[illegible] dation of NADH and greater oxygen utilization by m[illegible] tochondria. The latter is believed to be due to increase[illegible] utilization of adenosine triphosphate (ATP) which i[illegible] creases the availability of ADP to mitochondria (45, 125[illegible] Both increased liver oxygen consumption (149) an[illegible] ethanol metabolism are abolished by administration o[illegible] the antithyroid drug, propylthiouracil (150). Thus, th[illegible] hypermetabolic condition in the liver of chronically al[illegible] cohol-fed animals appears to be thyroid-hormone deper[illegible] dent (44).

During alcohol metabolism, up to 60 to 80% of oxyge[illegible] consumed by the normal liver is utilized in the oxidatio[illegible] of ethanol to acetate (151). An increased rate of ethano[illegible] metabolism following chronic ethanol consumption b[illegible] the MEOS system or by an increased rate of mitochon[illegible] drial reoxidation of reducing equivalents should increase[illegible] oxygen consumption by the liver. We have emphasized the importance of expressing liver oxygen consumption as oxygen consumed by total liver rather than per unit o[illegible] liver weight (11). This is necessary since chronic ethano[illegible] administration increases liver size largely due to an in[illegible] crease in intracellular water rather than in the numbe[illegible] of cells (147-152).

Whether an increased rate of oxygen consumption is a mechanism for increased metabolism of ethanol has been challenged by other investigators who studied isolated hepatocytes (46, 153) and isolated perfused liver (154). However, with the exception of a well-controlled study by Cederbaum et al. (47), metabolic tolerances (rate of alcohol metabolism) were not reported and thus it is not clear whether their *in vitro* preparations were metabolizing more alcohol. Isolated hepatocytes may be abnormally leaky, as shown by stimulation (75%) of oxygen consumption upon addition of succinate (47). Our studies show that the hypermetabolic state occurs in hepatocytes isolated from alcohol-fed animals in which viabilities are

over 90% and succinate-stimulated respirations does not exceed 15 to 20%. Increased oxygen consumption in hepatocytes from alcohol-fed rats is directly related to cell viability (155).

While the hypermetabolic state produced by chronic ethanol consumption requires the presence *in vivo* of thyroid hormones, the levels of these hormones are not increased in the blood of ethanol-fed rats (154) or alcoholics (156). Some investigators (154, 157) have confused a permissive role of thyroid hormones with the need for increased circulating thyroid hormone levels or generalized hyperthyroidism. It is not necessary to have an increased circulating levels to explain a permissive effect of thyroid hormones or the effect of propylthiouracil (PTU). Acute ethanol administration displaces thyroxin from plasma proteins and increases the accumulation of thyroxin by the liver (158). A localized hyperthyroid state resulting from an increase in the number of thyroid hormone receptors in the liver is also possible in the absence of changes in circulating thyroid hormones (159). Normal thyroid function appears to be necessary for production of hepatic damage by choline-deficient diets (169), carbon tetrachloride (161, 162), anoxia (163), chloroform (164), acetaminophen (165), and organomercurials (166). Each of these conditions is prevented or reduced if the animals are previously thyroidectomized or treated with antithyroid drugs. In other studies, administration of thyroid hormones increased liver damage produced by chloroform (164), carbon tetrachloride (161), or anoxia (163).

We have postulated that an increased oxygen requirement in the liver following chronic alcohol consumption results in a steeper oxygen gradient along the sinusoidal length. Zone 3 (centrolobular area), the periphery of the liver acinus (167), has a relative state of hypoxia compared to Zone 1 (periportal) (168, 169). Cell necrosis occurs in alcoholic hepatitis preferentially in Zone 3 (170). Thus, it is conceivable that necrosis in this area may be hypoxic.

Not every alcoholic has Zone 3 necrosis and an increase in the rate of oxygen consumption by the liver is not by itself sufficient to cause cell death. Several factors may precipitate a critical state of hypoxia in which cell viability is no longer possible. Complications which occur often in alcoholics and may reduce the availability of oxygen to critical levels include anemia derived from gastrointestinal bleeding (171, 172), malnutrition or alcohol-induced metabolic abnormalities (173, 174), pulmonary dysfunction induced by alcohol itself or infections (175, 176), smoking (177, 178), changes in the hemoglobin saturation curve (179), reduction in liver blood flow, etc. (180). Multiple additive combinations of these factors are likely to occur in alcoholics.

We tested the "hypoxia hypothesis" by subjecting chronic alcohol-fed animals to three conditions of reduced availability of oxygen to the liver. These were: (a) atmospheres containing low oxygen tensions (149); (b) anemia induced by bleeding (181), and (c) ligation of the hepatic artery (182). Each condition resulted in necrosis in Zone 3 in alcohol-fed animals, but not in controls. Necrosis in Zone 3 was blocked by PTU and methimazole, two antithyroid drugs which act at different levels (183). Rats made hyperthyroid (184) or given ureogenic aminoacids (185), two other conditions that result in increased oxygen consumption by the liver, have Zone 3 necrosis when exposed to hypoxia. In the latter condition, necrosis was prevented by previous administration of PTU (185).

Following alcohol withdrawal in the rat (186, 187), liver blood flow decreases while, in man and baboons, hepatic venous oxygen tension is reduced (188–190). Conversely, oxygen tensions in the hepatic vein are elevated in baboons and dogs (190, 191) following administration of large, intoxicating doses of ethanol. This effect is primarily due to increased hepatic blood flow. In rats chronically treated with ethanol, an increased portal systemic shunting may occur (186) which may redistribute blood away from hepatocytes. Such an effect could produce higher venous oxygen tensions which do not represent events at the end of functional sinusoids.

The results of studies in man on the effect of ethanol on splanchnic blood flow are contradictory. Stein et al. (192) infused ethanol at 0.5 to 0.8 gm per min and found a 21% increase in splanchnic blood flow and cardiac output. Other investigators infused ethanol at 0.09 to 0.12 gm per min and found no change or a decrease in splanchnic blood flow (193, 194). Ethanol may increase splanchnic blood flow at high concentrations and have no effect or decrease splanchnic blood flow at lower concentrations.

The evidence that hypoxic liver damage can be induced preferentially in alcohol-fed animals and the protective effect of PTU led to a clinical trial of PTU (195) in treatment of alcoholic liver disease. The first study in which PTU or placebo were administered for a maximum of 45 days to 133 recently abstinent alcoholics gave encouraging results. PTU-treated patients showed a faster and more profound recovery in a variety of clinical and laboratory indices. In accordance with the working hypothesis, a positive effect of PTU occurred in patients with active alcoholic hepatitis, with or without cirrhosis, but not in patients with inactive cirrhosis in whom hepatocellular necrosis was absent. PTU is not expected to be effective if treatment is started long after ethanol withdrawal, since the hypermetabolic condition subsides with time. In our clinical trial of PTU (195), patients with alcoholic hepatitis were started on PTU within 1.9 ± 0.4 days of alcohol withdrawal. This constraint existing in acute studies is not encountered in chronic studies with ambulatory patients, the majority of whom continue to drink (196). Another consideration in short-term trials is that PTU which, in animal models, protects by blockade of thyroid gland activity (176), is likely to have a lag time for depletion of circulatory levels of thyroid hormones. Thus, it is not expected to act in patients in whom death is imminent. The lower mortality in patients treated with PTU was not statistically significant (195). It can be observed in tables for rates and proportion (197) that to avoid a Type II error for a population with a mortality of about 20%, detection of a 50% reduction in mortality by PTU would require a minimum of 474 patients in the study. For detection of a 25% reduction in mortality, 1,528 patients would be required (197, 198). This simple analysis was disregarded in a recent study (195) in which, with 67 patients, the authors concluded that PTU did not alter the mortality rate. The same study concluded that PTU did not accelerate the improvement of liver tests. Further interpretation of these data awaits publication *in extenso*.

Role of Acetaldehyde

Mole to mole, the alcoholic's biological system is as much exposed to acetaldehyde as to ethanol (122). Acetaldehyde is reactive and toxic and binds nonenzymatically to phospholipids (200), amino groups of amino acid residues, and sulfhydryl groups (106, 201). Acetaldehyde reacts with serotonin, dopamine, and norepinephrine, yielding pharmacologically active compounds (201, 202).

In general, one can separate studies on the effect of acetaldehyde into those showing alterations which may directly or indirectly result in cell death and those showing alterations in specific cell functions which cannot be linked to cell destruction. The extent to which the latter changes may result in altered hepatocytic function of pathogenic importance is impossible to ascertain. Among the first are studies correlating acetaldehyde with production of neoantigens (88). Acetaldehyde may also activate cytoxic lymphocytes (87, 90) directed against the liver. The significance of these findings (104, 105) is discussed in another section of this review. Secondly, an interesting report indicates that large doses of the product formed from condensation of noradrenaline and acetaldehyde produce massive hepatocellular necrosis in rats (203). A third mechanism which could indirectly lead to cell necrosis is production of lipid lipoperoxides and free radicals (204) which have been implicated in the pathogenesis of cell necrosis by carbon tetrachloride (205), acetaminophen (206), bromobenzene (207), and other hepatotoxins.

Acutely, large doses of ethanol reduce hepatic glutathione levels which serve as a scavenger for free radicals (208–211). Acetaldehyde is a likely candidate for this effect since the effect of ethanol is abolished by pyrazole and is enhanced by disulfiram which inhibits acetaldehyde dehydrogenase (212). The decrease in hepatic glutathione levels after ethanol may result from complexing of acetaldehyde with L-cysteine to form a hemiacetal and further transformation to L-2-methylthiazolidine-4-carboxylic acid (213, 214). Administration of the β-mercaptoethylamines, cysteine, and methionine increases the survival of mice given ethanol at lethal doses (208).

The microsomal drug oxidizing system can generate OH^- and O_2^- (26, 215, 216). In alcohol-fed animals, free radicals may be produced in large amounts because of increased microsomal mass which, when combined with reduced glutathione levels, may result in hepatotoxicity. This hypothesis is consistent with the finding that, in naive rats, large doses of ethanol (6 gm per kg) are required to produce detectable evidence of lipid peroxidation. In animals receiving alcohol chronically, a dose of 3 gm per kg of ethanol can produce peroxidation which is prevented by administration of the glutathione precursor, methionine (210). Peroxidation occurs only at maximal levels of glutathione reduction (211), which may explain some discrepancies regarding production of lipoperoxides following acute ethanol administration (217, 218). Videla et al. (211) propose that these discrepancies may be due to the time after ethanol administration at which the lipoperoxides were measured. Reduction in glutathione levels is time-dependent and relates to the dose of alcohol administered. It should be mentioned that recent studies (219) cast doubt on the relationship between diene formation, which is believed to correlate with free radical formation and lipid peroxidation, and cell necrosis.

Among the effects of ethanol which alter cell functions and in which acetaldehyde appears to be involved is the synthesis and secretion of liver proteins. Mookerjea and Chow (220), Sorrell et al. (221, 222), and Baraona et al. (223–226) have shown that secretion of newly synthesized glycoproteins and albumin is inhibited by alcohol *in vitro* and *in vivo*. Acetaldehyde appears to be involved in both of these effects because they are abolished by pyrazole and addition of acetaldehyde mimics the effects of alcohol (224). Baraona and coworkers (224, 225) suggest that protein export is impaired by acetaldehyde through an effect on microtubules which are required for export of plasma proteins from the hepatocyte. Acetaldehyde appears to interact with microtubules by competing for the site of colchicine binding. Following alcohol administration, microtubules have been reported to be shorter and thicker than in controls (224).

In addition to inhibiting protein secretion, alcohol inhibits liver protein synthesis in fasted animals *in vivo* and in isolated perfused liver (227, 228), and in hepatocytes *in vitro* (229). Since, in the absence of chronic liver disease, alcohol consumption does not lead to marked changes in circulating proteins, it has been suggested (226) that these effects are related to the relative lack of amino acids required for protein synthesis in the studies *in vitro* or in fasted animals *in vivo*. By this interpretation, changes in the redox state produced by ethanol further reduce the levels of amino acids, such as alanine, and inhibit protein synthesis. Addition of methylene blue or pyruvate counteract changes in the redox state produced by ethanol and abolish the inhibitory effects of ethanol on protein synthesis *in vitro* (226).

In summary, acetaldehyde can react with amino groups of proteins and phospholipids which may explain alterations in protein synthesis and export, and mitochondrial alterations. Acetaldehyde condensation with membrane proteins may form neoantigens of importance in cell death by immunological mechanisms. Reaction of acetaldehyde with smaller molecules, such as glutathione, may promote lipid peroxidation. In addition, acetaldehyde inhibits the transformation of pyridoxine into pyridoxal (5) phosphate, which contains an aldehyde group (230). Except for possible hepatotoxicity of acetaldehyde complexes with norepinephrine, the significance, in terms of liver pathology, of formation of condensation products of acetaldehyde *in vivo* is not known.

Calcium as Mediator of Cell Death

In 1969, Judah (231), in a review of mechanisms leading to hepatocellular death, concluded: "We are now acquir-

ing considerable knowledge which tells what one can do to a cell metabolically and *not* kill it." Knowledge about what kills a cell is much less certain. According to Farber (232), cell death may not be a passive phenomenon, as is generally conceived, but rather an active one in which the essential step is an induced response of the cell. If the classical concept is considered to be a cell homicide, Farber sees cell death as self-inflicted, or cell suicide. Cell death does not necessarily result from almost complete inhibition of protein and RNA synthesis (233), extensive disaggregation of polysomes (232), severe depression in ATP and adenine nucleotide content (234), large shifts in K^+ and Na^+ (233), loss of respiratory control or adenine nucleotide translocase activity, decrease in the heme portions of cytochromes aa, and C+Cl, dinitrophenol-activated ATPase, and the ability of dinitrophenol to stimulate O_2 uptake, or changes in mitochondrial ultrastructure characterized by swelling, loss of a tightly folded and contorted inner membrane, and appearance of amorphous matrix densities (235).

For the past 20 years, numerous studies reveal that a common concomitant of cell death by a number of hepatotoxins (236–238) and hypoxia (239–242) is massive accumulation of intracellular calcium. It is not clear whether Ca^{++} enters the cells as a result of terminal damage which results in necrosis. This would not be surprising as there is a gradient of calcium from extracellular fluid ($10^{-3}M$) to a free intracellular concentration (10^{-6} to $10^{-7}M$) (243).

In a fascinating study in isolated hepatocytes, Schanne and coworkers (244) showed that Ca^{++} is necessary in the extracellular medium for hepatocyte necrosis to occur when the latter is triggered by eight different hepatoxins. Whereas ethanol does not cause necrosis of hepatocytes *in vitro*, the combination of ethanol with sublethal concentrations of several hepatotoxins results in hepatocellular necrosis which is Ca^{++} dependent (245). This is an exciting area of current research. In other systems, agents such as phenothiazines which protect against cell necrosis, inhibit calcium flux (246). Phenothiazines prevent necrosis induced by chemicals (236, 238, 247), anoxia (235, 239), diets (248), and virus (249).

Chien and coworkers (240, 241) suggest that Ca^{++} influx activates phospholipases, which deplete cell membranes of crucial phospholipids and impair essential processes including active extrusion of Ca^{++} into the extracellular compartment (250, 251).

Hepatocyte Enlargement

Hepatocyte enlargement and hepatomegaly are common alterations in alcoholic liver disease (14, 23, 252–255). About 80 to 90% of alcoholics with liver disease have clinically detectable hepatomegaly (14). Despite this very high prevalence, little research has been conducted in this area. Historically, researchers and clinicians may have arbitrarily considered hepatomegaly as resulting from fat deposition and, therefore, not deserving specific attention beyond the mechanisms that led to fatty liver. Many investigators have considered that fatty liver is a benign condition which quickly disappears after alcohol withdrawal (252).

Interest in this area was renewed by Baraona and coworkers (152, 223), who analyzed the contribution of hepatocyte enlargement to hepatomegaly in animals fed alcohol chronically. As shown by Baraona et al. (152) and later confirmed by us (147, 256), hepatomegaly is caused by an increase in hepatocyte size and not by an increased number of hepatocytes. Total fat accounts for only 20 to 30% of the increase in liver weight. Total protein content accounts for approximately 20% of the increase in liver weight and about 50 to 60% is accounted for by an increase in water. We have observed that all the increase in liver water can be accounted for by an intracellular increase in water, without a change in extracellular water (147, 256). This reduces the ratio of extracellular/intracellular water and indicates a relative compression of the extracellular space. We have proposed (147, 256) that the semirigid liver capsule restricts the expansion of the hepatocytes which enlarge mainly at the expense of the extracellular and vascular compartments.

An increase in cell volume is likely to compress capacitance and resistance vessels and to increase intrahepatic pressure. We have observed that hepatomegaly of 50% obtained in young rats fed alcohol chronically is associated with an increase in intrahepatic pressures of about 60% (256, 257). Smaller hepatomegaly, obtained by withdrawing alcohol from the diet for 4 to 8 days, or in old animals, did not increase intrahepatic pressures (258, 259). Moderate cell expansion is likely to occur primarily at the expense of capacitance (low resistance vessels) in the liver without increasing intrahepatic pressure.

In alcoholics with hepatomegaly and clinical liver disease, hepatocyte size correlates positively with intrahepatic (256) and wedged hepatic pressures (Blendis et al., unpublished observation), *independently* of the histological diagnosis of fatty liver, alcoholic hepatitis, or cirrhosis (256). When patients with cirrhosis are differentiated by the presence or absence of elevated portal pressures, hepatocellular size is a strong predictor of the pressure abnormality (257). When serial biopsies and pressure determinations were performed, a marked increase in hepatocytes size always accompanied an increase in pressure and the contrary was observed in all cases in which hepatocyte size was reduced (257).

These observations are provocative because they deviate from the classical concepts on the production of portal hypertension. It is currently believed that nodule formation in cirrhosis compresses the venous tract outflow and is the most important factor in the production of portal hypertension. If this were true, portal hypertension should be largely restricted to cirrhosis; however, normal intrahepatic pressures occurred in 25% of patients with well-established alcoholic cirrhosis (257). About 40% of patients with serious alcoholic liver disease without cirrhosis have elevated intrahepatic pressures in the same range as those in patients with cirrhosis. Elevated portal pressures occur in alcoholic fatty liver and in hepatomegaly induced by short-term drug treatment (260). Baboons fed alcohol chronically may have increased portal pressure in the absence of cirrhosis (261). Although portal hypertension appears to be an early manifestation of alcoholic liver disease, these studies do not negate the possible role of nodules or fibrosis in causing, contributing to, or sustaining portal hypertension.

Fibrosis in Alcoholic Liver Disease

Fibrosis is an important morphological landmark of chronic liver disease. Some authors consider that fibrous tissue deposition may reach an irreversible state, the so-called "committed precursor state", from which there is either inexorable progression to cirrhosis or, at least, no restoration of normal liver, leaving a permanent scar (262). Most authors consider cirrhosis as irreversible (263), although this is controversial (264). There are many reasons why cirrhosis has a bad prognosis. As liver loses its normal architectural pattern, functional and hemodynamic abnormalities develop, and liver acini are replaced by regenerative nodules which contribute to portal hypertension (262, 265). Abnormal venous communications shunt blood from the portal vein directly into the hepatic veins (266, 267). Furthermore, sinusoids are altered by deposition of collagen in the space of Disse and eventual formation of a continuous membrane under the sinusoidal epithelium. The latter abnormality has been called "capillarization of the sinusoids" (268). These alterations impair liver microcirculation by reducing sinusoidal blood flow and reduce the exchange of substances between the blood and the liver.

Several types of cells are implicated in collagen synthesis: fibroblasts (269), parenchymal cells (270, 271); Ito cells or perisinusoidal lipocytes (272–274), and myofibroblasts (274, 275) [contractile fibroblasts which have not been observed in noncirrhotic livers, but are seen in cirrhosis (276)].

Fibrous Tissue in Alcoholic Liver Disease

In cirrhosis, the total content of liver collagen is increased by 2- to 6-fold and involves all types of collagen and noncollagen components of fibrous tissue including fibronectin, laminin, myofibrils, and elastin (76, 264, 277–282). In livers with less than 20 mg of collagen per gram of wet weight, the ratio of Type I/Type III collagen is the same as that in normals; in those with more than 20 mg of collagen, Type I appears to be predominant (281). Of special interest because of its implications to hepatocyte function, is the marked increase in perisinusoidal collagen, including collagen Type IV and the noncollagenous basement membrane protein, laminin (283), which is not detected in the Disse space of normal livers (281, 284). There is also an increase in basement membrane collagens and components A, B, and E (278, 280, 284–289). The increase in collagen IV, fibronectin, laminin, and collagens A, B, and E in the perisinusoidal spaces correlate with morphologic capillarization of the liver sinusoids (268), which is often observed in active and late cirrhosis (284).

At the stage of fatty liver formation, here is an increase in interlobular Type IV collagen, fibronectin, and laminin (284). In rats after 6 months of alcohol administration, despite no light microscopic evidence of fibrosis or necrosis, there is an increase in the fibrous and ground substance components of collagen (290). In alcohol-treated baboons, a moderately increased synthesis and accumulation of collagen and activity of proline hydroxylase occur at the stage of fatty liver in the absence of necrosis (291). In mice, chronic ethanol administration increases sulfated mucopolysaccharide synthesis and proline hydroxylase content, as detected autoradiographically in the walls of liver sinusoids (292). In baboons and mice, Ito cells proliferate (292, 293). In biopsies from human alcoholic hepatitis (but not fatty livers), there is an increase in total collagen (294), collagen synthesis (295), extractable glycosaminoglycurans and hyaluronic acid (277), and prolyl and lysyl hydroxylase activities (294, 296–300). In rats with cirrhosis which was chemically induced by various agents, an increase in prolylhydroxylase activity correlates with increased lysylhydroxylase (301, 302). The significance of increased prolylhydroxylase activity, as evidence by increased collagen synthesis, is not clear. Prolylhydroxylase activity may be increased in the absence of a detectable increase in liver collagen (264).

POSSIBLE FACTORS INVOLVED IN THE ACCUMULATION OF COLLAGEN IN ALCOHOLIC LIVER DISEASE

The accumulation of collagen in the liver can result from: increased synthesis, decreased degradation, or a combination. It has been assumed that fibrosis in cirrhosis results from collapse of preformed fibers in areas of cell dropout (303). Current evidence suggests that fibrosis results from newly formed collagen (298, 304, 305). The cause has not been defined and might result from the interplay of several factors.

Direct Effect of Ethanol

(a) Ethanol increases the incorporation of proline into collagen of chopped embryonic chick tibia (306), cultures of mouse hepatocytes (307), and liver biopsies obtained from patients with active alcoholic hepatitis (295). (b) By virtue of the change it induces in the redox potential, alcohol metabolism leads to formation of more lactate from pyruvate. Lactic acid may increase fibrogenesis by increasing prolylhydroxylase activity (308) or inhibiting proline oxidase (309, 310), an effect which results in marked increase in the proline pool. (c) It has been claimed that the rate of collagen synthesis is controlled by the availability of free proline in the liver (311, 312). The free proline pool is increased in conditions of increased collagen formation (313, 314). Proline occurs in higher concentrations in the serum of patients with alcoholic hepatitis (315). In rats treated with carbon tetrachloride, there is decreased proline oxidase activity and catabolism which may increase free proline and collagen synthesis (316). The malnutrition effect on reducing collagen synthesis in rats treated with carbon tetrachloride may be due to reduction in the proline pool (317). Resistance to the fibrogenic effect of ethanol in rats has been attributed to the finding that the proline pool does not increase appreciably in rats fed ethanol (290), although this finding is controversial (291, 318). Proline loading by diet in rats treated with carbon tetrachloride does not increase collagen synthesis (319). Circulating proline levels produced by ethanol may be due to

creased proline synthesis (318) or hepatocyte release (316). (d) Chronic ethanol administration increases collagen formation in response to a suture (catgut) implanted in the liver (320). (e) Immunological responses to neoantigens produced by ethanol or acetaldehyde. As discussed above, the supernatant of lymphocyte cultures, when exposed to autologous liver from patients with alcoholic hepatitis, stimulates collagen synthesis and this effect is enhanced by ethanol and acetaldehyde (295, 321). (f) As stated above, the concept that fat accumulation is responsible for fibrosis has been replaced by the view that necrosis is required. There is no doubt that cell injury is a powerful stimulus for fibrogenesis and it plays a role in the fibrogenesis after repeated episodes of alcoholic hepatitis (322–324). In animals, continued liver cell necrosis induced by hepatoxic agents (i.e., carbon tetrachloride) eventually results in increased fibrous tissue and cirrhosis (325). Nevertheless, the suggestion that cell necrosis is the only stimulus required for collagen synthesis may be too restrictive. For example, in baboons treated with alcohol, liver collagen increases and cirrhosis occurs in the absence of necrosis or Mallory hyalin (102). In experimental liver injury, formation of basal membranes and capillarization of sinusoids occur in the absence of necrosis (236). Sclerosis of the terminal hepatic vein, frequently seen in alcoholic liver disease, can occur in early states of the disease, before detectable necrosis (323, 327–329). (g) As discussed above, administration of ethanol may decrease the oxygen content of sinusoidal blood, particularly in Zone III, an area in which collagen formation is predominant. Anoxia increases formation of collagen in bone tissue cultures (330). (h) Recent evidence in arteries (331, 332), bile ducts (333), and kidneys (334) indicate that increased intracellular pressure stimulates fibrogenesis by increasing proline hydroxylase activity and collagen synthesis. Perhaps collagen synthesis in the Disse space follows an increase in pressure produced by the increased hepatocytic expansion following alcohol consumption (257, 259). In essence, intraluminal pressure in the liver may be a fibrogenic stimulus. We have shown an excellent correlation between intrahepatic (256) or corrected wedged hepatic pressure (Blendis et al., unpublished) and collagen desposition in the space of Disse. Thirty years ago, Moschcowitz (335) postulated that fibrosis in the congested liver (336), spleen (337), and in the wall of the portal vein (336) followed an increased pressure within these structures.

Decreased Degradation of Collagen

The turnover of collagen in rats receiving carbon tetrachloride changes from an initial 20 to 30 days to 300 days in the stage of irreversible cirrhosis (338). This change may result from: decrease in mesenchymal cells in fibrotic septa (339); reduced lysosomal activity of macrophages which phagocytize collagen (340, 341), or increased concentration of glycosaminoglycans (condroitin sulfate B and keratosulfate) which are more resistant to enzymatic digestion (338, 342). Mucopolysaccharase is necessary for removal of ground substance which accompanies fibrous tissue and is decreased to ⅓ of the normal activity in irreversible cirrhosis (76). Increased collagenase activity occurs in rats treated with carbon tetrachloride (343). In acute liver damage, i.e., induced by ethionine, the turnover of collagen is increased by 50% (344). Collagenase activity also increases after alcohol consumption in animals (345). Indirect studies by Henley (346) suggest that alcohol may reduce fibrous tissue degradation. Thus, the importance of collagenase in collagen accumulation in alcoholic liver disease is conflicting; enzyme activity decreased in some studies (347) and increased in others (345). The role of collagenase(s) and their specificity in collagen metabolism is not clearly understood (348, 349). The paucity of data in this area reflects methodological problems in measurement and identification of collagenase, and the lack of a small animal model of alcohol-induced fibrogenesis.

Pathogenic Implications of Collagen Accumulation

Because the liver forms several types of collagen which are produced by different cells in different locations, it is difficult to conceive that any increase in collagen content will always have the same functional impact. The fact that *some* individuals with well-established cirrhosis, as diagnosed by light microscopy, are asymptomatic with regard to functional alterations and have normal portal pressures (350) indicates that formation of septa and nodules may not have the pathogenic importance classically attributed to them (6, 14, 16–19). In some patients with cirrhosis, portal pressure falls rapidly upon withdrawal from alcohol (351, 352) which suggests that other factors play a role in the pathogenesis of portal hypertension. Generally, collagen in alcoholic liver disease is more prominent around the periphery of the acinus (Zone III) (76, 262, 323, 324, 353), surrounding the terminal hepatic vein (central vein) (323, 327–329) and in the space of Disse (275, 284–288). The spectrum of collagen in the space of Disse ranges from loose fibril deposition to thick bundles which widen the perisinusoidal space. In some cases, a continuous basement membrane transforms the sinusoids into capillary-like structures (268). Collagen in the space of Disse is independent of cirrhosis (256), separates hepatocytes from their milieu of exchange, and flattens the microvilli of the hepatocyte (32, 254, 259, 354). Thus, it is possible that increased collagen at this site impairs the transport of nutrients and oxygen between blood and hepatocyte. In our experience, functional alterations in alcoholic liver disease correlate with Disse collagen better than with any of the common light microscopic abnormalities including terminal hepatic vein sclerosis, Mallory hyalin, fat, inflammation, and necrosis (15, 354, 355). Of all of these, only necrosis correlated, albeit weakly, with the presence of collagen in the space of Disse (256). This is understandable if necrosis represents the end stage of a continuum of hepatocellular damage.

In essence, collagen in the space of Disse is functional, akin to intrahepatic shunting or collateral circulation in the sense that substances in blood are not available to the hepatocyte. Collagen in the Disse space and shunting of blood may potentiate each other in producing hepatocellular dysfunction, damage, and necrosis.

SUMMARY

It is clear that we have not been able to transform most of the information available in this field into knowledge. It should also be noted that no analysis by active researchers in a field can be devoid of bias, since investigators are more likely to tackle, in their own work, areas that intuitively seem more fruitful to them. Our review is no exception. We are critical of the classical histological characterization of alcoholic liver disease and believe that this might have misguided research because many alterations that define classical criteria have not been convincingly shown to correlate with hepatocellular dysfunction and hemodynamic alterations. We propose that cirrhosis, as diagnosed by light microscopy, *correlates* with more fundamental changes which occur not only in cirrhosis and lead to hepatocellular dysfunction and hemodynamic alterations. The mechanisms leading to hepatic dysfunction remain elusive. Cell necrosis is obviously at the end of a continuum from normalcy to irreversible hepatocellular dysfunction. Studies should continue to be directed to understanding the pathogenesis of necrosis which may reflect dysfunction of several processes in series. Protecting against necrosis in the last steps of the series does not guarantee protection against dysfunctions that lead to death or liver disease. Therefore, in this sense, the perspective in alcoholic hepatitis is different from that in fulminant hepatitis of other etiologies.

Emphasis has been given to collagenization of the Disse space. When compared with the morphological parameters that define the classification of liver disease as fatty liver, alcoholic hepatitis, or cirrhosis, collagen in the Disse space provides the best relationship with a combination of clinical and biochemical indices.

Two other areas that we have emphasized are the production by ethanol of a state of relative hypoxia in Zone III of the acinus and its role in hepatocellular necrosis, and the recent recognition of the relationship between hepatocyte enlargement, the reduction in relative extracellular space, and increase in intrahepatic pressure. Portal hypertension in alcoholics is closely associated with hepatocyte enlargement independently of the classical histological classification.

We have analyzed the information that appears certain and many hypotheses searching for a coherent explanation for alcoholic liver disease. Camus' (356) reflection on science may be relevant in this context: " . . . science that was to teach me everything ends up in a hypothesis . . . lucid wonders in metaphor. . . . I realize that, if through science, I can seize phenomena and enumerate them, I cannot, for all that, apprehend the world . . . . . and you give me the choice between a description that is sure but that teaches me nothing and hypotheses that claim to teach me but that are not sure. . . . "

Acknowledgment: We wish to acknowledge the excellent secretarial assistance of Evelyn Yates in the preparation of this review.

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the effects of chronic alcohol intake on basal ganglia diseases [3, 4]. Alcohol in large doses has been reported to have relieved the symptoms of unilateral parkinsonism [6], as well as to be detrimental to parkinsonian patients [5]. Mullen *et al.* [7], reported generalised chorea associated with alcoholism and Carlen [5] reported transient orafacial dyskinesia during ethanol withdrawal.

Chronic alcohol intake and withdrawal have been variously reported to cause subsensitivity of striatal dopamine-sensitive cyclic adenosine monophosphate [8], behavioural supersensituvity of dopamine receptors in the caudate nucleus and nucleus accumbens [9], and increased spiroperiodol binding sites in the neostriatum [10].

Hypersensitivity of dopamine receptor sites as a consequence of chronic dopamine blockade has been proposed to trigger Gilles de la Tourette syndrome [1, 2]. Chronic alcohol intake has been shown to block dopamine release in the striatum [11]. This could give rise to dopamine receptor supersensitivity [12]. At withdrawal of alcohol, the release of dopamine would no longer be blocked, resulting in hyperactivity of the dopaminergic system in the striatum with consequent involuntary movements.

In our patient, this would have been aggravated by underlying basal ganglia pathology (evident by the history of past habit spasms) that could have intensified the occurence of the abnormal movements and tics. Although the occurence of Gilles de la Tourette syndrome is rare, it is surprising that no other reports of its association with alcohol withdrawal has been made.

This is particularly so, in view of the enormous incidence of alcohol abuse coupled with the fact that alcohol is known to cause such variable effects in basal ganglia disorders [4].

This report however, suggests that alcohol withdrawal may occasionally precipitate the symptomatology of Gilles de la Tourette syndrome most probably by altering striatal dopaminergic balance.

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Underestimation of Recalled Alcohol Intake in Relation to Actual Consumption

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Summary

Drinking of 58 males was observed for six evening hours in simulated restaurant surroundings. Interviews on alcohol consumption were carried out the next day (in two cases two days) after the drinking session. Recalled mean number of drinks was ten drinks, the actual mean intake 11 drinks. Degree of underestimation in recall was positively associated with the actual amount imbibed. On the average, heavy consumers underestimated their intake by 12 per cent and light consumers by four per cent.

Introduction

One crucial question in measuring alcohol consumption is how recall of intake is associated with the actual consumption of the respondent. It has been argued that heavy drinkers tend to underestimate their alcohol consumption more than light drinkers but the evidence for this argument is far from convincing [1]. However, dissimulation, as measured by the Lie Scale of the Eysenck Personality Questionnaire, has been found to correlate negatively with reported alcohol consumption [2]. Such a tendency, if true for most drinking populations, could undermine results on the health and social risks of alcohol use and distort the observed relationships between alcohol intake and its correlates in general. Unfortunately, solid empirical evidence for or against selective under-reporting is hard to come by, since precise measurement of the actual alcohol intake is usually not possible. Some data are, however, available from an experiment where real drinking situations were simulated in order to study drinking behaviour. Data from this experiment have now been analysed focusing on the association between actual and recalled alcohol intake.

Subjects and methods

The subjects and the design of the experiment providing the present data have been described earlier in detail [3]. Briefly, the subjects were male skilled workers, aged 25–54 years, recruited by informal contacts from the metal industries in Helsinki. Alcoholics and those guilty of crimes of violence were excluded. The subjects were studied in groups of four, and members of a group were required to have shared at least one earlier drinking session together. The experiment involved 15 groups, all starting drinking at 6 p.m. and stopping at 12 p.m. in a room disguised as a restaurant room for private parties. The subjects were told that aim of the experiment was to study the influence of alcohol upon behaviour, and each group was given discussion tasks while the investigators observed social interaction behind a one-way mirror window.

Subjects were instructed that they were free to choose how much they like to drink. All drinks were provided free of charge by a waitress, who kept a log of the drinks. Behind the one-way mirror window, observers registered the actual intake and behaviour. Types of beverages included beer, vodka, gin, and brandy. The latter two were also available as pre-mixed long drinks. Transport to home was arranged to prevent continuation of drinking.

One day (in two cases two days) after the experiment the subjects were interviewed at lunchtime at their working place and asked how much they had drunk during the group session. Numbers of drinks and types of beverages were recorded. One of the subjects was not able to give a clear opinion, and the file of another case was not found for the reanalysis. The present study is thus based on 58 responses.

Results

On the average, the subjects had around 11 drinks but recalled having had 10 drinks during the experiment. There was considerable variation in the number of drinks consumed (Table 1).

Recalled intake correlated moderately with actual consumption. Spearman rank order correlation coef-

Table 1. Actual and recalled numbers of drinks

	Mean	Mode	Range
Actual	11.4	11	7–16
Recalled	10.5	10	6–15.5

ficient between recalled and actual number of drinks was .65 ($p<.001$), and the coefficient between recalled and actual amount of alcohol was .63 ($p<.001$).

Of the 58 males, 13 recalled exactly the number of drinks consumed, 22 were mistaken by one drink, while four overestimated and 19 underestimated their intake by more than one drink. The product-moment correlation coefficient between the underestimation in recalling (actual minus recalled number of drinks) and the actual number of drinks was significant ($r=+.34$; $p=.004$).

The sample was divided into light and heavy consumers with 11 drinks or less as a cutpoint to obtain two groups corresponding in size as closely as possible. This yielded 31 subjects who had imbibed 11 drinks or less and 27 subjects who had had 12 drinks or more. The mean degree of underestimation was four per cent among light consumers but 12 per cent among heavy consumers (Table 2). T-test (pooled variance estimate) indicated a significant difference ($p=.007$).

In contrast to the above, the actual volume of alcohol and the volume calculated on the basis of the recalled numbers of drinks and types of beverages did not differ (Table 3). The mean actual consumption was 17.5cl, and the recall estimate 17.6cl. Nor was the correlation between the actual intake and the error in recall estimate significant ($r=+.10$;$p=.22$). This apparent discrepancy

Table 2. Mean numbers of drinks among light and heavy consumers

Variable	Number of drinks	
	Light consumers (7–11 drinks)	Heavy consumers (12–16 drinks)
Actual intake	9.76	13.37
recalled intake	9.40	11.72
Difference	0.35	1.65
Number of cases	31	27

Table 3. Actual and recalled alcohol consumption (cl)

	Mean	Range
Actual	17.5	11.4–24.0
Recalled	17.6	10.1–26.2

was brought about by the fact that, on the average, the subjects underestimated less the number of drinks with high alcohol content (gin, brandy, vodka) than the number of drinks with low alcohol content (beer, long drinks).

Discussion

The experimental study providing the data of the present investigation showed, first, that group norms favoured heavy drinking, secondly, that drinking more than the other members of the group was esteemed and, thirdly, that drinking less than the others was disapproved [3]. Such a normative atmosphere could be expected to encourage over-reporting of alcohol consumption. In addition, forgetting was minimized, since the subjects were interviewed on the next day (in two cases on the second day) after the experiment. Even under these stringent conditions a slight underestimation in the mean number of recalled drinks was observed. The error in recall was clearly correlated with the actual alcohol intake. The higher the actual intake, the higher also the mean number of drinks left unreported. This finding suggests that heavy drinkers tend to underestimate their alcohol intake relatively more than light drinkers. Due to the nature of the study population this finding cannot, however, be generalized to alcoholics, who perhaps have the highest tendency to underreporting with probably the largest impact to the apparent distribution of alcohol consumption in general populations. Moreover, even if recall of number of drinks is influenced by the level of actual consumption, it is still possible, as in the present series, that this bias may be compensated for by differentials in recall of various beverage types. Finally, Spearman correlation coefficients were considerably below unity suggesting that ranking of individuals based on recall estimates may not correspond to the actual rank order of alcohol consumption. Whether this undermines studies on alcohol-related hazards awaits further clarification.

Acknowledgement

I thank Professor Kettil Bruun for his help and advice in this research.

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British Journal of Addiction 80 (1985), 217–
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Book Reviews

Measurement in the Analysis and Treatment of Smoking Behavior

Edited by J. Grabowski and C. S. Bell
NIDA Research Monograph 48. Department of Health and Human Services, Washington, 1983. 121 pp.

For many years research into smoking was seduced by its apparent simplicity. Here was a behaviour which provided psychologists with readily observable discrete acts easily counted and analysed. The focus on overt responses with its Skinnerian overtones naturally led to theoretical accounts of smoking which were couched almost entirely in behavioural terms. Psychologists were not the only ones to be beguiled. Epidemiologists too felt there was no need to go beyond self-reported smoking habits. Sometimes consumption might be multiplied by the tar yield of the cigarette smoke to derive an index of tar exposure, but no direct measures were made of the exposure of the respiratory tract to tobacco smoke and the dose of smoke constituents entering the blood stream.

This situation has now changed, and the last few years have seen something of a paradigm shift in the field. Biochemical markers of smoke intake have been developed and their application has forced a radical reappraisal of the nature of smoking. It has become apparent that self reported cigarettes per day is at best a crude measure of the quantity of smoke inhaled, and the relevance of tar and nicotine yields of cigarettes as measured by standard machine smoking has been seriously questioned by data showing the pervasiveness and overriding importance of individual patterns of puffing and inhalation that can generate similar intakes from cigarettes of widely differing nominal deliveries. In the area of smoking cessation biochemical validation of claims of abstinence has revealed deception rates that vary widely from one study to another and range up to 40 per cent. More generally there has been a shift in emphasis from purely behavioural to pharmacological aspects of the smoking habit. As measures of nicotine and its major metabolite, continine, have become available, so has interest focused more on smoking as a form of nicotine self-adminstration.

This volume indicates an increasing awareness of the need for account to be taken of these measurment advances. The monograph is a summary of presentations made at a meeting convened by the U.S. National Institute on Drug Abuse and the National Cancer Institute in August 1982. The topics covered range from the core one of the use of biochemical markers of smoke intake through to survey methods in the evaluation of children's smoking, and problems in the design and evaluation of cessation studies.

Benowitz provides an excellent summary review of biochemical markers, focusing on carbon monoxide, thiocyanate, nicotine and cotinine. As well as commenting on the specificity and sensitivity of each of these as a marker of smoke consumption, there is a welcome emphasis on the cost of each measure, and the point is made that choice of a biochemical test must be influenced by the research question at issue. if the aim is simply to categorize people into smokers and non-smokers, then expired air carbon monoxide is a simple and inexpensive measure providing adequate information. On the other hand blood cotinine concentration is probably the best measure to use to obtain a quantitative guide to daily nicotine consumption. Although the degree of individual variation in the fractional conversion of nicotine to cotinine is not yet known, it is provocatively estimated that a blood concentration of cotinine of 100 ng/ml represents an average 24 hour consumption of 12 mg of nicotine. If this relationship is confirmed it will permit a simple estimation of smokers' daily nicotine intake, a measure which would be of great value.

Kozlowski's chapter gives an interesting account of the historical origins of the 2 sec 35 ml puff/min. standard machine smoking regimen used for determining tar and nicotine yields. It turns out that the choice of these particular puffing parameters was largely arbitrary and never reflected smokers' actual puffing even on the high yield plain cigarettes sold in the 1930s. It comes as no surprise, then, that machine smoked yields of modern cigarettes should bear little relationship to yields as

MH-2A.15

SERIAL NO:

HOSP. NO:

PSYCH. NO:

UNIT :

ALCOHOL RESEARCH PROJECT - SELF REPORTED INFORMATION

AGE: SEX: NAME:

A. CAGE QUESTIONNAIRE

If the answers to the following questions are 'YES' encircle them:-

1. Have you ever felt you should cut down your drinking?
2. Have people annoyed you by criticizing your drinking ?
3. Have you ever felt bad or guilty about your drinking ?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hang-over ?

B. SADD QUESTIONNAIRE:

Think about your most recent drinking habits and answer each questions placing a tick (✓) under the most appropriate heading :-

	Never	Sometimes	Often	Nearly always
1. Do you find difficulty in getting the thought of drinking out of your mind ?	.....	.....	....	....
2. Is getting drunk more important than your next meal ?	.....	.....	....	....
3. Do you plan your day around when and where you can drink ?	.....	.....	.....	.....
4. Do you drink in the morning, afternoon and evening ?	.....	.....	....	.....
5. Do you drink for the effect of alcohol without caring what the drink is ?	.....	.....	.....	.....
6. Do you drink as much as you want irrespective of what you are doing the next day ?	.....	....	....	....
7. Given that many problems might be caused by alcohol do you still drink too much ?	....	....	...	....
8. Do you know that you won't be able to stop drinking once you start ?	.....	.....	....	....

9. Do you try to control your drinking by giving it up completely for days or weeks at a time ? ....

10. The morning after a heavy drinking session do you need your first drink to get yourself going ? ....

11. The morning after a heavy drinking session do you wake up with a definite shakiness of your hands ? ....

12. After a heavy drinking session do you wake up and retch or vomit ? ....

13. The morning after a heavy drinking session do you go out of your way to avoid people ? ....

14. After a heavy drinking session do you see frightening things that later you realize were imaginary ? ....

15. Do you go drinking and next day find you have forgotten what happened the night before ? ....

C. RELAPSE PRECIPITANTS INVENTORY :-

Encircle the appropriate facts concerning you.

DO YOU GET THE IRRESISTIBLE URGE TO CONSUME ALCOHOL ....

1. When you pass a pub or wine shop ?
2. When you are drinking with other people who are drinking ?
3. When you feel no one really cares what happens to you ?
4. When you feel tense ?
5. When you have to meet people ?
6. When you start thinking that just one drink would cause no harm ?
7. When you feel depressed ?
8. When there are problems at work ?
9. When you feel you are being punished unjustly ?
10. When you feel afraid ?
11. When you are on a holiday ?
12. When you feel happy with everything ?
13. When you have money to spend ?

14. When you remember the good time when you were drinking ?
15. When there are arguments at home ?
16. When you are full of resentments ?
17. When you feel irritable ?
18. When you are at a party ?
19. When you start thinking you are not really hooked on alcohol ?
20. When you feel yourself getting very angry ?
21. When there are special occassions like festivals and birthdays etc. ?
22. When you start feeling frustrated and fed up with life ?
23. When you feel tired ?
24. When you feel disappointed that other people are letting you down ?
25. When you have already taken some drink ?

D. AVERAGE FREQUENCY OF DRINKING DURING LAST 30 DAYS :-

Encircle the most appropriate heading conserning you.

1. Under 4 drinks a day. (One drink = 13.6 g/d of ethanol)
2. Between 4 to 8 drinks a day. (4 drinks = 60 g/d " ")
3. 9 or more drinks a day. (9 drinks = 120g/d " ")

E. ESTIMATE OF DURATION OF ALCOHOL CONSUMPTION :-

Answer the following questions regarding alcohol consumption as best as you can.

1. How long have you been consuming alcohol ?
2. How long have you been drinking alone without company ?
3. How long have you been drinking in the mornings ?
4. How long have you been getting 'shaking' of the hands if you don't consume alcohol ?

F. ESTIMATE OF THE PSYCHOSOCIAL DISRUPTION :-

Encircle if the answer is 'YES' for the following facts regarding you.

1. In the last 12 months have you had major family disruptions : eg. separation, divorce or threats of divorce ?
2. In the last 12 months have you had any difficulty with law due consumption of alcohol ?
3. In the last 12 months have you had difficulties with business/job due to consumption of alcohol ?

4. In the last 12 months have you had ~~xix~~ financial difficulties or debts due to alcohol consumption ?
5. In the last 12 months have you been changing friends or changing place of residence due to alcohol consumption ?
6. In the last 12 months have ever attempted suicide ?
7. In the last 12 months have been feeling sad for weeks or months at a stretch ?

IF YOU HAVE NOT ANSWERED ANY OF THE PREVIOUS QUESTIONS TRUTHFULLY PLEASE GO BACK AND CORRECT THEM. THIS INFORMATION REGARDING YOU WILL HELP YOUR DOCTOR IN TREATING YOU CORRECTLY.

DEPT. OF PSYCHIATRY. COMPILED BY DR. THOMAS M.J.

Serial No:

Hospital No:

Psychiat: No:
UNIT :
MRD No:

Date of Adm:

ALCOHOL DEPENDENCE - CHECK LIST

History taken by:- Reliable/unreliable

THIS IS A LIST OF SIGNIFICANT HISTORICAL FINDINGS. IF ANY OF THEM NEEDS ELABORATION, AS SOME WILL REQUIRE, PLEASE DETAIL THEM IN THE EXACT FILE, USING THE CODE NUMBER OF THE QUESTION IN THE LEFT-HAND SIDE MARGIN. (EG: A4, or B3 etc.)

A. EVOLUTION OF DRINKING PATTERN

Fill in the duration (in number of years), prior to the present consultation, the symptoms listed below manifested itself:-

1. Social drinking.
2. Drinking alone.
3. Morning dinking.
4. Preference to drinking companions and bars etc.
5. Excuses from work for variety of reasons because of alcohol.
6. Shifting from costlier forms of alcoholic beaverages to cheaper forms.
7. Repeated consious attempts at abstinence.
8. Alcoholic 'Black-outs'.
9. Patients subjective complaints that he cannot stop drinking once he starts with a small quantity (Loss of control).
10. Alcoholic tremelousness (Early morning shakes).
11. Alcoholic halucinosis (Auditory hallucinations).
12. Withdrawal siezures (Rum fits).
13. Delirium tremens.
14. Drinking despite strong medical contraindications to ~~the~~ known to the patient.
15. Accidents while intoxicated with alcohol.

mortality associated with abstention was confined to those who had become abstainers and was not found in lifetime abstainers.

A major difficulty is in the inference that *not* drinking is the cause of the slightly greater mortality in the abstainers. Personality characteristics, believed to be relevant to death from heart disease, have not been studied in abstainers: for example abstainers might contain a greater proportion of those driving, ambitious individuals prone to coronary disease than light drinkers.

More important is that (i) the effect noted by Marmot *et al.* is very small (the relative risk of abstainers is only 1·6 that of light drinkers); (ii) it apparently occurs with reported drinking as low as 1 unit (half a pint of beer) a week to 1 unit a day, which is a small dose if a direct pharmacological action is hypothesized; (iii) it disappears at fairly low levels of drinking (4 units/day); (iv) it is not demonstrable in the 40–49 year olds, only in the 50–64 year olds; (v) a similar effect is not demonstrable in the study already described of Dyer *et al.* (data analysed by me). Further specifically designed studies are now required.

5 Natural History of Problem Drinking

Epidemiologists have demonstrated that the problem drinker is not an individual irredeemably condemned but rather that people move into and out of problem drinking. Surveys record low rates of drinking problems after age 50. Drew (1968), examining the ages of alcoholics known to agencies, concluded that the prevalence of alcoholism in the population diminishes more rapidly with age than can be accounted for by mortality and successful treatment. One-half to one-third of respondents in two large US surveys who reported a given "problem" no longer reported that problem when re-interviewed 4 years later. Though some accrue a different alcohol-related problem in the meantime it is by no means inevitable (Clark & Cahalan, 1976). Ojesjo (1981) re-interviewed after a 15 year interval a general population cohort and found that of the 96 alcoholics identified originally 29 were now inactive or "much improved"; (25 had died). Work in this area has shown that, of the external influences, changes in social circumstances such as job and personal relationships are important.

6 Per Caput Consumption and Prevalence of Harm

A most pressing question is whether influences on mean consumption, such as availability of alcoholic beverages and prevailing attitudes to drinking, affect the prevalence of harmful consequences of drinking. Although this is related to the issue discussed above, of whether rises in mean consumption are linked with rises in proportion of heavy consumers, it is clearer to discuss each issue separately. It is, of course, the prevalence of harmful consequences that is practically more important than the numbers of heavy drinkers. Falling real price, increasing sales outlets and greater advertising, as already mentioned, may have had a role in increasing consumption since 1945. These factors are unlikely, however, to be sufficient explanations for the rise in prevalence of harm in this period. Other relevant changes in society include: increasing secularization, continuing weakening of the extended family and the blurring of sex roles, to name but a few.

Nevertheless, comparisons between nations and regions undoubtedly show covariation between per caput consumption and cirrhosis mortality. Furthermore, there is considerable evidence that within regions there is covariation over time between consumption and indices of harm (Bruun *et al.* 1975; Skog, 1980), though there is no example where a sudden decrease in per caput consumption has occurred that was not in wartime. This correlation is not seen so clearly in a recent report by de Lint (1981) of changes in the Netherlands in the period 1950–75.

Two of the examples cited above (section 2(vi)) of successive surveys in the same population provide data on changes in problem rates. In the Finnish study (Simpura, 1978), where per caput consumption increased by 67% from 1969 to 1976, an increase in the percentage of drinkers reporting problems also occurred: "Worrying about controlling my drinking" was reported by 15% of men in 1969 and 29% in 1976 (women, 4% and 14%); "social problems" were reported by 22% of men in 1969 and 32% in 1976 (women, 3% and 7%). However, the proportion having problems rose at every consumption level, meaning that either a certain amount of alcohol caused more problems in 1976 than in 1969 or, more likely, the threshold at which people admitted a problem had fallen.

The London comparison between 1965 and 1974 (Cartwright *et al.* 1978) also does not provide strong evidence. Although the authors found that an increase in admitted problems had accompanied the 47% rise in per caput consumption, 3 of the 5 problem items were rather trivial. Because of the small sample size in the 1974 study, only 2 individuals were identified who had had 4 items and none who had had 5.

It is not going beyond the current evidence to state that a change in per caput consumption is a marker of change in problem rates and, indeed, that recent trends (in particular increased availability) have made a contribution to rise in the indices of harm. What is not known is whether this association can be put into reverse, though it is widely advocated as the most expedient solution given the magnitude of the alternative task of educating whole populations to drink safely.

APPENDIX: SCREENING METHODS

i *Screening for Problems*

Questionnaires such as the Michigan Alcoholism Screening Test (MAST) (Selzer, 1971) and the Severity of Alcohol Dependence Questionnaire (SADQ) (Stockwell *et al.* 1979) have shown that a variety of populations will admit, in a pencil and paper exercise, to a range of alcohol-related problems and symptoms. Some of the MAST items are rather trivial. The SADQ restricts itself to "dependence" items. Where skilled personnel are available reliability can be improved by direct interviewing (Chick, 1980). Decisions about cut-off points in terms of number and severity of problems are an arbitrary matter. The time-frame in such instruments is also arbitrary. Many of the MAST questions are phrased in the form "Have you ever ...?", thus identifying "cases" who may now be "in remission".

EPIDEMIOLOGY OF ALCOHOL USE AND ITS HAZARDS *J Chick*

ii *Screening for Heavy Consumption*

As in survey work, consumption is best elicited by taking a very recent period and asking the subject in detail about each drinking occasion during that period. He should be asked to recall his leisure activities and his daily routine for each day, to jog his memory. In countries where drinking tends to be relatively infrequent such as Norway, the past 4 weeks is a suitable period. In Britain, the last 7 days suffice. It has been shown that in working populations those who claim their last 7 days were atypically heavy tend to be reporting a trivial difference (Chick *et al.* 1981b). However, in hospital samples, patients whose life leading up to admission was far from normal should be asked to detail a "typical" week. If they do not have a typical week, a "typical heavy week" is the next best measure.

Informants, such as spouse, have not been shown to improve the accuracy of self-reported consumption. However, several blood tests may be abnormal in regular drinkers and return to normal, with a roughly logarithmic decline over 2–3 weeks, when drinking ceases. Mean red-cell volume (MCV) and γ-GT are the cheapest and most commonly available of the existing tests, though they lack power. Chick *et al.* (1981a) describe their use as indicators of heavy drinking in working men. Fifty per cent of men who admit drinking over 450 g (56 units) a week have a γ-GT of >50 iu (false positives about 15%) and 23–32% have a MCV of over 98 fl (false positives about 5%), after men with other causes of increased values, such as taking anti-convulsants or specific physical disorder, have been excluded. Raised MCV is commoner in heavy smokers and in men whose pattern of drinking is sustained rather than episodic.

The false-positive rates, when self-reported consumption is the criterion, are in part probably due to lying and minimizing. The crucial study, of the risk of having a raised value on either of these two tests in a population whose consumption is known with certainty to be slight, has yet to be conducted.

Of the more expensive tests which have been proposed, abnormal heterogeneity of transferrin is still being investigated (Stübler *et al.* 1979) and the ratio of α-aminobutyric acid to leucine is probably only useful in detecting alcoholic patients who have drunk heavily and very recently (Chick *et al.* 1981c). Phillips (1981) has measured alcohol content of a sweat patch which collects for up to 10 days. The fact that drinking is being monitored may alter habitual consumption, however, and clearly a degree of co-operation is required.

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Counselling to check alcoholism

COIMBATORE

The Inner Wheel Club of Coimbatore main and the Rotary Club of Coimbatore jointly organised a seminar-cum workshop recently to consider the social problems created by alcoholism and drug addiction and to devise ways and means to prevent them besides rehabilitating those already affected.

The seminar was inaugurated by Rotary Governor Mr. P. N. Gopal of district 320 who emphasised the need for launching a campaign to educate the poor and the backward classes about the dangers of alcoholism and drug addiction and to mobilise opinion against consumption of alcohol.

The participants in the seminar included the past District Governor, Mr C. Govind, Dr. George Thomas, Dr. G. Lakshmipathy, Dr. Gurudas, Professors N. Surendra Prasad, Mrs. Devi Balakrishnan, Balasubramaniam, V. Ganesh, T. Jothimani and K. Muralidharan besides representatives of students from various educational institutions in the city. From the police side, Mr. Sreedharan, Additional Superintendent of Police took part in the discussions and explained the legal and enforcement aspects of prohibition and narcotic control. Mr. V. K. Jawaharlal, President, Alcoholics Anonymous and Mrs. Sarojini Anantharaman cited specific cases and family experiences with alcoholics.

In a paper presented on the occasion, Dr. G. Lakshmipathy dealt with 'Alcoholism and drug addiction yesterday, today and tomorrow'. He said the problem had assumed alarming proportions due to relaxation of prohibition and indiscriminate opening of arrack shops. Dr. Lakshmipathy pointed out that the future would be bleak if the problem was not tackled with a sense of commitment and with the overall welfare of society. He said the voluntary service organisations had a very important role to play in educating the vulnerable sections of the public about the evils of drinking.

During the panel discussion the causes for the spread of alcoholism were analysed by speakers. The participants expressed the view that lack of parental care mingled with peer group influence made adolescents victims of alcoholism. The view was expressed that in college campus alcoholism and drug addiction occurred due to curiosity or experimentation, inability to cope with academic stress, imitation of models, feeling of a false sense of insecurity and undesirable associations. It was suggested that alternatives to drinking must be devised and encouraged and supportive centres must be set up to provide counselling and guidance to students getting into the habit.

The doctor participants and psychiatrists clarified that creativity and intelligence were not stimulated in anyway by addictive behaviour but on the other hand it led to chromosomal abnormalities. It was explained how consumption of alcohol by pregnant women led to congenital defects, mental retardation etc., among children.

The speakers laid emphasis on the responsibility of the Government in preventing drug addiction and alcoholism. They said the Government instead of relying on arrack shops to increase its revenue should think of alternative methods of raising revenue and put an end to this social evil. The consensus of opinion among the speakers was that the Government should not shirk its responsibility for political gains and exploit the gullibility of the weaker sections but must act to improve the well-being of the socially and economically backward people.

The Inner Wheel Club of Coimbatore main has charted out a follow up programme on the basis of the recommendations of the seminar. The programme envisages distribution of handbills in both English and Tamil in educational institutions, slums, villages and offices explaining how the public at large could help in the eradication of alcoholism and drug addiction, display of posters urging the Government not to locate arrack and toddy shops in the vicinity of educational institutions, temples, hostels, industries, etc., closure of arrack shops on holidays and strict adherence to timings for the sale of liquor and strict enforcement of law that liquor shall not be sold to persons below 21 years.

The club has also decided to launch audio visual programmes to create awareness among the youth about the hazards of drug and alcohol addiction and to bring about better interaction between parents and children and also between teachers and children.—Coimbatore Staff Reporter

ALCOHOL AND DRUG USE AND RELATED PROBLEMS IN THE MEDICAL PROFESSION

PROFESSOR J.R.B. BALL*

At meetings where much learned discussion occurs about the misuse of legal and the use of illegal drugs within the general community, with comment about the psychosocial and biological causes of such behaviour, the context often suggests that the problems are all out in "society" but in fact they also are found within the helping professions. As well as looking at the "public's" problems the medical profession needs to look at its own frames of reference, personality problems, professional and social pressures and the other factors which contribute to doctors' misuse of such agents. Of course in recent times much critical attention has been paid to the medical profession. McCall's magazine commented, "physicians are poor husbands, poor fathers, absent companions, prima donnas and about as useless in bed as an electric blanket when the power is cut off". It might also be added that they have some other problems! So far within Australia local information is inadequate and inaccurate but the problems can be outlined by reference to work which has taken place abroad; one might assume that physicians in Australia may not differ much from those in U.K. and U.S.A.

Earlier it had been though that doctors, by virtue of their role and lifestyle, suffered a high incidence of physical disorder but this does not seem to be so. British doctors have a standard mortality ratio of 81, i.e. taking 100 as the norm; that is they are 19% better off than the general population in relation to age. Doctors standard mortality rate for cancer is 73% (1).

Amongst United States doctors, for 1938-1942 the mortality rate was equal to that of the general population, but less than for other professions, whilst between 1969-1973, the mortality rate for male doctors was 74.7% and for female doctors 84.1%, as against the general population matched for age. Relevant factors possibly related to the situation in Britain and the United States could be the physicians' good socio-economic status, easier recognition of illness and, earlier and more appropriate treatment being sought and given (2,3).

However United States doctors appear to have a high incidence of affective disorders, drug abuse and alcohol addiction (4,5,6,7,8). In relation to completed suicide this figure ranged from 27-39 per 100,000 per year, which was far higher than that found in the general U.S.A. population (9). Female physicians appeared to kill themselves at four times the rate of the general U.S.A. population. Lately whilst supporting the higher rate of suicide in female physicians - equal to that of their male colleagues, the general rate for physicians in the U.S.A. has been shown to be similar to other professional which is a better comparison than with the general population (10).

Doctors were also said to have unhappy marriages giving rise to the question - Do the unhappy marriages cause greater general vulnerability for physicians or does the physician's greater vulnerability and maladaptation plus other factors, help make their marriages unsatisfactory? (11,12,13,14)

British doctors have a standard mortality ratio for suicide of 335%, cirrhosis of the liver 311% and accidental injuries 180%, matched against the population (100%). Work in Scotland shows that physicians have a mean admission rate to

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psychiatric units of 449 per 100,000, whilst other social class I patients only reached 205 per 100,000. There was no significant difference in relation to admissions for schizophrenia, pre-senile psychosis or senile psychosis, but the differences were highly significant in regard to drug dependence, alcoholism and depression. The structure of health services and the pattern of their use, in the British Isles suggests that the figure could be a fairly accurate guide (2,3,15). It is also claimed that 1% of doctors in the U.S.A., are or will become narcotic dependent, whereas for the general population of the United States 1 per 3,000 are so affected. Information from the United States indicates that 10% of all physicians will become dependent on psychoactive drugs or alcohol sufficient to impair the practice of medicine at some time during their careers, that 10% of physician alcoholics or narcotic addicts, will commit suicide, and that 7% of all physicians are or will become alcoholics at some time during their careers and that one-half of the alcoholic physicians will become dependent on other drugs. More recent United States information (which does not quite match with that from the United Kingdom), is that the occurrence of alcoholism amongst physicians is similar to matched professional groups within the population but that narcotic addiction is 30-100 times more frequent than in the general population and also much higher than that found in matched professional groups within the population and sedative hypnotic abuse is also thought to be common amongst physicians (16,17).

Australian figures are almost certainly quite inaccurate, but here as found elsewhere, pharmacists, dentisits and veterinary surgeons seem to have little drug dependence, or at least are rarely reported to have such problems. One can argue that pharmacists and veterinary surgeons (animals can't explain or complain) can more easily cover up their drug misuse and dependence and avoid detection, whilst dentisits have less direct access to narcotics, hypnotics and sedatives than physicians. One can of course see cases from such professions within the patient population, but the general 'private' impression from colleagues working with the drug dependent and from local official sources is that dependency has been rarely officially reported amongst members of such professions and is infrequently seen in clinical practice. Such evidence clearly relates only to those who seek help, or are 'found out'. The Victorian state figures (18) over a 20 year period show that there have been sixty two (62) cases of serious drug dependence 'reported' of whom fifty four (54) were physicians, six (6) were nurses, one (1) was a dentist and one (1) a pharmacist. Pethidine or morphine was used in 46 cases and all of the other cases were multiple users of Fortral, Palfium, Percodan, and amphetamines. During this period the number of registered medical practitioners in Victoria rose from 5,000 + (est.)(1964) to 10,616 (1984). Many physicians know of numbers of colleagues who have never been reported to the Medical Board and/or to the 'drug dependency unit' when suffering from narcotic dependency and/or misuse of other drugs including alcohol.

If we assume that Australian physicians are similar to their colleagues in N. American and the United Kingdom, then we might expect that up to 1% are or will become narcotic dependent, that a relatively high proportion will become sedative or hypnotic dependent, that the use of alcohol will be a least comparable to that of matched groups within the population and possibly higher if we are closer to the British rather than the United States situation. (If it is not so then the explanation should prove illuminating).

The factors which may determine such differences from the general population and similar non-medical professional groups must include aspects of vocational choice, student selection, training and life style within the medical profession. 'Nice' vocational motives for entering the medical profession such as the search for honour, prestige, good income and the gratification of altruistic inclination certainly apply to many within the profession but a multitude of other factors may also affect such choice (14,19,20.)

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Dynamic influences may operate such as introspective identification with parents or some other significant figure who might have a healing or physician role, or compliance with the parental image of oneself, i.e. meeting the parental expectations in reference to vocational choice. A quest for omnipotence and expression of the need to dominate certainly seems to exist in many physicians; this may be associated with the denial of dependency by making others dependent upon one's self. A more esoteric possibility is the suggestion that one becomes a healer as part of a means of reparation of fantasy damage to initial love objects, in effect doing a 180 degree turn. The flight from death, the transmutation of anxiety about one's own death and the death of others, into the healing role where one combats death and associated with this the need to administer to others and not to oneself has also been suggested as an influence in some individuals. In like vein the patient is the shadow, in a Jungian sense, of the doctor; i.e. the sick or needful part of ourselves.

In regard to the suggestions of marital disharmony and problems within doctors' families, some suggest that partly this is the product of the difficulties which physicians may have with unqualified relationships, thus we 'find' vulnerable and sick people and marry them, or alternatively need the spouse to become a patient to whom the physician can then guardedly safely relate; a family member can become 'a patient' deliberately to get attention and consideration. The childrens' problems which emerge in some medical families, can illustrate a mechanism whereby the child becomes the 'delegate' or vicariously meets the parental behavioural needs which cannot be met within themselves or expressed without professional and/or social difficulties. Certainly in such ways the spouse or the child by illness or other abnormal behaviour achieves the attention of physician, parent or spouse and of society in some instances! (12,13,14,21,22)

Whatever the dynamic influence which determine a physician's vocational choice and operate within the doctor's immediate environment and in the family, a multitude of compounding problems also operate and can predispose to substance abuse. These include the tendency expressed by many physicians to deny problems within themselves; this is often associated with the overt or covert collusion of one's colleagues and/or family, who fail to see or will not see the tension, distress and even behavioural disturbance of the sick physician. This may represent a kind of misplaced love or friendship. One might consider that the real test of a relationship is that one is prepared to lose the friendship or love of the person for whom one is concerned by expression of the perceived need for help to that other person and even take more direct action if necessary. Overwork and fatigue are important influences which are to some extent related to the style of work and the situation in which it occurs; they are also an expression of the personality for which the medical student and then physician has been selected. Most physicians work hard and tend to overwork. Few doctors would not begin early and finish late, i.e. well beyond assigned and perhaps appropriate hours of employment. In most forms of practice, doctors work long hours and yet, all things being considered, for many of them it seems that the need which is satisfied is to work rather than to earn. In fact nowadays excessive hours and generation of extra income may produce financial rewards which prove simply uneconomic; this serves just to highlight the mechanism.

Compounding problems for the physician are the development of any physical illness, which tends to be denied, to be worked through or over, and easy access to drugs becomes a problem. Many cases of drug dependency begin when doctors in a state of exhaustion, depression and/or physical discomfort from one cause or another resort to medication to keep going, sometimes hypnotic/sedative drugs and far too often, narcotics.

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Underlying general problems which can have a marked influence on such matters are the indications of pre-disposing vulnerable personality, personal development and family history which if further confirmed all raise questions about selection for undergraduate training (20,23,24). Additional complicati factors are the lack of help at the undergraduate level to deal with emerging problems and clearly emerging as a major concern, having female gender. The problems for female doctors are quite serious, the medical role may provide greater difficulty for the female student and then doctor because of the highly competitive nature of the training and work, the demands which the female physician makes upon herself and the complex problems of role and role diffusion in relation to practice of medicine and the domestic and other responsibilities which female physicians may also assume.

For all doctors matters such as the assumed and assigned roles, the role model with which one identifies as a student then young physician, the arduous competitive nature of the training with limited opportunity (and often less encouragement) for the student to engage in other activities or interests, the illusional sense of or wish for omnipotence, the fear of failure and the horri reality that ultimately we all must fail add to the difficulties of the doctors vocation.

Divorce is a particular problem for physicians, the divorced in the general population are 3 times more likely to kill themselves than the married, whereas divorced doctors are 13 times more likely to kill themselves than their marrie colleagues.

Things which suggest existing or emerging problems amongst medical colleagues (as for anyone else) which might need help and exploration include the appearance of strain or obvious worry, decrease of efficiency and loss of ambition, new hypersensitivity to criticism, sudden and unreasoning hostility to almost any suggestion, undue argumentativeness and irritability, marked change in habitual personality reactions, peculiar behaviour or mannerisms not exhibited before, noticeable fatiguability, loss of weight, insomnia, and apparently inexplicable inappropriate disruption of domestic or other personal relationships.

Doctors classically give the reasons for drug abuse as overwork, chronic fatigue and physical disease. Several conditions which must co-exist to effect narcotic addiction in anyone, no less physicians include:-

a) a predisposing personality

b) the availability of drugs (an obvious problem for physicians)

c) a set of other circumstances which bring these factors together, plus

d) some situation which operates to trigger the actual use and to remove the inhibitions, for example stress/illness.

In considering what can be done to reduce or prevent such maladaptive behaviour some have suggested 'better' selection of those who are to be trained in medicine. Several studies indicate that physicians who later became drug dependent could have been spotted as vulnerable at the time of selection or whilst undergraduates and helped at that time (23). One hesitates to say whether or not vulnerable persons should be excluded from training or dismissed from medical school as our history includes too many very creative and able persons who for one reason or another became drug dependent for some while. Had they not been allowed to complete training or to continue or return to practise the profession might have been the loser and much more the patients and the society which they served. Certainly there seems to be the need for good observation and adequate support systems within

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the medical schools and throughout professional life the need for support from colleagues and family, with adequate provision of consultative and treatment facilities (24,25).

People handle stress in many different ways apart from the use of chemical agents. Common coping responses used by professionals include 10 common techniques: changing to engrossing non-work or play activity such as reading, community affairs, coaching sports, a variety of hobbies and outdoor activities: analysing the stress producing situation and changing the strategy of attack upon it, deciding what is worth worrying about and what is not, accepting less perfection and delegating tasks when tensions build: working harder, working longer hours, take on more responsibility: talking through with others on the job, discuss with contemporaries and having a bitching session with one's peers: changing to a completely different work task or job activity: talking through with spouse: withdrawing physically from the situation for a while, taking a break, engaging in physical exercise: trying to compartmentalise work and home life, working hard on the job but when at home learning to blank out job problems: building resistence to stress by regular sleep, regular exercise and good health habits. Overall the most effective techniques seem to be the last five which have been highlighted.

The basic survival kit for anyone, not least the physician, seems to include a sense of self respect, an appreciation of human limitations and of one's own, an ability to set priorities and to set limits, reasonable but not undue attention to oneself, a capacity to recognize and accept the natural course of events, the recognition of one's personal vulnerability and the development of satisfactory dependence on significant others in one's family and amongst one's colleagues.

Both in the direct interpersonal situation with drug dependent colleagues and in general, one needs to have a supportive, caring role and not a critical denigratory punitive and destructive one.

Many physicians who become dependent can be helped with good supervision, appropriate control of the work situation and regular and random serological and urinary controls to monitor drugs. Some will need intensive psychiatric help and this should be easily available (26,27). Most of all every physician and every student needs to bear in mind the classic first principals in this area:-

1. We are very well trained to take care of others and in general we do that very well.

2. We do not care well for ourselves and often take unkindly to having this pointed out to us.

3. We should never directly prescribe for ourselves any preparation which has an influence on our minds, hypnotic, sedative, tranquillizing, stimulating or whatever. The use of such drugs should be at the discretion of an appropriate colleague whom one is properly consulting.

If we were able to obey these rules this would help to minimize drug dependency amongst us. This would also be a further expression of our caring for those placed in our charge for the use of narcotic/sedative/stimulant preparations by ourselves, on ourselves, must carry the risk of some damage to our professional judgement and skills.

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ACKNOWLEDGEMENT

"THIS PAPER IS BASED ON VERBAL PRESENTATION ON DRUGS AND YOUTH ALTERNATIVES AND STRATEGIES GIVEN AT THE FIFTH ANNUAL CONFERENCE MELBOURNE JULY 1985 AND SUBSEQUENTLY PUBLISHED IN AUSTRALIAN DRUG AND ALCOHOL REVIEW JANUARY 1986 ISSUE VOLUME 5. NO. 1. THE AUTHOR IS GRATEFUL TO THE EDITOR OF THE REVIEW FOR PERMISSION TO PUBLISH THIS ARTICLE."

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Parkinsonism Provoked by Alcoholism

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Seven chronic alcoholics, aged 53 to 70, demonstrated transient signs of parkinsonism provoked by alcohol withdrawal or chronic severe intoxication. All showed improvement or recovery when they abstained or decreased their alcohol intake for several days to weeks. Animal studies have demonstrated impaired striatal dopaminergic function during severe ethanol intoxication or withdrawal. Chronic alcoholism apparently can exacerbate or uncover latent central dopaminergic deficiency.

Carlen PL, Lee MA, Jacob M, Livshits O:
Parkinsonism provoked by alcoholism.
Ann Neurol 9:84–86, 1981

Acute alcohol intoxication has been reported to trigger akathisia, dystonia, and cogwheeling in young adults taking neuroleptics [7], suggesting that alcohol can impair central dopaminergic mechanisms in humans. During the past three years we have observed transient parkinsonism during alcohol withdrawal or chronic severe alcohol intoxication in seven chronic alcoholics. Parkinsonism provoked by alcoholism has not previously been reported.

Patient 1

A 53-year-old man with a ten-year history of alcohol abuse (24 beers and several glasses of wine daily) entered the hospital for alcohol withdrawal. He had been admitted for alcohol withdrawal two and three years previously and had had documented alcoholic liver disease for seven years. One day after admission he was noted to have marked poverty of movement, bilateral resting tremor of the feet, generalized cogwheel rigidity, and a stooped, somewhat shuffling gait with loss of associated movements. Within five days all clinical signs of parkinsonism disappeared.

A CT scan showed mild to moderate generalized cerebral atrophy. An electroencephalogram (EEG) was normal. Liver function studies showed minimal elevation of bilirubin and serum glutamic-oxaloacetic transaminase (SGOT), and the patient had mild hepatomegaly. Parkinsonian features had been noted on two previous admissions.

Patient 2

A 56-year-old man had drunk 3 to 4 quarts of gin per week for the past twenty years. A month perior to admission he had increased his intake to 1 bottle of gin daily. Two weeks before admission he was noted to have a slow gait and tremor at rest. His wife, a nurse, related that during two prior drinking episodes the patient had shown similar signs of parkinsonism, which resolved with abstinence.

Examination showed an emotionally depressed man with slowed speech, resting tremor, and cogwheeling rigidity of the right arm. He had a postural tremor in both arms. His gait was wide based and he was generally bradykinetic. He had palmomental and snout reflexes. Three days later, it was noted that he had no parkinsonian tremor and his cogwheel rigidity had decreased. Nine days after admission, no rigidity was noted and his bradykinetic gait had greatly improved. Investigations in the hospital showed no biochemical evidence of liver disease. An EEG showed minimal abnormalities.

Patient 3

A 62-year-old woman who had been drinking up to 24 beers daily for twenty years had a one-year history of resting tremor in the arms which was reportedly increased during alcohol withdrawal. She was noted to have a shuffling gait, cogwheel rigidity and resting tremor in the arms, an expressionless and flat facies, emotional lability, and decreased insight into her condition. She was diffusely bradykinetic.

Four days after this visit, having maintained abstinence, she was admitted to the hospital with an unsteady, shuffling gait. Her parkinsonian signs had decreased. She had no evidence of liver disease. CT scan showed moderate ventricular and sulcal atrophy, and bilateral basal ganglia calcification. She was reassessed six weeks later and ostensibly had not used alcohol. Signs of parkinsonism were still present but were further diminished.

Patient 4

A 64-year-old man had drunk heavily for thirty years, including a quart of whiskey per day and 24 beers per week for the previous four months. Admission was prompted by a seizure. During the first 48 hours in the hospital the patient experienced withdrawal symptoms, including a postural tremor that was controlled with chlordiazepoxide. On the third day after admission he was noted to have a bilateral pill-rolling tremor of the upper extremities with a frequency of 3 to 5 per second. He had masked facies, positive glabellar tap, cogwheel rigidity of the arms, and

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Received May 1, 1980, and in revised form June 27. Accepted for publication July 5, 1980.

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 0364-5134/81/010084-03$01.25

bradykinesia. His posture was stooped, and he had a shuffling gait with loss of arm swing. Gradually over the next week, all clinical signs of parkinsonism resolved. A CT scan demonstrated mild to moderate generalized cerebral atrophy. The EEG showed mild, generalized slowing, and liver enzymes were modestly elevated.

Six and twelve months previously, during brief periods of alcohol withdrawal, he had noted a marked tremor which was quite different from the pill-rolling tremor of the present admission. However, he had also noted a slowness and stiffness of movement during the first days of each withdrawal episode.

Patient 5

A 66-year-old woman who had averaged 6 to 8 beers per day since the age of 30 had a long history of tremor (probably of the postural or benign essential type) which was decreased with alcohol intake and increased during withdrawal. For two years prior to admission she had complained of a pill-rolling tremor, particularly in the right hand, which increased and gradually spread to both arms. Six months before admission the patient noticed increased abnormal involuntary movements of the tongue and lips. There was no history of tranquilizer use.

On admission, two days after her last drink, the patient was noted to have masked facies and a slowed, stiff gait without associated arm swinging. Her gait was also ataxic. The glabellar tap response was positive. She had a coarse flexion-extension tremor of her fingers, greatest on the right side, which decreased during voluntary movements. Cogwheel rigidity was present in both arms. She had constant lip smacking and repeated protrusions of her tongue. Generalized hyperreflexia was present, but her plantar reflexes were flexor. She was disoriented to place and date and had impaired recent memory. There was no biochemical evidence of liver disease. CT scan showed marked cortical and generalized cerebral atrophy. Two weeks after admission the patient was started on Sinemet because of the signs of parkinsonism. She improved after one week. Sinemet was discontinued, and four weeks later she had a normal gait, no rigidity, and almost no lingual-oral dyskinesia.

Patient 6

A 70-year-old man had abused alcohol since the age of 18, particularly in the past ten years. Two years previously he had been diagnosed as having alcoholic liver disease on the basis of abnormal liver function studies, mild hepatomegaly, and an abnormal liver scan. He was admitted with severe alcohol withdrawal symptoms. His posture was stooped, and he walked in short, shuffling, unsteady steps with loss of arm swing. Over three weeks in the hospital his gait improved but did not return to normal. A second admission four months later was for severe alcohol withdrawal symptoms. Nine days after admission, neurological consultation showed impaired fine motor movements, bradykinesia, rigidity, stooped posture, and a shuffling gait with loss of arm swing. A CT scan demonstrated moderate cerebral atrophy. EEG showed mild generalized slowing. Over the next two weeks his gait again improved but was not completely normal. The patient had first noted a shuffling gait two years previously and had been aware of a deterioration of gait during previous episodes of alcohol withdrawal.

Patient 7

For several months a 70-year-old woman had complained of a progressive shuffling, slowed gait and impaired memory. Her husband had noted increased bradykinesia after several drinks, which decreased by the next morning. She had averaged at least 8 oz of vodka daily for many years. Examination revealed impairment in orientation, recent memory, calculation, and general knowledge. She had a slowed gait with loss of associated swinging of the left arm, resting tremor of the left arm, and decreased facial expression. She also demonstrated bilateral postural tremor and mild intention tremor of the left arm, impaired tandem gait, and mild proximal muscle weakness. Liver function tests were normal. CT scan showed moderate diffuse cerebral atrophy.

Reexamination three months later, after she had reduced her alcohol intake, demonstrated no resting tremor, a fluid gait with associated arm swinging, and more expression to her face. The signs of dementia and cerebellar dysfunction had also diminished.

Discussion

Although alcoholism-induced Parkinson disease has not to our knowledge been reported previously, this usually transient syndrome must not be rare. Our patients were all in the appropriate age range to develop idiopathic Parkinson disease. None were receiving neuroleptic drugs. Patients 1, 2, and 4 had a history of two prior parkinsonian episodes before admission. Patients 2 and 5 had a history of parkinsonian tremor, and Patient 6 had shown a mild shufflling gait one to two years prior to admission (Table). Patients 1, 3, 4, and 6 developed their parkinsonism during the first few days of alcohol withdrawal, Patients 2, 5, and 7 only during heavy drinking; Patient 3 had both modes of presentation. Patient 5 also developed a lingual-oral dyskinesia while drinking. Three patients demonstrated a withdrawal syndrome along with their parkinsonism.

This syndrome differs from the chronic progressive acquired hepatocerebral degeneration syndrome described in chronic alcoholics with severe liver disease and portal-systemic shunting [9]. Most of those patients have extrapyramidal neurological signs other than parkinsonism. Although three of our patients had mildly elevated liver enzymes and two had moderate hepatomegaly, none had other signs of liver disease and none had a history of hepatic encephalopathy, which was present in 80% of the series of Victor et al.[9].

The effects of ethanol on dopamine metabolism in the basal ganglia may help to explain the syndrome

Patient No., Age (yr), and Sex	Parkinsonism Provoked during: Alcohol Withdrawal	Parkinsonism Provoked during: Chronic Intoxication	Liver Abnormalities	CT Scan	Recovery	Other Comments
1. 53, M	+	–	Mild	Atrophy	Full, 5 days	Two prior episodes
2. 56, M	–	+	None	Not done	Almost complete, 2 wk	Two prior episodes
3. 62, F	+	+	None	Atrophy and basal ganglia calcification	Partial, 6 wk	Pill-rolling tremor for 1 yr
4. 64, M	+	–	Mild	Atrophy	Full, 1 wk	Two prior episodes
5. 66, F	–	+	None	Atrophy	Parkinsonism: full, 6 wk; dyskinesia: partial, 6 wk	Pill-rolling tremor for 2 yr, lingual-oral dyskinesia for 6 mo
6. 70, M	+	–	Mild	Atrophy	Partial, 7 wk	Two-year history of mild shuffling gait
7. 70, F	–	+	None	Atrophy	Almost complete	

we observed. A subhypnotic dose of ethanol reduces dopamine turnover in the substantia nigra and caudate nucleus in rats [1]. Increased striatal dopamine release is seen in acutely intoxicated rats with blood alcohol levels under 300 mg/dl; decreased striatal release occurs with higher blood alcohol levels [4]. Striatal dopamine release is also reduced during the first few days of ethanol withdrawal [4]. In mice, ethanol withdrawal is associated with diminished responsiveness of striatal dopamine-sensitive adenylate cyclase activity [8].

These biochemical changes, if present in humans, could help to explain why four of our seven patients seemed to develop or greatly augment their signs of parkinsonism during alcohol withdrawal. The reason why other patients developed parkinsonism during prolonged drinking episodes could be related to higher blood ethanol levels, periods of relative withdrawal, or other factors. We propose that our patients had underlying parkinsonian pathology, the effects of which were intensified by chronic alcohol intoxication and withdrawal. All the patients improved with maintained abstinence. The six who had CT scans showed cerebral atrophy, an expected finding in chronic alcholics [2, 3, 5, 6]. Patient 3 also had bilateral basal ganglia calcification. These findings indicate that one should wait a few weeks before starting antiparkinsonian medication in recently abstinent alcoholics with newly diagnosed mild parkinsonism. Alcohol abuse would be expected to be detrimental to parkinsonian patients.

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Propranolol and Chlordiazepoxide Effects on Cardiac Arrhythmias During Alcohol Withdrawal

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The pattern of cardiac arrhythmias and their treatment, by propranolol and chlordiazepoxide, during the first 48 hr of alcohol withdrawal has been studied. Prior to treatment, the incidence of serious and life-threatening arrhythmias was found to be very low and uncorrelated with most biochemical parameters. Propranolol treatment, while efficacious in controlling arrhythmias, was limited due to its association with hallucinations. Chlordiazepoxide was associated with poor early control of arrhythmias. The combination of propranolol and chlordiazepoxide was found to perform best overall with substantial reductions in arrhythmias and the fewest treatment failures.

IT IS KNOWN that cardiac rhythm abnormalities occur with greater frequency in chronic alcoholics during intoxication[1] and withdrawal[1,2] than would be expected in patients of similar age without cardiovascular disease. What is not known, however, is whether the presence of such abnormalities represents an increased risk to intoxicated and withdrawing chronic alcoholics, whether such abnormalities persist during alcohol withdrawal, and whether factors can be identified that predispose chronic alcoholics to arrhythmias, especially during withdrawal. A randomized double blind clinical investigation has been conducted to answer some of the questions raised above and to evaluate the efficacy of two medications, chlordiazepoxide and propranolol, in modifying the pattern of occurrence of cardiac arrhythmias during withdrawal. Chlordiazepoxide has been shown to be a safe and effective therapy of alcohol withdrawal;[3,4] however, its effect upon electrocardiographic events in alcoholic population is not known. Propranolol has been shown to be efficacious in mild to moderate withdrawal, though the response of cardiac arrhythmias in withdrawing alcoholics has not been documented. Because of its beta adrenergic blocking and quinidine-like properties, propranolol may be potentially useful in alcohol withdrawal where some arrhythmias have been hypothesized to arise from increased plasma catecholamine concentrations.[1]

MATERIALS AND METHODS

Seventy-two intoxicated male chronic alcoholics who presented to the emergency department were admitted to the Intensive Care Unit (ICU) of the Addiction Research Foundation Clinical Institute. All patients had a 12-lead electrocardiogram (ECG) and chest x-ray and underwent a physical examination. On the basis of the admission ECG and chest x-ray, patients with evidence of congestive heart failure, cardiac ischemia, and arrhythmias (except tachycardia) were excluded as was any patient requiring emergency medical treatment other than for their intoxication. Following the admission assessment, the monitoring of a lead II ECG was commenced using an ECG telemetry transmitter (HP 78100A). The transmitted signal was received by a local receiver (HP 78100A), the output of which was connected to a standard patient monitor in the ICU monitoring station and to a 12-hr ECG tape recorder (Avionics, 385-A-12-H Holter recorder). The preceding arrangement permitted complete freedom of movement by the patient, 12-hr storage of the ECG by a standard electrocardiographic recording system (therefore, the generation of tapes compatible with a playback system to permit reading of the ECG) and display of the ECG to staff in the event of detection of life threatening cardiac-rhythms. In addition, the telemetry receiver produces an alarm if either the transmitter battery fails or, more importantly, if a lead falls off the patient. Constant monitoring of the ECG permits continuous assessment of the viability of the signal prior to recording thereby overcoming problems of data loss experienced in a previous study[1] using a Holter recorder affixed on the patient.

During the initial ECG monitoring period, blood alcohol concentrations (BAC) were estimated hourly by breath testing until values reached zero. At this point, patients were reassessed by a physician to determine their suitability for continuing in the study. The nature of the investigation was explained and willing patients gave consent to participate further. Those with asthma, diabetes requiring insulin, or a

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Supported in part by the Clinical Institute of the Addiction Research Foundation.

Received for publication November 22, 1979; revised manuscript received March 25, 1980; accepted April 17, 1980.

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0145-6008/80/0404-0012$01.00/0

hemoglobin of less than 10 g/dl were excluded. Patients receiving antiarrhythmia medications, phenytoin, propranolol, or any benzodiazepine were not included.

Sixty patients who consented to participate and who satisfied the admission requirements entered the second phase (medication phase of the investigation). Prior to the first medication, blood samples were drawn for determination of serum electrolytes (K, Na, Cl), blood CO_2, creatine phosphokinase (CPK), hydroxybutyric dehydrogenase (HBD, SMA-12), complete hemogram* and plasma catecholamines.[3] Samples were drawn at 24 and 48 hr for repeat testing of all biochemical parameters except catecholamines. The time of the last drink prior to admission was also ascertained.

Patients were randomly assigned to one of four treatment groups: (1) placebo; (2) propranolol, 40 mg; (3) chloridiazepoxide, 25 mg; and (4) propranolol, 40 mg and chlordiazepoxide, 25 mg. All patients received (double blind) one capsule and one tablet every 6 hr for 48 hr. The placebo group received placebo, identical in appearance to the active drug in both capsule and tablet; the propranolol group received a placebo capsule and propranolol tablet; the chlordiazepoxide group received chlordiazepoxide in capsule and a placebo tablet and the combined therapy group received chlordiazepoxide in capsule and a propranolol tablet. Blood pressure and heart rate were measured 1 hr following each medication.

The ambulatory ECG was recorded for periods 12–24 hr and 36–48 hr following the first medication. All available ECG tapes for the 60 patients who entered the study were subsequently analysed on a beat-to-beat basis with an Avionics 350 C playback apparatus. The individual who read and scored the tapes was blind to the treatment assignment for each patient. The system operates at 60 times real time and if abnormalities are detected, the tape can be rewound slightly and the section containing abnormalities played back at normal speed for further analysis. The nature and incidence of arrhythmias detected for each tape were recorded in a summary.

The ECG summaries were read blindly by an independent observer who assigned a "composite arrhythmia score" to each 12-hr period based upon the type and frequency of cardiac arrhythmias reported. The composite score was the sum of the various component scores which could be assigned to each ECG summary and which are shown in Table 1. The scores shown in Table 1 are derived simply from their rank, which was proposed according to the perceived importance of certain arrhythmias in alcohol withdrawal.[1]

The composite arrhythmia score forms a convenient summary of arrhythmia patterns that are often different among and within patients. It was not intended to provide a rigorous predictor variable of arrhythmia risk to patients.

Sinus tachycardia was defined as a pulse rate of 100 or greater. Persistent sinus tachycardia refers to increased heart rate for more than 75% of the total recording time. Ventricular premature beats were defined as having a QRS morphology different from sinus beats, not preceded by a P wave, being premature and being followed by a compensatory pause. Atrial premature beats were defined as preceded by a P wave, having a normal QRS configuration and being premature. Nodal premature beats were the same as atrial premature beats, except that they were not preceded by P waves.

Analysis of data was by one-way, and two-way analyses of data with unequal numbers per treatment, Newman Keul's tests for differences between means[6] and Student's t tests (2 tailed) where appropriate.

Table 1. Components of Composite Arrhythmia Score

Type	Score
Normal	0
Transient sinus tachycardia	1
ST segment and T-wave changes	2
Persistent sinus tachycardia	3
Nodal rhythm, junctional or atrial premature beats	4
Atrial flutter or paroxysmal persistent atrial tachycardia	5
Unifocal ventricular premature beats (VPBs) (infrequent, <3/12 hr)	6
Multifocal VPBs (infrequent)	7
Unifocal VPBs (frequent, >3/12 hr)	8
Multifocal VPBs (frequent, >3/12 hr)	9
Ventricular tachycardia or life-threatening conduction disturbances	10

RESULTS

During the premedication period, the ECG recordings documented the occurrence and frequency of arrhythmias in the period of declining intoxication and early withdrawal. Table 2 summarizes the type and frequency of cardiac arrhythmias for the entire sample during this initial premedication phase. From the sample, 12 patients could be identified who had high composite arrhythmia scores, mainly due to the presence of ventricular premature contractions. Biochemical results for those 12 with high scores were compared with the remainder who had lower scores to determine if any biochemical test existed that could identify those with more serious arrhythmias and, therefore, predict without the aid of sophisticated long-term ECG monitoring facilities, those for whom specialized treatment might be anticipated. The results of the comparison (Table 3) can be summarized by stating simply that while some values were not within the normal range, there was no statistical difference between the two groups for values on any biochemical test.

Of the 60 patients who entered the medication

*Biochemistry and hematology determinations were by standard clinical laboratory assays and techniques. Details, if required, are available on request.

Table 2. Summary of Incidence of Arrhythmias in Chronic Alcoholics During Late Intoxication Period and Early Abstinence

	Number	Frequency
All arrhythmias		
VPBs	9	17
Atrial or nodal premature beats	9	17
T-wave changes	2	4
Sinus tachycardia	50	93
Normal ECG	4	7
Atrial dysrhythmias		
Sinus tachycardia	24	44
Persistent sinus tachycardia	26	48
Atrial premature beats	5	9
Nodal premature beats	6	11
Ventricular dysrhythmias		
Unifocal VPBs	9	17
3–10 beats/12 hr	5	9
11–40 beats/12 hr	1	2
41–51 beats/12 hr	1	2
>51 beats/12 hr	2	4
Multifocal VPBs	2	4
Ventricular tachycardia	0	0

Frequency refers to the percentage number of incidents relative to the total number of tapes successfully read for this premedication period ($n = 54$).

phase, 47 completed it. The age range of these patients was 27–65 yr (mean ± standard deviation: 44 ± 9.4 yr). There was no statistical difference in patient ages among treatment groups. Two patients refused to stay for the 48-hr medication period and 11 experienced adverse reactions while receiving medications and were removed from the study to receive appropriate alternative therapy. The 11 treatment failures were distributed among the 4 treatment groups; however, the cause for the failure was frequently specific to a particular group. There were 4 failures in the placebo group, all due to complications of alcohol withdrawal; 2 patients developed severe withdrawal necessitating pharmacotherapy and 2 had seizures. There were 4 failures in the propranolol group, all due to the development of severe hallucinosis; most experienced both auditory and visual hallucinations following 3–4 doses of propranolol and all were agitated and disturbed at this point. The 2 failures in the chlordiazepoxide group were a result of complications of cardiovascular origin; 1 patient had a complete heart block and 1 patient was discontinued because of extreme hypertension. The single failure in the combined therapy group was due to the development of hallucinations.

The 47 patients who completed the investigations were examined for the response of the 3 most frequently encountered arrhythmias prior to and following the various drug treatments (see Table 4). For each of the 3 types of arrhythmias, no significant difference (chi square test) could

Table 3. Mean (± SEM) Results of Biochemical Testing Performed Just Prior to Medication for the Group With High Composite Arrhythmia Scores and Group With the Low Scores

Biochemistry	High Arrhythmia Scores	Low Arrhythmia Scores	Normal Range*
Na	140 ± 0.7	141 ± 0.6	137–147 meq/liter
K	4.1 ± 0.07	4.1 ± 0.06	3.6–5.5 meq/liter
Cl	98 ± 1.4	99 ± 0.7	98–109 meq/liter
CO_2	24 ± 0.8	25 ± 0.4	24–34 meq/liter
CPK	76§ ± 17	102§ ± 16	5–45 IU/liter
SGOT	36§ ± 14	36§ ± 5	8–30 IU/liter
BUN	9 ± 1	10 ± 0.6	8–20 mg/dl
HBD	183 ± 21	192 ± 8	120–210 IU/liter
Bilirubin			
Total	0.68 ± 0.10	0.75 ± 0.06	<1.0 mg/dl
Direct	0.20 ± 0.04	0.23 ± 0.02	<0.5 mg/dl
Alkaline phosphatase	36§ ± 3	37§ ± 2	56–244 IU/liter
Plasma epinephrine	358§ ± 88	155§ ± 55	94–98 pg/ml†
Plasma norepinephrine	828* ± 117	547§ ± 132	278–286 pg/m††
Alcohol elimination rate	212 ± 17	217 ± 11	100–220 mg/hr‡
Initial blood alcohol concentration	2083§ ± 306	2520§ ± 162	0 mg/liter

*Values except where noted are from the Laboratory Manual of the Clinical Institute Addiction Research Foundation, Clinical Laboratory Biochemistry Department. Values are from laboratory tests for annual examinations of healthy ARF employees.

†From Sole, M.J. (personal communication).

‡From Shumate, et al.[7]

§Outside normal range.

Table 4. Response as a Function of Treatment Group for the Three Most Commonly Encountered Arrhythmias and Composite Arrhythmia Score

Arrhythmia	Study Period	Placebo	Propranolol	CDZ	Comb.
Ventricular	Premed	3	1	3	2
(VPBs)	12–24	1	0	3	1
	36–48	1	0	1	1
Atrial or	Premed	2	2	2	2
nodal	12–24	2	2	5	0
	36–48	2	2	2	2
Sinus	Premed	10	10	12	12
tachycardia	12–24	7	2	10	6
	36–48	4	2	11	3
Arrhythmia	Premed	5.27	3.09	3.61	4.46
score	12.24	2.64	1.18	5.08	1.50
	36–48	2.36	0.91	2.54	2.08

Premed refers to the initial taping following admission (see text), 12–24 and 36–48 hr are the two 12-hr recording periods following medication. Entries in the table (except Arrhythmia score) are the number of patients with the associated arrhythmia.

be found in the incidence of arrhythmias for any treatment at any time following medication compared with placebo except that, at 36–48 hr, the incidence of sinus tachycardia for the chlordiazepoxide group was significantly greater than placebo [χ^2 (corrected) = 4.02, $p < 0.05$]. As noted in Table 4, however, there was a tendency for chlordiazepoxide to be associated with a higher incidence of ventricular and atrial arrythmias 12–24 hr following medication. In addition, there was very little tendency of chlordiazepoxide to reduce sinus tachycardia even when compared to placebo.

Table 4 also shows the mean composite arrhythmia scores for each treatment group for the 3 ECG recording sessions. As shown, there is a marked tendency for scores to decrease throughout the 48-hr withdrawal period. While the effect as a function of recording session was significant ($F_{2,86} = 11.86$, $p < 0.001$), chlordiazepoxide was associated with the only increase in score at 12–24 hr relative to the premedication session. Chlordiazepoxide group scores were significantly greater than those for all other treatment groups ($p < 0.05$, by Newman-Keuls[5] with treatments containing propranolol and $p < 0.055$ for comparison with placebo). By the 36–48-hr period, however, all therapies performed equally well. The response shown in the bottom of Table 4 is consistent with the other information shown in the table indicating that the rather convenient method of constructing a composite

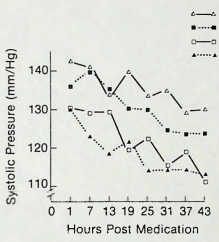

Fig. 1 Systolic blood pressures for the four treatment groups as a function of time following the first medication.

arrhythmia scoring system does not greatly distort the evaluation of the relative efficacy of the drug treatments.

Blood pressure and heart rates were elevated for all groups at the time of the first medication; however, values declined towards normal levels over the medication period. There was a highly significant difference between groups for systolic blood pressure ($F_{3,41} = 4.83$, $p < 0.01$), diastolic blood pressure ($F_{3,41} = 9.48$, $p < 0.0001$) and heart rate ($F_{3,41} = 4.85$, $p < 0.01$) and, as shown for blood pressure in Fig. 1, the difference among groups could be attributed to the difference between treatments containing propranolol and the other two treatments.

Following the first biochemical testing it was found that CPK, SGOT, HBD, and alkaline phosphatase tended to be elevated above normal while the BUN tended to be subnormal. For all treatment groups, CPK, SGOT, HBD, and alkaline phosphatase decreased towards normal levels over the 48-hr medication period, and there was no difference between treatment groups.

DISCUSSION

The sample of chronic alcoholics examined in this investigation was representative of intoxicated chronic alcoholics presenting to our hospital. The incidence and severity of cardiac arrhythmias, therefore, represent those observed in the terminal phases of ethanol intoxication and early withdrawal. While the incidence of some types of arrhythmias is clearly much higher than would be expected for the nonalco-

holic population without cardiovascular disease, there was no potentially life-threatening cardiovascular incident that could be attributed to acute intoxication and withdrawal. As a result of this finding it would be difficult to argue for the profitability of instituting, into hospital care, the intensive monitoring of the electrocardiographic events of presenting chronic alcoholics unless a diagnosed heart condition so indicated. Cardiac arrhythmias have been shown to be more prevalent in late intoxication and withdrawal and becoming less so by 48 hr in withdrawal. It was found that no simple biochemical test was correlated with the frequency or incidence of more severe arrhythmias during the late stage of alcohol intoxication and early stage of withdrawal. Thus, it may be true that while the occurrence of cardiac arrhythmias may be related to the presence of ethanol and elevated epinephrine and norepinephrine blood levels as suggested by Abbasakoor et al.,[1] the differences in severity and frequency of electrocardiographic abnormalities cannot be accounted for by differences in ethanol, epinephrine, and norepinephrine levels. A likely explanation may be that the observed differences among alcoholics is a result of variable cardiac sensitivity to ethanol and/or plasma catecholamine concentrations.

Both treatments containing propranolol were associated with the greatest reductions in arrhythmias, particularly early in withdrawal. In addition, both treatments resulted in the greatest decrease of elevated heart rates and blood pressures corroborating the observations of Sellers et al.[3] Of particular interest and importance in this investigation, however, is the very high incidence of hallucinations in the withdrawing alcoholic treated with propranolol alone (27%). Central nervous system effects of propranolol, in particular, hallucinations, normally occur only occasionally (1%)[8,9] and are usually associated with long-term propranolol therapy[10–12] involving high daily doses of the compound. The considerable sensitivity of some of the alcoholics in this investigation raises the question of interaction of the severe withdrawal state and propranolol. French et al.[13] demonstrated that β adrenergic receptors become subsensitive during prolonged ethanol ingestion and supersensitivity of β receptors is known to occur approximately 72 hr in withdrawal,[14] possibly due to either an increase in number or affinity of β receptors.[15] Early in withdrawal, a state exists whereby adrenergic receptors are subsensitive,[16] a condition that may give rise to the clinical manifestation of some of the signs and symptoms apparent in early withdrawal.[17] It is known, for example, that early in withdrawal adrenergic antagonists increase seizure scores caused by early withdrawal.[18] It may be likely, therefore, that the presence of a β-adrenergic blocking agent early in alcohol withdrawal may be sufficient to exacerbate some early signs and symptoms of withdrawal, especially hallucinations.

Chlordiazepoxide was found to be less effective in reducing arrhythmias within 12–24 hr compared with either placebo or treatments containing propranolol. During that period chlordiaxepoxide was associated with an increase in atrial arrhythmias and a failure to substantially reduce the incidence of ventricular arrhythmias and sinus tachycardia. In addition, the two treatment failures that were the result of cardiovascular complications occurred in patients who were receiving chlordiazepoxide. By 36–48 hr, chlordiazepoxide performs as well as other treatments except that the incidence of sinus tachycardia remains frequent. There is a paucity of reports in the literature regarding the role of chlordiazepoxide in cardiac arrhythmias. What reports there are do not involve alcoholics and generally comment on antiarrhythmic properties of the drug.[19] Only one report exists[20] to imply that the benzodiazepine, diazepam, may be implicated in arrhythmias. While the association of chlordiazepoxide with atrial and ventricular arrhythmias is very difficult to account for, there is some evidence to support the idea that chlordiazepoxide may produce increases in heart rate, or more correctly, antagonize a reduction of heart rate that would occur normally (cf. placebo group). Rao et al.[21] reported slight increases in heart rate following diazepam. In addition, it can be noted that benzodiazepines may have cholinergic blocking properties, though such properties have hitherto not been considered of any clinical importance.[22]

The combination of propranolol and chlordiazepoxide appears to go a long way towards overcoming some of the treatment limitations found in using the drugs separately. There was only one treatment failure in this group and that was due to hallucinations, very likely a result of the presence of propranolol. The combination

treatment was associated with significant reductions in arrhythmias, systolic and diastolic blood pressures, and heart rate. There were no cardiovascular incidents such as those associated with chlordiazepoxide alone. The mechanism by which combination therapy is able to perform better than each drug alone is difficult to speculate since the interaction of both compounds is likely to be complex. Sellers et al.[3] demonstrated that while a combination of propranolol and chlordiazepoxide was more efficacious than placebo in ameliorating the signs or symptoms of withdrawal, propranolol alone performed better. It was noted, however, that the withdrawal displayed by alcoholics in that study was mild to moderate and that no incidents of hallucinations were produced. In the present study, however, more severe cases of acute alcohol intoxication and withdrawal were included. With the demonstrated risk of using propranolol in withdrawing alcoholics, some of whom may be in severe withdrawal, and with the limited effectiveness of chlordiazepoxide in rapidly controlling cardiovascular parameters, the combination of propranolol and chlordiazepoxide may provide, in appropriate patients, a more effective therapy in withdrawal.

ACKNOWLEDGMENT

The authors ackowledge the considerable work of the staff of the ICU in assuring the successful conduct of this investigation, and, in particular Roseanne Buski who brought all of the vast amount of data together for analysis. We thank Angelo Tesoro for performing the catecholamine assays and Dr. E. Whiteside and Dr. C. Robertson for their participation and D. Durand for his help in setting up and maintaining the telemetry recording system. We thank Karen Kaplan for her expertise in implementing the computer analyses of the data.

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ALCOHOL AND DRUG ABUSE AT BMHC

Marc Galanter, M.D.

I. Teaching:

The Division serves as the principal vehicle for teaching in the area of alcoholism and drug abuse at the College of Medicine. These disease areas are most pressing, since they affect 10% of the population, and constitute 53% of the total social cost of psychiatric and mental disorders.

Training begins at the undergraduate level with a module in the first year of the Human Behavior sequence. During the psychiatry clinical clerkship, a weekly seminar and patient interview sequence is conducted at Bronx Municipal Hospital Center for student clerks. This module emphasizes the acuisition in skills of clinical interviewing of the substance abuser, diagnostic issues, and the selection of appropriate treatment modalities relative to the variety of abuse syndromes. In addition, electives on both clinical and research issues are available to first and fourth year students.

On the post-graduate level, psychiatric residents are offered a structured sequence of experiences. Lecture series are given in the first, second, and third post-graduate years, emphasizing issues relevant to the trainees' work at that level. Thus, in the PGY I year, this is done in relation to the emergency room experience, emphasizing issues of diagnosis, evaluation of occult addiction, and management of acute intoxication and withdrawal. Seminar series in subsequent years deal more with issues of long-term treatment.

Central to the residents' training is the provision of a sequence of individual supervision sessions for each of the psychiatric residents in both the first and third PGY years. In the first year, six sessions of individual supervision are centered around the emergency service; in the third year, individual supervision sessions focus on patients presenting in the long-term treatment services of the ambulatory clinic and on the liaison service. In the fourth year elective experiences are offered, generally involving the combination of clinical administration and supervised research activities.

The Division has also offered fellowships with an emphasis on clinical administration and research. Publications emerging from these fellowships have focussed primarily on the evaluation and treatment of addictive illness in the general hospital setting.

Training for social work students and interns is also provided, with the placement of two to four interns from Hunter College and Yeshiva University social schools for each academic year.

The Division has also been the site of a federally-funded Career Teacher Program in Alcohol and Drug Abuse. This activity has served to provide a national focus for organizing and disseminating teaching information in the substance abuse area. This past year, this has culminated in the American Medical Society on Alcoholism appointing the Division's Director as Chairman of the Committee, which will undertake the establishing of criteria for training in the credentialling of physicians in the alcoholism field.

II. Clinical Activities:

The Division is centered at the Comprehensive Alcoholism Treatment Center of the Bronx Municipal Hospital Center, where some 500 patients each year are treated on a twenty-bed acute inpatient unit. After their need for detoxification is first addressed, patients are integrated into the long-term treatment program. The C.A.T.C. has 500 patients in active treatment and provides 15,000 patient visits each year, group and individual therapy being primary modalities. The program also maintains an active vocational and recreational therapy program, in addition to the medical care described above. A variety of special rehabilitation modalities are maintained, including women's groups to meet specialized needs during the course of treatment, and a self-help program geared to developing communal assistance for social rehabilitation and abstinence.

The Division provides liaison activities, dealing directly with the physicians responsible for general care of drug abusing and alcoholic patients at Bronx Municipal Hospital Center. We have found that 24% of these patients are diagnosable as alcoholic, and large numbers as drug abusers. Most have come to the hospital for treatment of the consequences of this primary disease, and it is essential to initiate rehabilitation during the phase of general medical care. Supervision of psychiatric residents who serve as liaison officers is a primary aspect of this.

The following statistics for the 1984-5 year are relevant.

1. Inpatient department

Beds: 20.

Mean length of stay: 10.

Number of admissions: 620.

Sex ratio,male/female: 84%/16%.

Age range

18-20: 1%

21-34: 38%

35-49: 25%

50-64: 17%

65 and over: 19%

2. Outpatient department

Total number of patients treated: 489.

Patients carried over from prior year: 402.

New patients for year: 262.

Sex ratio, male/female: 73%/27%.

Ethnic groups

Caucasian 20%.

Black 42%.

Hispanic 38%.

Treatment modality

Individual therapy: 10% of patients, 1083 visits.

Group therapy: 95% of patients, 10,289 visits.

Family counseling: 10% of patients, 756 visits.

III Research Activities

A variety of research activities are ongoing, in areas directly related to alcoholism and drug abuse and in allied fields. Project areas are listed

below along with the parties principally responsible for their implementation.

1. Self-help treatment for alcoholism (Marc Galanter, M. D.). This project funded by the Commonwealth Fund is directed at investigating an innovative approach to alcoholism rehabilitation. The treatment relies on self-help techniques and a sizeable input of patient direction into clinical activities, allowing for diminished staffing and lower cost.

3. Neuroendocrine studies on alcoholics and diabetic patients (Jacob Jacoby, M.D., Ph.D.). A number of projects focus on neuroendocrine abnormalities in medical illnesses such as diabetes, and in syndromes secondary to chronic alcohol abuse. Coordination here is undertaken with the Departmental preclinical laboratories and with the Department of Medicine.

3. Ethnic characteristics of alcoholic and borderline patients (Ricardo Castaneda, M.D.). Evaluation of the incidence of different pathologic syndromes, including the borderline personality disorder, are examined in relation to the different ethnic groups treated in the program. Consideration of relative treatment success in the alcoholism program for different ethnic groups is also being undertaken.

4. Verbal production in psychoanalytic sessions (Georges Moroz, M. D.). The transcripts of psychoanalytic patients are analyzed for repeated themes and structures. This allows for an understanding of cognitive and affective issues which emerge in the course of the therapeutic experience and also assist in understanding the nature of the therapeutic process.

5. An evaluation of the "Recovery" self-help program (Marc Galanter, M. D.). With support from the Recovery self-help organization, an evaluation is underway to ascertain the way in which this self-help program aids chronic psychiatric patients. The need for professional help and pharmacotherapy are examined in relation to parameters of group involvement.

6. Psychology of religious cults (Marc Galanter, M. D.). The psychological

outcome of long-term membership and marriage in the Unification Church is examined. Correlates of improved emotional well-being and enhanced conformity to group demands are examined in relation to scores on standardized psychological instruments.

7. Recent Developments in Alcoholism (Marc Galanter, M.D., Editor). This book series, with volumes released annually, overviews recent research findings. The current volume addresses combined alcohol and drug problems, typologies of alcoholics, the withdrawal syndrome, and renal and electrolyte problems.

Effects of drinking

When I was young, we had a neighbour, who was in business and earning a fair amount. He had a nice family: a wife who was devoted to him and 4 daughters. Every night, he would come home drunk & shouting and attack his wife and children. Many a night, my mother had to bring the wife and children to our house and keep them there, till the man was asleep. When sober, the man was a nice husband and father. Gradually, he made a mess of the business and left the locality.

When a medical student, I had a friend who used to indulge in alcohol. Under its influence, he used to cry saying that he was wasting his father's money and that he was a disgrace to his family.

I have a friend who is now a doctor in USA. One day he drove, under the influence of alcohol, straight into a wall. The car became a total loss but he escaped.

There was a ~~neurologist~~ physician, who travelled by motor cycle. He saw two lights coming towards him. Thinking that they were two motor cycles on either side of the road, he drove in the middle. It was a large military truck and the doctor was admitted for neurosurgery.

*Alcohol produces changes in the functions and behaviour of the person. The person becomes unsteady in gait. This was one of the signs that used to be relied upon by doctors in the casualty. The person is asked to walk in a straight line. Under the influence of alcohol, he is unable to do so. There is loss of co-ordination. The speech is slurred and often incoherent. Difficulty is experienced in carrying out even simple tasks. Vision and hearing are impaired.

The changes in behaviour are even more evident.

* Social drinking is a common experience. The person mainly ~~becomes~~ talkative and begins boasting.

The effects of alcohol abuse lead to impairment of work and intellect.
person becomes accident prone
absenteeism

hand tremor
impairment of liver function — cirrhosis of liver
scars on body due to accidents and fights
Change in many laboratory findings — enzymes, MCV
withdrawal symptoms
pancreatitis
gastritis

The effect of alcoholism has been devastating. All countries are worried about the problem of drinking. (In March 1985, Mikhail S. Gorbachev mounted an attack on drinking.) According a the 1980 report in USSR, 60–80% of all thefts and robberies, 75% of murders and rapes, 45% of divorces and broken marriages were due to alcoholism. Productivity dropped by 15–20% after heavy drinking during holidays and pay-days. An All-Union Voluntary Society for the Struggle for Sobriety has been formed. The short-term results have been encouraging. The long-term effects are yet to be seen.

USA is equally worried. In 1971, President Richard M. Nixon signed a bill creating the National Institute on Alcohol Abuse and Alcoholism. The institute was mandated to manage, treat and prevent the nation's growing problem of alcohol abuse.

Powerful public voluntary organisations have come up like MADD — Mothers against drunk driving and SADD — Students against driving drunk.

What makes a person abuse alcohol?

Stress? Does alcohol reduce stress? Drinker seeks relaxation.

Alcohol reduces inhibitions. Alcohol is a depressant of the central nervous system. The inhibiting centres in the cerebral cortex get depressed at a faster rate.

But this theory has not been supported by animal and human research. Alcohol dampens perception.

Alcohol in low doses has a biphasic effect. If a person takes 1 ml/kg of alcohol or say 60 ml on an empty stomach, blood alcohol concentration rises relatively quickly in the first 60–90 min. During this phase, the drinker feels 'high' or 'euphoric', is most impaired in motor and cognitive tasks and appears to be more aroused than he was prior to the first drink. In the descending phase of the cycle (after the first 90 min) and lasting for some 5–6 hours after consumption, alcohol has a more depressant effect, with unpleasant feelings. The drinker passes through the same blood alcohol levels in the descending phase also.

Alcohol may be effective in the response to stress not because it reduces tension but because it stimulates a sense of personal power. The person perceives a lack of control over important events in life. The person under stress experiences the feeling that he is a helpless victim of external forces. During the phase of heightened arousal, as the blood alcohol curve ascends, many drinkers sense an enhanced degree of personal power or control. The dominant theme in the fantasies of drinking males is one of enhanced social power.

Stressed individual may be viewed as one who experiences himself as prey to complex and confusing life events or as feeling caught in a work or family situation

that seems beyond his control. Drinking then may be seen not simply as an attempt to induce relaxation or reduce psychic tension but to regain sense of personal power and control.

Effects of drinking

The effects of alcoholism can be extremely bad. These effects can be on the individual, the family, the industry, the society and the country. Alcoholism is a universal problem, whether it be a capitalistic country, a socialist society or a communist nation. It was because of the devastating effects affecting millions and millions of people that the leaders of these countries have addressed themselves to the solution of these problems.

Mikhail S. Gorbachev mounted a serious attack on excessive drinking, immediately after he took charge of Soviet Russia. Legislation was passed in 1985 to curb drinking. USA has been equally active. In 1971, President Richard M Nixon signed a bill creating the National Institute on Alcohol Abuse and Alcoholism. Powerful voluntary organisations have come up like MADD — Mothers against drunk driving and SADD — Students against driving drunk. In our country, ~~the Indian National Congress~~ in the early part of Independence, prohibition was introduced but later withdrawn.

It is easy to give the effects of alcohol on the individual; ~~unsteadiness~~ These are classical: unsteady gait, slurred speech, difficulty in carrying out even simple task; impairment of motor functions and sensations including vision and hearing. Hand tremor is seen

Behavioural changes are marked: talkative, boastful, aggressive, quarrelsome. The responses vary with the from individual to individual and situation.

The person becomes accident prone. Absenteeism is very common. Withdrawal symptoms occur when alcohol is withheld.

The liver is one of the organs badly affected, with alcoholic hepatitis, fatty degeneration and cirrhosis of liver. Cirrhosis of liver is directly related to alcohol consumption. Another organ badly affected is the pancreas — acute and chronic pancreatitis may be seen. Gastritis occurs very commonly. * Alcoholism affects other organs also, including the central nervous system.

Laboratory findings are indicative of damages to liver and other organs.

The effect on the family are disastrous. Many families are broken up. In Russia and some other countries, about 45–50% of divorces and broken marriages are due to alcoholism. In our own country, the percentages of such broken & marriages due to alcoholism are significant but often, the poor wife suffers. We see the effects in battered wife and abused children. Sometimes, there is an arrangement by which the wife goes in and even encourages the husband to drink, to buy peace.

On the industry also, the effects are pronounced. There is a loss of productivity. Absenteeism is common. Accidents occur quite often. Both these affect the working of the factory or industry. Decision-making becomes erratic. Production schedules are upset. Damage could be caused to machinery and property.

Society is affected. Drunken brawls occur. Fights are precipitated. Robberies and thefts become common. Murders and rapes occur much more frequently under the influence of alcohol. Accidents due to drunken driving take heavy toll.

All these — the effects on the individual, the family, the industry, and the society total up to affect the country. Some body has to pay for the i'-e's.t. and that is the country — treatment

illness caused by alcoholism, accidents leading to loss of life, limbs and compensation, broken families affecting the education of the children, rehabilitation, loss of production and productivity.

We should consider another aspect - What are effects for which the person takes to alcohol abuse. Often, we think that it is taken, at least in the initial stages to reduce stress - tension relieving; relaxation. But there is another view supported by animal and human experiments. Alcohol is taken for the sense of personal power. Alcohol in low doses has a biphasic effect. If a person takes 1 ml/kg of alcohol (say, 60 ml) on an empty stomach, blood alcohol concentration rises relatively quickly in the first phase (60-90 min). During this phase, the drinker feels 'high' or 'euphoric'; is most impaired in motor and cognitive tasks and appears to be more aroused than he was prior to the first drink.

A L C O H O L I S M

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Drinking has been in existence from time immemorial but it was not much of a problem. Fermented juices were taken occasionally; the alcohol content was low. Drunkenness was infrequent and socially looked down upon in our country.

Today, alcoholism is a major problem in the country. There are at least 3 million alcohlics in India. It is a problem for the individual (causing many diseases like cirrhosis of liver and being a risk factor for other major diseases such as heart disease), the family (broken or unhappy families) and the society (crimes, fights, accidents and loss of productivity). It was because of the realization that alcoholism is a major problem that prohibition became a major policy of our Independence movement.

The problem of alcohol abuse is both urban and rural. The Indian Council of Medical Research study covering a number of urban centres including Bangalore found that 20% of urban consumers of alcohol are totally dependent on it.

Problem drinking is very high in industries, varying between 5% and 15%. With the large number of large scale industries in the public and private sectors in Bangalore, the problem is one of high concern. The economic costs of alcoholism are very substantial. In the developed countries, the Employees Assistance Programme for Alcoholism has been introduced successfully. It augurs well that the Managements and trade unions in Bangalore are now seized of the problem.

contd.2

Who is an alcoholic?

A person who has become physically, physiologically or psychologically dependent on alcohol is an alcoholic. There is a compelling urge or craving for alcohol. The body adapts itself to alcohol. There is tolerance. There is dependence. Withdrawal produces symptoms which can be severe. Alcoholism is an illness, manifested by behavioural and clinical disorders. It is generally progressive but can be arrested.

Factors leading to alcohol abuse

Any type of person can become an alcoholic. Some people are more prone to become problem drinkers - alcoholic personality ? childhood delinquency ? familial ? genetic ? Certain factors can precipitate alcohol abuse - stress, loss of job, death of spouse, sudden improvement in income. A pre-existing psychiastric problem like depression may lead to alcohol abuse.

Alcohol causes aggressive, silly behaviour. There is unsteadiness of gait and slurred speech. Difficulty is experienced in carrying out even simple tasks. Vision is impaired as also hearing. Alcohol can cause acute drowsiness, deep sleep and coma.

Early identification of problem drinkers

It is necessary to identify the "hidden alcoholic". Management is hopefully much easier at that stage than after the person has become a chronic alcoholic. There are many indicators available. No single indicator is absolute. But a composite group of indicators can be useful. Among them are psycholigical, clinical and laboratory manifestations.

contd.3

1. Psychological

(i) The first one is a history, including drinking habits - frequency and quantity (how often ? how much ?) Usually the person gives a reliable answer to the frequency but the quantity is often unreliable. Heavy drinking (about 60 g/ day of ethanol) indicates problem drinking.

(ii) Increased tolerance to alcohol. The person needs more and more alcohol to get the same effect.

(iii) Drinking quickly, gulping the first drinks, skipping meals while drinking.

(iv) Concern or worry about drinking but unable to stop or reduce drinking. The family also gets worried about the drinking.

(v) Intellectual impairment. To a keen observer who knows the person well, this may be the first indicator.

(vi) Work impairment is an indicator.

(vii) Accident prone at the workplace and elsewhere.

(viii) Absence from work.

(ix) Change in friends, keeping company with heavy drinkers.

2. Clinical

(i) Hand tremor may be one of the earliest symptoms.

(ii) Alcoholic fator by day.

(iii) Nausea and vomiting in the morning.

(iv) Signs and symptoms of acute or chronic pancreatitis

(v) Hepatomegaly and evidence of impairment of liver functions

(vi) Scars on the body (due to accidents and fights).

3. Laboratory

There are many markers which can point to alcoholism.

(i) Gamma glutamyl transpeptidase (GGT)

(ii) Serum glutamic oxaloacetic transaminase (SGOT)

(iii) " alkaline phosphatase

(iv) " glutamic pyruic transaminase (SGPT)

- contd.4

(v) Mean corpuscular volume (MCV)

(vi) Serum high density lipoprotein cholesterol (HDL-C)

(vii) Abnormal transferrin

(viii) Random blood alcohol level

Chronic alcoholism

Chronic alcoholism is indicated by

1.1. high/frequent consumption of alcohol
2. withdrawal symptoms; black-outs
3. physical violence

2.1. battered children/wife
2. psychosomatic complaints, depression or anxiety in spouse
3. divorce/separation

3.1. impaired work performance
2. loss of sense of responsibility
3. absenteeism
4. prone to accidents

4.1. liver disease - cirrhosis of liver
2. malnutrition
3. pancreatitis
4. gastritis; peptic ulcer
5. congestive cardiac failure
6. neurological disorders

5.1. anxiety
2. depression
3. suicide attempt

6.1. loss of friends
2. change of friends (drinking)
3. loss of interest in recreation

7.1. debts
2. poor living conditions

Treatment

Alcoholism can be treated. The management is multidimensional and multi-step. The objectives of treatment are

1. management of the acute episode of intoxication and detoxication,
2. breaking the dependence on alcohol,

contd.5

3. treating the alcohol withdrawal reactions,
4. remedying the chronic health problems due to alcohol,
5. changing the life-style, attitude and personality characteristics, and
6. providing support to the individual and the family to cope with the situation.

Motivation

One of the important requirements for the success of treatment is motivation to seek help and willingness to undergo treatment. Many persons are desirous of breaking the habit but the motivation may not be strong enough. We have to build up motivation.

Team effort

Treatment should involve the psychiatrist, clinical psychologist, social worker and (in industries) the personnel welfare officer, as also an understanding and supportive family and in industries, management.

Counselling

Good counselling can be effective. It must emphasise responsibility, personal health, work and interpersonal relationships.

Educational material:

Good educational material, appropriate to the level of the person must be made available. Good literature must be produced as also audiovisuals.

Relaxation methods can help to allay tension and anxiety and contribute to better treatment.

Family therapy: Drinking behaviour might serve as an adaptive function for the individual or the family. Alcoholism could be a symptom of larger family problems. Family members can and should provide support to wean away the problem drinker.

contd.6

There is also need to help the family. Counselling of the family members is needed.

Group therapy

Interaction with others who are also dependent on alcohol can help. Members of the group share and discuss their problems.

One successful group therapy was through Alcoholic Anonymous, founded in 1935 by Bill W, who was an alcoholic (real name: William Giffith Wilson). It arose from a long talk for hours with another alcoholic: Dr.Robert Smith. By talking together and sharing their weaknesses in giving up drinking, the two men found that the urge to drink passed off. Alcoholics Anonymous has grown to at least a million members throughout the world.

Combining family and groups can be helpful. Multiple couples group therapy is to be tried. The difficulty is in finding couples and groups comparable in age, education, socio-economic status and severity of alcoholism.

Work therapy is worth trying as part of the total treatment.
Detoxification: During the acute stage, there is need for hospitalization. If there are complications, they must be treated. Management of the acute alcohol intoxication and the concomitant withdrawal syndrome will depend on

(i) patient's condition
(ii) nutritional status
(iii) severity of alcohol dependence, and
(iv) overall medical evaluation.

Treatment of complications

The most important and direct morbidity caused by alcohol is cirrhosis of the liver. Mortality due to cirrhosis in different countries is closely related to the amount of alcohol consumed, irrespective of the type of beverage. The risk factor for development of cirrhosis is given by the product of the average daily consumption of alcohol multiplied

..contd.7

by the period of consumption at that level. Fatty degeneration of liver and alcoholic hepatitis may be seen.

Pancreatitis (acute and chronic) can be caused by alcohol. Gastritis is another clinical manifestation. Anaemia and clotting disorders can occur. Neurological diseases affecting central and peripheral nervous system may be seen. The heart may be affected by cardiomyopathy. Vitamin deficiencies may occur.

Pharmacotherapy of chronic alcoholism.

Treatment with drugs does not have an important place in the management of chronic alcoholism. In the initial stages, there may be a place for anxilytic drugs and antidepressants. So also drugs are useful to control withdrawal symptoms.

One drug which is useful is disulfiram (antabuse). It causes an aversion reaction when alcohol is taken. Disulfiram blocks the oxidation of alcohol at the acetaldelyde stage, raising its concentration in the blood by 5-10 times, and causes reaction. 125 - 250 mg of the drug is administered per day or once in 3 days (the effect often lasts for a week). Taking as little as 7 ml of alcohol can bring about the reaction. Care should be taken as the side-effects and contra-indications are many and the reaction can be severe.

Disulfiram action was discovered accidentally. Two Danish Scientists took the drug themselves to assess its safety as a vermifuge. While on disulfiram, they went to a cocktail party with disastrous results. They inferred that the drug might be useful in preventing alcohol consumption.

Other drugs like calcium carbamide and metronidazole and other aversion techniques are being tried.

Research

Research is needed if we are to solve our problems in the future. "If you do not think of the future, you cannot have one". There are many areas of research.

contd.8

1. Survey of alcohol use
   - licit and illicit
   - youth; different socio-economic groups
   - employed persons
   - industries
   - extent of alcoholism in the population
2. Control measures
   - legal
   - educational
   - social
   - limiting use of alcohol
   - limiting problems arousing out of use of alcohol.
   - cause of spread of alcoholism.
3. Preventing alcohol abuse
   - factors governing use of alcohol.
4. Identifying the problem drinker
   - development of markers of alcohol consumption
5. Adverse effects of alcohol use
   - individual
   - family
   - society
   - at the work place
6. Mechanism of tolerance to alcohol
7. Drinking and accidents
   - on the roads
   - in the factory
8. Alcohol and diseases
   - liver
   - cardiac
   - neurological
   - psychiatric
9. Treatment
   - psychological
   - pharmacological
   - individual
   - family
   - group
   - low cost interventions
10. Follow-up
    - Evaluation of recovery
    - Prevention of relapses
    - Employee assistance programmes

MH-2A-23

DSM III criteria for alcohol abuse and alcohol dependence

Diagnostic criteria for alcohol abuse	Diagnostic criteria for alcohol dependence
A. Pattern of pathological alcohol use:	A. Pattern of pathological alcohol use:
(i) need for daily use of alcohol for adequate functioning	(i)
(ii) inability to cut down or stop drinking	(ii)
(iii) repeated efforts to control or reduce excess drinking by periods of temporary abstinence	(iii)
(iv) restricting drinking to certain times of the day, binges	(iv)
(v) blackouts — amnesic periods for events occurring while intoxicated	(v)
(vi) continuation of drinking despite a serious physical disorder that the individual knows is exacerbated by alcohol use	(vi)
(vii) drinking of non-beverage alcohol	(vii)
B. Impairment in social or occupational functioning due to alcohol use:	B.
(i) violence while intoxicated	(i)
(ii) absence from work.	(ii)
(iii) loss of job	(iii)
(iv) legal difficulties (eg, arrest for intoxicated behaviour, traffic accidents while intoxicated	(iv)
(v) arguments or difficulties with family or friends because of excessive alcohol use.	(v)
	Either tolerance or withdrawal.
C. Duration of disturbance of at least 1 month	C. Tolerance: need for markedly increased amounts of alcohol to achieve the desired effect or markedly diminished effect with regular use of the same amount.
	D. Withdrawal: development of alcohol withdrawal (eg. morning "shakes" and malaise relieved by drinking) after cessation of or reduction in drinking.

Alcoholism

Research

Issues: Does the unitary disease concept permit reliable diagnosis for the purposes of early detection and treatment planning?

Is treatment effective?

Can the costs of treatment be contained?

Are there distinct subgroups of alcoholic persons?

Can treatment efficacy be improved by matching individual patients to specific types of treatment?

Should abstinence be the singular goal of treatment?

Can some problem drinkers engage in nonabusive drinking?

II- Behavioral, Psychological and Attitudinal.

1. Drinking despite strong medical contra-indication known to patient
2. Drinking despite strong, identified, social contra-indication
   (a) job loss for intoxication
   (b) marriage disruption because of drinking
   (c) arrest for intoxication
   (d) driving while intoxicated.
3. Patients' subjective complaint of loss of control of alcohol consumption.

Persons at high risk of alcoholism

(a) a family history of alcoholism, including parents, siblings, grandparents, uncles and aunts

(b) coming from a broken home or home with much parental discord, particularly where if the father was absent or rejecting but not punitive.

(c) being the last child of a large family or in the last half of the sibship in a large family.

(d) heavy drinking is often associated with heavy smoking

Criteria for diagnosis of alcoholism.

To identify individuals at multiple levels of dependency.
Pathological dependency on ethanol.

I. Physiological dependency

A1. Physiological dependence as manifested by evidence of a withdrawal syndrome when the intake of alcohol is interrupted or decreased without substitution of other sedation.

(a) gross tremor

(b) ~~hallucinations~~ hallucinosis

(c) delerium tremens, usually starting between the first and third day after withdrawal and minimally includes tremors, disorientation and hallucinations.

2. Evidence of tolerance to the effects of alcohol.

(a) a blood alcohol level of more than 150 mg without gross evidence of intoxication

(b) consumption of one-fifth of a gallon of whiskey or an equivalent amount of wine or beer daily for more than one day, by a ~~135~~ 180 lb individual.

3. Alcoholic "black-out" periods

B. Clinical major alcohol-associated illnesses.

(i) Fatty degeneration (in the absence of other known cause).
(ii) Alcoholic hepatitis
(iii) Laennec's ~~=~~ cirrhosis
(iv) Pancreatitis (in the absence of cholelithiasis)
(v) Chronic gastritis
(vi) Haematological disorders — Anaemia; clotting disorders
(vii) Wernicke — Korsakoff syndrome
(viii) Alcoholic cerebellar degeneration
(ix) Peripheral neuropathy
(x) Toxic amblyopia
(xi) Alcoholic cardiomyopathy
(xii) Beriberi
(xiii) Pellagra.

Disulfiram implants

A long acting disulfiram implant may possibly last as long as six months.

Citrated Calcium Cyanamide (Calcium carbamide)

Alcohol reaction milder.

Metronidazole

Drugs as aversive stimuli

Associate a highly unpleasant stimulus with the alcoholic beverage.

Emetine / apomorphine | causing vomiting ; problem of timing.

Psychotomimetic drugs

Lysergic acid diethylamide (LSD) — generally ineffective

Treatment of withdrawal symptoms —

Chlordiazepoxide (and probably other benzodiazepines) is safe and effective and can abort or avert delirium tremens.

Promazine is contra-indicated.

Alcoholic liver disease

Mortality due to cirrhosis in different countries closely correlated with the amount of alcohol consumed and is not related to the type of beverage ingested.

Risk factor for development of cirrhosis given by the average daily consumption of alcohol multiplied by the time of consumption at that level.

Drugs which interfere with alcohol consumption

Disulfiram interacts with ingested alcohol and produces flushing, sweating, palpitations, dyspnoea, hyperventilation, tachycardia, hypotension, nausea and vomiting. These are followed by drowsiness with complete recovery after sleep. The reactions are presumably dose dependent.

250 mg of disulfiram a day or less 7 ml of ethyl alcohol	sufficient to produce the reaction

Disulfiram is fat soluble and long acting; patients may still react for as long as seven days after they stop taking disulfiram.

Disulfiram action was discovered by mistake. Two Danish scientists took the drug themselves to assess its safety as a vermifuge. While on disulfiram, they went to a cocktail party with disastrous results. They inferred, correctly, that the drug might be useful in preventing alcohol consumption.

Disulfiram probably acts by blocking the metabolism of alcohol, inhibiting the further oxidation of acetaldehyde. The drug also inhibits the conversion of dopamine to nor-epinephrine by blocking the enzyme dopamine β hydroxylase.

Patients on disulfiram should avoid drug-based forms of alcohol.

Disulfiram should not be given to organically confused, psychotic, severely depressed or suicidal patients.

Disulfiram is contraindicated in pregnancy because of the possible danger to the foetus and perhaps in heart disease.

If a patient is taking diphenylhydantoin, the dose must be decreased; toxicity may result since disulfiram slows its metabolism.

April 10, 1987
82.02/415/87.

Mrs. V Ramakrishnan
5/9 Milton Street, Cooke Town
BANGALORE - 560 005

Dear Mrs. Ramakrishnan,

Sub: Request for setting up a Ward for Alcoholics

Ref: Your letter dated March 24, 1987

I am thankful to you for your letter and the concern you have expressed in your letter. I would have liked to give a completely positive response to your suggestion as alcoholism is an important area where help is needed and can be given.

We are having plans to start a unit to deal with alcoholism under our proposed family care centre. Unfortunately the two qualified and experienced persons (one ~~was~~ a counsellor with Doctorate from NIMHANS and another a Psychiatrist who had been working at St. John's Medical College Hospital) whom we had identified as the possible persons to be in charge of the Unit are no longer available in the city.

We shall continue to look for others and, if we find suitable persons, we would like to start the Unit. We would not like to take up responsibility till we are able to discharge it well.

We have not been thinking of putting up a new ward for the purpose, because

(1) Our emphasis was to be on prevention and education, and

(2) We do not have the resources to put up a new ward and we are unable to re-assign one of the existing wards for the purpose.

With regards,

Yours sincerely,

CM
13/4

Dr. C. M. Francis
Director

Mrs. W. Ramakrishnan

Residence :
5/9, Milton Street, Cooke Town
BANGALORE-560 005 ☎ 562161
Office :
Reserve Bank of India
Nrupathunga Road
BANGALORE - 560 002

24.3.1987

The Director (Shri C.M.Francis)
St. Martha's Hospital,
Nrupathunga Road,
BANGALORE.

Dear Sir,

Request for setting up a Ward for Alcoholics.

I wish to bring these few lines for your consideration and necessary action.

In Reserve Bank of India a number of employees have got themselves addicted to alcohol thus ~~rmx~~ ruining themselves physically and mentally and their jobs are also at stake. In the past few years many premature deaths due to excessive consumption of alocohol have rendered their families homeless with no hope of a bright day in their lives.

St. Johns Medical Hospital and Nimhans have been providing medical assistance as they have equipped themselves with a ward which has rendered phychiatric treatment as well as by administration of anti-abuse drugs to alcoholics and in many cases the victims have improved/got rid of their addiction and have been cured and have been leading happier and better lives thereafter.

Your hospital being ~~xi~~ in the heart of the City would facilitate such people immensely if a ward is set up for the purpose. We have about 200 severe cases which would be referred to you from time to time and I am sure if your hospital establishes a ward on these lines, the service rendered to such addicts would help the victims to live longer, cleaner and better lives making a better society.

I hope you would consider the project and do the needful in the matter.

Thanking you,

Yours faithfully,

(MRS. W. RAMAKRISHNAN)

WINIFRED RAMAKRISHNAN

FIH - 2A - 24

T.T. RANGANATHAN CLINICAL RESEARCH FOUNDATION
3 D'Monte colony, Madras 18

WHO ?..... ME ?
(For family members, relatives and friends of Alcoholics)

In order to determine whether or not, the Alcoholism of another person affects you, it is well to check over a List of Twenty questions and answer TRUTHFULLY each one of them.

1. Do you lose sleep because of a problem drinker ?

2. Do most of your thoughts revolve around the problem drinker or problems that arise because of him or her ?

3. Do you exact promises about the drinking which are not kept?

4. Do you make threats or decisions and not follow them through ?

5. Has your attitude changed toward this problem drinker (alternating between love and hate) ?

6. Do you mark, hide, dilute and/or empty the bottles of liquor or medication ?

7. Do you think that everything would be O.K. if only the problem drinker would stop or control the drinking ?

8. Do you feel alone - fearful - anxious - angry and frustrated most of the time ?

9. Do you find your moods fluctuating wildly - as a direct result of the problem drinker's moods and actions ?

10. do you feel responsible and guilty about the drinking problem ?

11. Do you try to conceal, deny or protect the problem drinker ?

12. Have you withdrawn from outside activities and friends because of embarrassment and shame over the drinking problem ?

13. Have you taken over many chores and outlets that you would normally expect the problem drinker to assume - or that were formerly his or hers ?

14. Do you feel forced to try to exert tight control over the family expenditure with less and less success - and are financial problems increasing ?

15. Do you feel the need to justify your actions and attitude and, at the same time, feel somewhat smug and self-righteous compared to the drinker ?

16. If there are children in the house, do they often take sides with either the problem drinker or the spouse ?

17. Are the children showing signs of emotional stress, such as -withdrawing - having trouble with authority figures- rebelling - acting out sexually ?

18. Have you noticed physical symptoms in yourself, such as nausea/ a 'knot' in the stomach/ ulcers/ shakiness/sweating palms /bitten finger nails ?

19. Do you feel utterly defeated - that nothing you can say or do will move the problem drinker ? Do you believe that he or shee can't get better ?

20. Where this applies, is your sexual relationship with a problem drinker affected by feelings or revolution; do you 'use' sex to manipulate -or refuse sex to punish him or her ?

(YES' to any THREE of these questions indicates that Alcoholism exists and is producing NEGATIVE CHANGES in the person answering them).

T.T. RANGANATHAN CLINICAL RESEARCH FOUNDATION (Regd)
No.6 Cathedral Road, Madras 600 086

DRINKING - THE KEY QUESTIONS

IF you have any questions about your drinking or if someone else is concerned about YOUR drinking, even though YOU are not, the following information will help you to identify the problem.

WHO.....ME ???

IN order to determine whether or not a person has drifted from 'Social' drinking into 'Problem' drinking, it is well to check over a List of Test Questions and answer TRUTHFULLY each one of them. No body else can do it for you.

1. Do you lose time from work due to Drinking ?
2. Is drinking making your home life unhappy ?
3. Do you drink because you are shy with other people ?
4. Is Drinking affecting your reputation ?
5. Have you ever felt Remorse after drinking ?
6. Have you ever gotten into financial difficulties as a result of drinking ?
7. Do you turn to lower companion and an inferior environment when drinking ?
8. Does your drinking make you careless about your family's welfare ?
9. Has your ambition decreased since Drinking ?
10. Do you crave a Drink at a definite time daily ?
11. Do you want a Drink the next morning ?
12. Does Drinking cause you to have difficulty in sleeping ?
13. Has your efficiency decreased since Drinking ?
14. Is Drinking jeopardising your job or business ?
15. Do you Drink to escape worries or troubles ?
16. Do you drink alone ?
17. Have you ever had a complete loss of memory as a result of Drinking ?
18. Has your Physician ever treated you for Drinking ?
19. Do you Drink to build up your self-confidence ?
20. Have you ever been to a hospital or institution on account of Drinking ?

If you have answered 'YES' to any 'ONE' of the questions, there is a definite warning that 'YOU MAY BE ALCOHOLIC'

If you have answered 'YES' to any 'TWO', the chances are that 'YOU ARE ALCOHOLIC'.

If you have answered 'YES' to any 'THREE' or more, 'YOU ARE DEFINITELY ALCOHOLIC'.

ONLY YOU CAN DECIDE:

Remember, ALCOHOLISM is a 'Progressive' disease; it never gets better while Drinking continues, only worse. And, if unchecked, leads to INSANITY or a PAINFUL and PREMATURE DEATH.

FAMILY ENVIRONMENT SCALE

INSTRUCTIONS

There are 90 statements in this booklet. They are statements about families. You are to decide which of these statements are true of your family and which are false. Make all your marks on the separate answer sheets. If you think the statements is TRUE or mostly TRUE of your family, make an X in the box labelled T (True). If you think the statement is FALSE or mostly FALSE of your family, make an X in the box labelled F (False).

1.	Family members really help and support one another.	T	F
2.	Family members often keep their feelings to themselves.	T	F
3.	We fight a lot in our family.	T	F
4.	We don't do things on our own very often in our family.	T	F
5.	We feel it is important to be the best at whatever you do.	T	F
6.	We often talk about political and social problems.	T	F
7.	We spend most weekends and evenings at home.	T	F
8.	Family members attend Religion and/worship places fairly often.	T	F
9.	Activities in our family are pretty carefully planned.	T	F
10.	Family members are rarely ordered around.	T	F
11.	We often seem to be killing time at home.	T	F
12.	We say anything we want to around home.	T	F
13.	Family members rarely become openly angry.	T	F
14.	In our family, we are strongly encouraged to be independent.	T	F
15.	Getting ahead in life is very important in our family.	T	F
16.	Normally go outside home to attend recreational/ entertainment programmes.	T	F
17.	Friends often come over for dinner to visit	T	F
18.	We don't say prayers worship in our family.	T	F

19.	We are generally very neat and orderly	T	F
20.	There are very few rules to follow in our family.	T	F
21.	It is hard to get things done at home without upsetting somebody.	T	F
23.	Family members sometimes get so angry they throw things	T	F
24.	We think things over for outselves in our family.	T	F
25.	How much money a person makes is not very important to us.	T	F
26.	Learning about new and different things is very important in our family.	T	F
27.	Nobody in our family is active in sports, like league, bowling, etc.	T	F
28.	We often talk about the religious meaning of Christmas / festival days.	T	F
29.	It's often hard to find things when you need them in our household.	T	F
30.	There is one family member who makes most of the decisions.	T	F
31.	There is a feeling of togetherness in our family.	T	F
32	We tell each other about our personal problems.	T	F
33.	Family members hardly ever lose their tempers.	T	F
34.	We come and go as we want to in our family.	T	F
35.	We believe in competition and "may the best man win".	T	F
36.	We are not that interested in cultural activities.	T	F
37.	We often go to movies, sports events, camping etc.	T	F
38.	We don't believe in heaven or hell.	T	F
39.	Being on time is very important in our family.	T	F
40.	There are set ways of doing things at home.	T	F
41.	We rarely volunteer when something has to be done at home.	T	F
42.	If we feel like doing something on the spur of the moment we often just pick up and go.	T	F

43.	Family members often criticize each other	T	F
44.	There is very little privacy in our family.	T	F
45.	We always strive to do things just a little better the next time.	T	F
46.	We rarely have intellectual discussions.	T	F
47.	Everyone in our family has a hobby or two.	T	F
48.	Family members have strict ideas about what is right and wrong.	T	F
49.	People change their minds often in our family.	T	F
50.	There is a strong emphasis on following rules in our family.	T	F
51.	Family members really back each other up.	T	F
52.	Someone usually gets upset if you complain in our family.	T	F
53.	Family members sometimes hit each other.	T	F
54.	Family members almost always rely on themselves when a problem comes up.	T	F
55.	Family members rarely worry about job promotions, school grades etc.	T	F
56.	Someone in our family plays a musical instrument.	T	F
57.	Family members are not very involved in recreational activities outside work or school.	T	F
58.	We believe there are some things you just have to take on faith.	T	F
59.	Family members make sure their rooms are neat.	T	F
60.	Everyone has an equal say in family decisions.	T	F
61.	There is very little group spirit in our family.	T	F
62.	Money and paying bills is openly talked about in our family.	T	F
63.	If there's disagreement in our family, we try hard to smooth things over and keep the peace.	T	F
64.	Family members strongly encourage each other to stand up for their rights.	T	F
65.	In our family, we don't try that hard to succeed.	T	F

66.

66. Family members sometimes attend lessons for some hobby or interest. T F

67. Family members often go to learn/acquire something new. T F

68. In our family each person has different ideas about what is right and wrong. T F

69. Each person's duties are clearly defined in our family T F

70. We can do whatever we want to in our family. T F

71. We really get along well with each other. T F

72. We are usually careful about what we say to each other. T F

73. Family members often try to one-up or out-do each other. T F

74. It's hard to be by yourself without hurting someone's feelings in our household. T F

75. "Work before play" is the rule in our family. T F

76. Listening Radio/Stereo/gramophone is more important than reading in our family. T F

77. Family members go out a lot. T F

78. The religious books are very important books in our home. T F

79. Money is not handled very carefully in our family. T F

80. Rules are pretty inflexible in our household. T F

81. There is plenty of time and attention for everyone in our Family. T F

82. There are a lot of spontaneous discussions in our family T F

83. In our family, we believe you don't ever get anywhere by raising your voice. T F

84. We are not really encouraged to speak up for ourselves in our family. T F

85. Family members are often compared with others as to how well they are doing at work or school. T F

86. Family members really like music, art and literature. T F

87. Our main form of entertainment is listening Radio/Stereo/ Gramophone. T F

88. Family members believe that if you sin you will be punished. T F

89. Generally we maintain our home neat and clean. T F

90. You can't get away with much in your family. T F

Please check ( ) the answer below that best describes your feelings, behaviour, and experiences related to a parent's alcohol use. Take your time and be as accurate as possible.

Name: Age

Sex : Education:

Religion: Caste:

Occupation: Income:

Position in the family: Status:

~~Family Background~~

Name	Relationship	Education	Occupation	Income
1.				
2.				

Drinking parent's name:

Occupation:

Income:

Duration of alcohol intake:

Treatment undergone:

Diagnoses:

1. Have you ever thought or known that one of your parents had a drinking problem? Yes/no

2. Because of a parent's drinking have you ever
   (a) lost sleep
   (b) worried
   (c) cried
   (d) all the above
   (e) none
   (f) others -specify

3. Did you ever encourage one of your parents to stop drinking?
   If yes - How? If no, why?

* Kindly elaborate on your answers whenever possible. If more than one item applies, list it-1,2, 3.....

4. Because your parent was drinking did you ever feel
   (a) alone
   (b) scared
   (c) nervous
   (d) angry
   (e) none of the above
   (f) all the above
   (g) others -specify

5. When your parent was drinking did you ever
   (a) argue
   (b) abuse
   (c) fight
   (d) others - specify

6. Because of a parent s drinking did you ever
   (a) threaten to run away
   (b) hurt yourself
   (c) threaten suicide
   (d) others - specify

7. Has a problem drinking parent e while drinking ever
   (a) yelled at you
   (b) hit you
   (c) threatened yoy
   (d) others - specify

8. Have any of your a family members been threatened when a parent was drinking?

   If yes - Who?
   How?

9. Have you heard your parents figat when one of them was drunk? Yes/ No

   If yes - About what did they fight

10. Did you ever protect another family member from a parent who was drinking ? Yes/ No
    If yes
    Whom and How?

11. Have you ever taken a parent's bottle of liquor and
    (a) hide it
    (b) empty it
    (c) broken It
    (d) other's specify

12. Do your thougats revolve around a problem drinking parent ?

Always/ Sometimes/ Rarely

13. Do you think about the difficulties caused by your parent's drinking ?

Always/ Sometimes/ Rarely

14. Did you ever wish that a drinking parent would
(a) moderate his drinking
(b) drink rarely
(c) stop drinking
(d) others - specify

15. Did you ever feel that you were the cause for your parent's drinking? Yes/ No
If yes - Why?

16. Due to alchhol use do you ever fear about your parents getting (a) divorced
(b) separated

17. Because of parent's drinking problem were outside activities ever
(a) withdrawn from
(b) avoided)

18. Have you ever stayed away from frimnds because a parent's drinking problem caused
a) embarassment
b) shame
c) ridicule
d) others - specify

19. In the midd le of an arguement or fight between a problem drinking parent and your other parent did you ever
(a) intervene
(b) take sides
(c) feel caught
(d) others - specify

20. Do you think that your parent dramk alchol because of
(a) you
(b) sisters/brothers
(c) other parent
(d) family members
(e) others - specify.

21. Do you feel that a problem drinking parent did not really love you?

Yes/ No

If yes - Why?

22. What do you think about a parent's drinking?
(a) resent
(b) dislike
(c) hate
(d) indifferent
(e) like
(f) others - specify

23. Because of a parent's alcohol use, have you ever worried about a parent's
(a) physical health
(b) emotional well-being
(c) social status

24. Have you ever been blamed for a parent's drinking?

25. Do you think your father is an alcoholic ?

26. Do you compare and want your home life to be like that of your friends without drinking parents?

27. Because of drinking has your parent ever made promises which could not be kept ?
Yes/ No
If yes, how often?

28. About the alcohol related issues in your family did you ever speak with a
(a) priest
(b) friend
(c) other family members
(d) brothers/sisters
(e) non-drinking parent
(f) others - specify

If no, Why?

29. Did you ever fight with your brothers and sisters about a parents drinking?

30. To avoid the drinking parent do you
(a) stay away from home
(b) come late
(c) stay in your room
(d) others - specify

31. Do you avoid the other parent's reaction to drinking?

Yes/ No

If yes, why? How?

32. Did you ever take over the chores and responsibilities at home that e were usually done by a parent before he or she developed a drinking problem?

Never/ sometimes/ often/ rarely/ always

33. Did you ever consult a professional about your parent's alcohol problem?

(a) psychiatrist
(b) psychologist
(c) social worker
(d) medical doctor

THANK YOU for your cooperation.

SOCIAL ASPECTS OF ALCOHOLISM [1]

By

Mrs. Lalita Bhatti, M.S.W., D.P.S.W. [2]

The recognition of the role of socio-psychological factors in causing and maintaining alcoholism has brought a change in the disease notion of alcoholism as a physical entity. Recently there has been an acceptance of the term 'Problem drinking' instead of 'Alcoholism'. It is argued that problem drinking places emphasis upon behaviour rather than on the person and thus avoids the more permanent label alcoholism, which tends to be attached to thepperson. This shift is mainly due to the fact that the popular approaches like-constititional, psychological and sociological cannot help to understand and treat alcoholism independent of each other.

Two facets of Social Aspects -

The workers who strongly ahdere to the constititional approch do not give any importance to the social factors in understanding the etiology of alcoholism. Most of them, of course, agree that alcoholism could be an exogeneous stress on the family and social milien of the alcoholic. During the third decade of this century, Knight (1937) and Chassell (1938) stressed the importance of understanding the total family in order to understand individual drinking behaviour. Meeks (1976) comments, 'drinking may indicate stress or dysfunction in a social system, may be secondary to dysfunction in a social system', seems to be quite appropriate. He is of the opinion that 'some alcohol problems may reside as much as in social structures and processes as in people'.

Socio-cultural theory of Alcoholism -

Most of the epidemiological surveys have, demonstrated that the incidence of drinking have a significant association with age, sex, social status, ethnicity, degree of urbanization, quality of marital and family life. These are all sociological and demographic variables. On the other hand, in the field of psychological research no unique personality type or a unique nosological group have shown definite correlation with alcoholism (Roebuck & Kessler, 1972). Therefore, as pointed out by Cahalan etal (1969), 'Whether a person drinks at all is primarily a sociological and anthropological variable rather than a psychological one', needs a very special consideration.

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1. Paper presented at Seminar on Alcoholism organised by the Dept. of Psychiatry, St. John's Medical College & Hospital and Indian Psychiatry Society, Karnataka State Branch, on 6-6-1983.
2. Department of Community Medicine, St. John's Medical College, Bangalore - 34

Twentieth century is the age of positivism and anxiety. The technological advancements in the European and Western Society have brought a severe degree of monotomy in life. This has affected the youth very much. He finds himself as an empty shell - his family as an empty shell - his whole system as an empty shell. He is in constant search of relations and significant others. He is desperately searching meaning of life. This is an indication of acculturation of society. Whenever a society is undergoing acculturation the culturally induced tensions will reach to an untolerable level. These tensions lead to existential doubts. Roebuck & Kessler (1972) believe that 'the choice of alcohol to relieve these is determined by (1) attitudes towards alcohol and (2) the availability of substitute means of satisfaction or tension release. A similar review is expressed by Cahalan (1970) and he contended that higher rates of problem drinking in lower socio economic group might be due to fewer opportunities for recreation and tension release.

Family of Alcoholics -

The behavioural scientists believe that Alcoholics' family of orientation is one of the very important factors in understanding the problem of alcoholism. In this regard several attempts have been made to explain the various socio-psychological tenets of family life. Most of the work in this field is based on the general system theory wherein the family has been considered as an open system. The expression of 'abusive drinking' as per the family system, is considered as a sign of stress within the family. Often it is seen drinking in family starts as a substitute in the absence of usual coping mechanisms. The other view with regard to the abusive drinking is that it maintains the family as a system. Alcoholism brings stability rather than disruption in the interactional behaviour in certain families. Jackson (1954) reported the following seven stages in the adjustment of the family to alcoholism.

intact families

1. Attempts to deny the problem
2. Attempts to eleminate the problem
3. Disorganization
4. Attempts to reorganize inspite of the problem
5. Efforts to escape the problem
6. Reorganization of the family
7. Recovery and reorganization of the whole family.

Parent child relations in the families of alcoholics -

Wittman (1939) has given an account of the parent child relationship. According to her the alcoholics have oversolicitors mother and a comparatively

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stern, fobidding father; the later, the person who inspired and awe or fear and who displayed inconsistant tendencies of severity and indulgence, thus producing in the child a feeling of insecurity and helpless dependence. Shiela Daniel also found that alcoholic parents were not consistant in their actions towards children.

Parental drinking attitudes -

Jackson and Connor (1957) have shown that alcoholics came more frequently from houses in which one parent drank - usually the father. With regard to the families of alcoholism in the Indian set up according to Bhatti (1982) Channabasavanna & Bhatti (1981) and Channabasavanna and Bhatti (1983) most of the alcoholics belong to anomic families. According to them majority of the cases came from the families having unhealthy communications, poor concern and lack of leadership. The individual members have their own way of life, style of interaction and personal convictions which are often idiosyncratic. They are highly individualistic and do not bother about other family members. They hardly have any discussion and no common ways are adopted to achieve the family goals. In extreme cases, except living under common roof the family members have nothing else in common. These are called the anomic families. Anomic families contribute heavily for alcoholism and drug addiction.

In such families, the individual self is given the highest importance by the family as such. Often in such families regular leader of the family moves out quite frequently and in his/her absence some other family member accepts the leadership. Such acceptance of leadership is always to fill the gap. Therefore the leader is quite mild, non-commital, highly indecisive, rarely enters into any kind of discussion and leaves everything to others; such a leader pretends to be a broad minded leader. Also in such families due to the permanent incapacitation or the death of the actual leader, some member of the family is forced to accept the leadership. The patterns of communications are quite confusing in these families. There is always an atmosphere of imposition and overacherence. When the leader conveys the messages they are interpreted in comparison with the way the messages were being conveyed by the original leader. At times the messages are quite contradictory. The messages reflect more often the covert meaning which remains a guess work for the receiver. In such families the leader does not posses the role of a leader, still plays the role of a leader. The roles are allocated but not accepted. The patterns of reinforcement are usually temporary in nature. Such families make use of negative patterns of reinforcement. In crisis, such families turn to governmental and voluntary agencies.

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Schematic analysis of family system of alcoholics

Type of family	-Anomic type
Type of self	-Individual self
Type of leadership	-Marginal and/or stop-gap leadership
Type of communication	-Messages without any meaning and misinterpretation of the messages
Type of role	-Cognitive discrepancy and discrepancy of role
Type of reinforcement	-Through coercion and punishment
Type of social support system	-Tertiary social support system

Social class and alcoholism

In general survey results indicate that percentage of drinkers increase with increasing social status. On the other hand rates of heavy drinking, heavy escape drinking and problem drinking among drinkers are highest in lower status groups.

The middle and lower upper class might be expected to have high rate of alcoholism because of the tensions and insecurity brought about by high speed of living, industrial and commercial activity and high pressures in life. The lower classes would be expected to have high rates because of their supposed lack of controls on drinking. One problem that confronts researchers who study the association between drinking patterns and social class is the matter of social class criteria. Sociologists utilise different methoddogies and criteria in the stratification area. The four most frequently used indicators of social class are income, education, occupation and some combination of these. Cahalan etal (1969) found that heavy escape drinkers had relatively lower income [illegible] problem drinking related to age, sex and urbanization. Men in all age groups have a higher frequency of drinking problems than do women. Cahalan holds that role differences between men and women explain men's heavier drinking. The frequency of drinking problems in the aggregate among men was found to be highest among those in their twenties, significantly lower among those in their thirties and forties, and tapering off among those in their fifties. The degree of urbanization is related in certain ways to drinking behaviour, depending upon two variables - age and socialstatus. In conslusion, I would like to reitrate that the research in the field have established that the etiology of alcohol abuse and aocoholism is multifactorial. Equally, it is proved beyond doubt that the management outcome is always better when the family of an alcoholic has participated actively in the treatment programme-well that is the relevance of social factors in alcoholism.

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Chemical Addiction Information Monitoring

C A I M TREATMENT CENTRE

For the treatment of Alcoholism,
Addiction to cocaine, heroin,
barbiturates, amphetamines, or
abuse of prescribed medications.

318, 15th Cross, Sadashivanagar, Bangalore - 560 080. Phone : 363438

Chemical Dependency

The loss of control and craving for alcohol or other mood-changing drugs is a treatable illness. It can happen to anyone, regardless of circumstances of person, background, race, sex, income profession or education.

Alcohol, cocaine, minor tranquilizers, barbiturates, heroin, amphetamines, LSD or mescaline, or abuse of doctor prescribed medication, can cause disease or ill-health and dysfunctioning of the body, mind, emotions, spirit (values) and relationships with family, occupation, and society at large.

TWO CLINICAL - MEDICAL FACTS

One : The pathologies (problems) that develop as the result of the misuse of any of the mind-altering or mood-changing drugs are roughly the same.

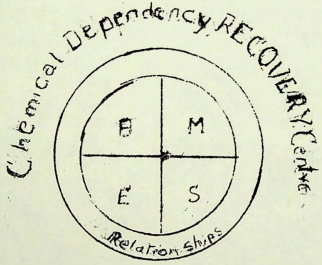
Two : The Recovery Process is essentially the same, regardless of the type of mind-altering or mood-changing chemical.

TREATMENT SERVICES

The C A I M Treatment Centre of Bangalore is an intensive, extensive 12-step centered service for the treatment of the disease of chemical dependency. The treatment methods include an in-patient program of variable length stay as indicated; as well as out-patient services individualized to the patient's needs. Counselors function with physicians to meet the medical and psychosocial needs of participants.

Family Therapy

Recovery requires that family members learn how to communicate with each other and deal openly with their feelings. Family Therapy is an integral part of any treatment plan, as the disease of Co-Dependency exists within the family system.



Restoring the Body, Mind, Emotion,
Spirit and Relationships

Goal

The program of the Chemical Dependency Recovery Centre is specifically focused and designed to restore the chemically dependent person and their family members to optimal health and functioning of Body, Mind, Emotion, Spirit (Values), and Relationships.

Achieving that Goal

In order to accomplish that clear goal of lasting recovery (getting clean and sober and practicing to stay that way comfortably), active participants in our program go through, or work on, seven areas or processes :

1. BODY

   Detoxification safely; optimize physical health.

2. MIND

   Learn about the disease of chemical dependency; and choose new ideas and attitudes over dysfunctional old ideas and belief systems.

3. EMOTIONS

   Choose to learn how to identify, clarify and express feelings.

4. SURRENDER

   Choose to learn how to use the superior tactic of surrender. Learn to improve the quality of acceptance of having the disease of chemical dependency.

5. PERSONALITY AND CHARACTER

   Choose to identify what aspects of your own personality or character have interfered - until now - with your ability to choose your own recovery, holistic health and happiness.

6. RELATIONSHIPS

   Choose to restore healthy relationships to yourself, to others (family, job, society), and to the natural HEALING POWERS of the Universe.

7. PROGRAM OF RECOVERY

   Choose to learn how to use the most effective long-term Program of Recovery for your own benefit, to ensure your own lasting recovery, once you leave the hospital.

Ways we can be of service :

* We provide Family Intervention and Help.
  Call 363438/385622/385234/385913

* We maintain an Open Door Policy and Practice which says that we are available to help anyone suffering from the disease of chemical dependency or co-dependency, family and friends, regardless of circumstances.

* We provide Education, Information, and Referral Services 385622/363438/385913/385234

* Speakers, seminars, and training provided to the general community, professional groups, and service organizations.

* , full-scale implementation of an Employee Assistance Program (including personnel training) at your agency or business by an experienced EAP Consultant.

* On-going Therapy meetings and numerous self-help meetings on the unit.

RECOVERY begins with a single phone call /
363438/385622/385234/385913

ADMISSION to the CAIM Treatment Centre is facilitated with an absolute minimum of bureaucratic fuss and delay.

"The good is too often the enemy of the best"

Bill W.

Criteria for the Diagnosis of Alcoholism

BY THE CRITERIA COMMITTEE, NATIONAL COUNCIL ON ALCOHOLISM

These criteria were compiled by a committee of medical authorities from the National Council on Alcoholism to establish guidelines for the proper diagnosis and evaluation of this disease. Criteria are weighted for diagnostic significance and assembled according to types: Physiological and Clinical (including major alcohol-associated illnesses) and Behavioral, Psychological, and Attitudinal. Because early diagnosis is helpful in treatment and recovery, manifestations are separated into their earlier and later phases. There are brief discussions of recurrent and arrested alcoholism, cross-dependence, and the types of persons at high risk of alcoholism.

THE PROBLEM of alcoholism has been receiving increasing interest in the past few years. Extensive treatment programs are being mounted, hospitals are beginning to accept patients for treatment, labor-management programs are attempting to identify alcoholic employees to give them special benefits and rehabilitation, third-party payments are being afforded by insurance carriers, and courts are making special disposition for rehabilitation. Therefore, it is important to establish a set of criteria for the diagnosis of alcoholism. To this end, the National Council on Alcoholism established a committee[1] to prepare a set of criteria, to submit it for criticism and documentation by other experts, and to publish it for the guidance of those involved in the diagnosis of alcoholism.

At the outset, it became apparent that we had undertaken a formidable task, for, despite a great deal of work in the past, much of the literature is burdened by anecdotal material and special assumptions made a priori, and there is a dearth of scientifically controlled observations on the natural course of the disease. In addition, people of many disciplines have made observations from their own points of view, which may be hard to reconcile, and there are not a few who, by their definition of disease, have eliminated alcoholism from the category of disease. But any tendency to withdraw from the field was overcome by the urgency of the task, and the committee herewith presents the results of its deliberations.

Diagnostic criteria may serve several purposes. They may be used *to ascertain the nature of a disease* from a cluster of symptoms. This was not the main goal of the committee. They may be used to promote *early detection* and provide *uniform nomenclature*, both objects of this endeavor. Criteria may be used to *prevent overdiagnosis*. This is important because of the psychological, financial, legal, and therapeutic implications in a diagnosis of alcoholism for the life of the patient. Criteria may be set for *treatment* purposes. Beyond indicating that a need for treatment exists, the committee believes that any indication of different modalities of treatment, except in broad terms, is beyond the scope of its mandate. Criteria may be set for *prognosis*; at present the prognosis for alcoholism is obscure.

Mainly, the committee expects the criteria to be used to identify individuals at multiple levels of dependency. The committee has endeavored to use objectively reproducible data that are obtainable from the patient, his immediate family, or his associates. These data have been weighted for their diagnostic significance. We have included material that would differentiate degrees of severity and that would allow for progression of the disease, where that exists, without prejudging

Editor's Note: These criteria are being published simultaneously in the *Journal* and in *Annals of Internal Medicine* by agreement of the Editors and the National Council on Alcoholism. See page 214 for an editorial by Morris Chafetz, M.D.

Reprints of this article may be obtained from the Publications Division, National Council on Alcoholism, Inc., 2 Park Ave., New York, N. Y. 10016, for $1 each. Remittance must accompany orders under $5. There is a ten percent discount for orders of 50 or more copies.

[1] Members of the committee are listed in Appendix I.

the possibility that cases of alcoholism may exist in which progression is not a factor. All but one consultant believed that, in alcoholism, there generally is a progression of the disease, although this might not necessarily be reflected by continually increasing drinking. Many consultants have exhorted us to concentrate more on "early manifestations." The reader will note a separation into early, middle, and late effects, which is a general guide. Our first intent, however, is that the person who is diagnosed as having alcoholism surely fits into that category.

The Nature of Alcoholism

The committee was unanimous in defining the disease of alcoholism as a pathological dependency on ethanol, as it is classified under Section 303.2 in the *Diagnostic and Statistical Manual of Mental Disorders*, second edition, of the American Psychiatric Association.

Aside from the legal difference between the distribution of alcohol and that of other drugs, there are important scientific differences. A drug is defined in two senses: it is a substance of use in medicine, and it is a habit-forming substance. It generally produces its effect in small quantities. Although alcohol does produce an effect with small quantities, it differs from other drugs in both senses in that large quantities over a long period of time are necessary for it to become habit-forming.

Another difference between alcohol and other drugs, particularly those of the opiate class, is the relative risk of addiction. Many people drink, but only ten percent develop the psychological and physiological dependency on alcohol that can be categorized as alcoholism. With opiates, the risk of pharmacological addiction is considerably higher. Many alcoholics believe that they were alcoholics from their first drink, that their reaction to alcohol was different from that of others. These retrospective data are suspect until and unless a clear difference is established between these individuals and others. Family incidence of alcoholism and other factors may indicate a portion of the population at high risk.

Whether *anyone* who drinks a sufficient quantity over a sufficient period of time will develop alcoholism, whether a specific biochemical or psychological difference leads to alcoholism, or whether both conditions (with other as yet undetermined factors possibly turning the balance) are necessary to cause alcoholism has not yet been established. Thus, whether there is a continuous or discontinuous progression from drinking alcoholic beverages to dependency on alcohol has not yet been clearly decided. Animal data suggest that anyone who drinks enough over a sufficiently long period of time will develop the signs of alcoholism. In the free state, however, neither all humans nor all animals choose the paths that lead to this condition. In establishing criteria for diagnosis, the committee wishes to avoid prejudging these issues of etiology.

On the other hand, once alcoholism is established, there is general consensus on its manifestations, and the committee thus feels it is appropriate to describe it as a disease, in agreement with the American College of Physicians, the American Medical Association, the American Psychiatric Association, and other bodies. Alcoholism fits the definition of disease given in *Dorland's Illustrated Medical Dictionary*, 24th edition:

> A definite morbid process having a characteristic train of symptoms; it may affect the whole body or any of its parts, and its etiology, pathology, and prognosis may be known or unknown.

Partial and intermittent forms of alcoholism pose some problems that will be treated separately. Isolated episodes of inebriation, even if they generate unfortunate consequences, are eliminated.

Divisions of Data

Data are assembled according to the type of material they represent. Therefore, there are separate data "tracks"—Track I: Physiological and Clinical, and Track II: Behavioral, Psychological, and Attitudinal. The Track II data are grouped together because behavioral manifestations, the easiest to determine and most objective to recognize, imply attitudinal and psychological manifestations.

There is no rigid uniformity in the progress of the disease, but, since early diagnosis seems to be helpful in treatment and recovery, manifestations are separated into "early," "middle," and "late." In addition to identifying early and late symptoms and

TABLE 1

Major Criteria for the Diagnosis of Alcoholism

CRITERION	DIAGNOSTIC LEVEL
TRACK I. PHYSIOLOGICAL AND CLINICAL	
A. Physiological Dependency	
1. Physiological dependence as manifested by evidence of a *withdrawal syndrome** when the intake of alcohol is interrupted or decreased without substitution of other sedation.** It must be remembered that overuse of other sedative drugs can produce a similar withdrawal state, which should be differentiated from withdrawal from alcohol.	
a) Gross tremor (differentiated from other causes of tremor)	1
b) Hallucinosis (differentiated from schizophrenic hallucinations or other psychoses)	1
c) Withdrawal seizures (differentiated from epilepsy and other seizure disorders)	1
d) Delirium tremens. Usually starts between the first and third day after withdrawal and minimally includes tremors, disorientation, and hallucinations.*	1
2. Evidence of *tolerance* to the effects of alcohol. (There may be a decrease in previously high levels of tolerance late in the course.) Although the degree of tolerance to alcohol in no way matches the degree of tolerance to other drugs, the behavioral effects of a given amount of alcohol vary greatly between alcoholic and nonalcoholic subjects.	
a) A blood alcohol level of more than 150 mg. without gross evidence of intoxication.	1
b) The consumption of one-fifth of a gallon of whiskey or an equivalent amount of wine or beer daily, for more than one day, by a 180-lb. individual ***	1
3. Alcoholic "blackout" periods. (Differential diagnosis from purely psychological fugue states and psychomotor seizures.)	2
B. Clinical. Major Alcohol-Associated Illnesses. Alcoholism can be assumed to exist if major alcohol-associated illnesses develop in a person who drinks regularly. In such individuals, evidence of physiological and psychological dependence should be searched for.	
Fatty degeneration in absence of other known cause	2
Alcoholic hepatitis	1
Laennec's cirrhosis	2
Pancreatitis in the absence of cholelithiasis	2
Chronic gastritis	3
Hematological disorders:	
Anemia: hypochromic, normocytic, macrocytic, hemolytic with stomatocytosis, low folic acid	3
Clotting disorders: prothrombin elevation, thrombocytopenia	3
Wernicke-Korsakoff syndrome	2
Alcoholic cerebellar degeneration	1
Cerebral degeneration in absence of Alzheimer's disease or arteriosclerosis	2
Central pontine myelinolysis } diagnosis only	2
Marchiafava-Bignami's disease } possible postmortem	2
Peripheral neuropathy (see also beriberi)	2
Toxic amblyopia	3
Alcohol myopathy	2
Alcoholic cardiomyopathy	2
Beriberi	3
Pellagra	3
TRACK II. BEHAVIORAL, PSYCHOLOGICAL, AND ATTITUDINAL	
All chronic conditions of psychological dependence occur in dynamic equilibrium with intrapsychic and interpersonal consequences. In alcoholism, similarly, there are varied effects on character and family. Like other chronic relapsing diseases, alcoholism produces vocational, social, and physical impairments. Therefore, the implications of these disruptions must be evaluated and related to the individual and his pattern of alcoholism. The following behavior patterns show psychological dependence on alcohol in alcoholism:	
1. Drinking despite strong medical contraindication known to patient	1
2. Drinking despite strong, identified, social contraindication (job loss for intoxication, marriage disruption because of drinking, arrest for intoxication, driving while intoxicated)	1
3. Patient's subjective complaint of loss of control of alcohol consumption	2

*See Seixas (1).
**Some authorities term this "pharmacological addiction."
***For equivalent amounts in wine and beer, see Appendix 2.

signs, each datum was graded according to its degree of implication for the presence of alcoholism. Of course, some of the more definite signs occur later in the course of the illness. But this does not mean that people with earlier signs may not also have alcoholism.

Various terminologies for these signs have been suggested; we propose to weight them and group them into three "diagnostic levels," with those weighted as "1" being the most significant.

Diagnostic Level 1. Classical, definite, obligatory: A person who fits this criterion must be diagnosed as being alcoholic.

Diagnostic Level 2. Probable, frequent, indicative: A person who satisfies this criterion is under strong suspicion of alcohol-

ism; other corroborative evidence should be obtained.

Diagnostic Level 3. Potential, possible, incidental: These manifestations are common in people with alcoholism, but do not by themselves give a strong indication of its existence. They may arouse suspicion, but significant other evidence is needed before the diagnosis is made.

Diagnosis

It is sufficient for the diagnosis of alcoholism that one or more of the major criteria are satisfied, or that several of the minor criteria in Tracks I and II are present; see tables 1 and 2. If one is making the diagnosis because of major criteria in one of the tracks, he should also make a strong search for evidence in the other track. A purely mechanical selection of items is not enough; the history, physical examination, and other observations, plus laboratory evidence, must fit into a consistent whole to ensure a proper diagnosis. Minor criteria in the physical and clinical tracks alone are not sufficient, nor are minor criteria in behavioral and psychological tracks. There must be several in *both* Track I and Track II areas.

Psychiatric Diagnosis

After a suitable evaluation, a separate psychiatric diagnosis should be made on every patient, apart from the diagnosis of alcoholism. Patients may suffer from schizophrenia, latent or overt; from manic-depressive psychosis, obsessive-compulsive neurosis, recurrent depression, anxiety neurosis, or psychopathic personality; or have no psychiatric constellation differing from normal. The diagnosis should properly be

TABLE 2

Minor Criteria for the Diagnosis of Alcoholism

CRITERION	DIAGNOSTIC LEVEL
TRACK I: PHYSIOLOGICAL AND CLINICAL	
A. Direct Effects (ascertained by examination)	
1. Early:	
Odor of alcohol on breath at time of medical appointment	2
2. Middle:	
Alcoholic facies	2
Vascular engorgement of face	2
Toxic amblyopia	3
Increased incidence of infections	3
Cardiac arrhythmias	3
Peripheral neuropathy (see also Major Criteria, Track I, B)	2
3. Late (see Major Criteria, Track I, B)	
B. Indirect Effects	
1. Early:	
Tachycardia	3
Flushed face	3
Nocturnal diaphoresis	3
2. Middle:	
Ecchymoses on lower extremities, arms, or chest	3
Cigarette or other burns on hands or chest	3
Hyperreflexia, or if drinking heavily, hyporeflexia (permanent hyporeflexia may be a residuum of alcoholic polyneuritis)	3
3. Late:	
Decreased tolerance	3
C. Laboratory Tests	
1. Major—Direct	
Blood alcohol level at any time of more than 300 mg./100 ml.	1
Level of more than 100 mg./100 ml. in routine examination	1
2. Major—Indirect	
Serum osmolality (reflects blood alcohol levels): every 22.4 increase over 200 mOsm/liter reflects 50 mg./100 ml. alcohol	2
3. Minor—Indirect	
Results of alcohol ingestion:	
Hypoglycemia	3
Hypochloremic alkalosis	3
Low magnesium level	2
Lactic acid elevation	3
Transient uric acid elevation	3
Potassium depletion	3
Indications of liver abnormality:	
SGPT elevation	2
SGOT elevation	3
BSP elevation	2
Bilirubin elevation	2
Urinary urobilinogen elevation	2
Serum A/G ration reversal	2
Blood and blood clotting:	
Anemia: hypochromic, normocytic, macrocytic, hemolytic with stomatocytosis, low folic acid	3
Clotting disorders: prothrombin elevation, thrombocytopenia	3
ECG abnormalities	
Cardiac arrhythmias; tachycardia; T waves dimpled, cloven, or spinous; atrial fibrillation; ventricular premature contractions; abnormal P waves	2
EEG abnormalities	
Decreased or increased REM sleep, depending on phase	3
Loss of delta sleep	3
Other reported findings	3
Decreased immune response	3
Decreased response to Synacthen test	3
Chromosomal damage from alcoholism	3

made in the dry state, since alcohol is anxiety-producing and can also bring out psychological mechanisms and traits that are not apparent without alcohol. In particular, the hallucinatory behavior induced by alcohol withdrawal is not to be equated with schizophrenic hallucinatory behavior.

Alcoholism with Intermittent or Recurrent Drinking

Intermittent or recurrent drinking may represent a phase in the course of alcoholism. This pattern should be noted separately. The same criteria control the diagnosis. In some individuals there are recurring episodes of inebriation that become more frequent over a period of years until a daily drinking pattern emerges. In many individuals daily drinking increases until the individual himself slowly becomes aware that physiological and psychological dependency exist. At this point periods of "going on the wagon" may occur, with a resulting intermittent or recurrent pattern of drinking. For most drinkers, there are lesser or greater periods of time when, because of circumstances or the acute effects of alcohol, drinking is not possible. This pattern is not inconsistent with other drug dependency situations, in which interruptions of use are commonplace and have been accepted without the necessity of making a separate category for them.

Even with a "steady" pattern of alcohol use, there are marked fluctuations in the blood alcohol level during each day. The patient with an alcohol problem, given free choice, does not, as one might assume, keep drinking to maintain a steady blood level of alcohol. It has been observed that men who

TABLE 2 cont.'d
Minor Criteria for the Diagnosis of Alcoholism

CRITERION	DIAGNOSTIC LEVEL
TRACK II. BEHAVIORAL, PSYCHOLOGICAL, AND ATTITUDINAL	
A. Behavioral	
1. Direct effects	
Early:	
Gulping drinks	3
Surreptitious drinking	2
Morning drinking (assess nature of peer group behavior)	2
Middle:	
Repeated conscious attempts at abstinence	2
Late:	
Blatant indiscriminate use of alcohol	1
Skid Row or equivalent social level	2
2. Indirect effects	
Early:	
Medical excuses from work for variety of reasons	2
Shifting from one alcoholic beverage to another	2
Preference for drinking companions, bars, and taverns	2
Loss of interest in activities not directly associated with drinking	2
Late:	
Chooses employment that facilitates drinking	3
Frequent automobile accidents	3
History of family members undergoing psychiatric treatment; school and behavioral problems in children	3
Frequent change of residence for poorly defined reasons	3
Anxiety-relieving mechanisms, such as telephone calls inappropriate in time, distance, person, or motive (telephonitis)	2
Outbursts of rage and suicidal gestures while drinking	2
B. Psychological and Attitudinal	
1. Direct effects	
Early:	
When talking freely, makes frequent reference to drinking alcohol, people being "bombed," "stoned," etc., or admits drinking more than peer group	2
Middle:	
Drinking to relieve anger, insomnia, fatigue, depression, social discomfort	2
Late:	
Psychological symptoms consistent with permanent organic brain syndrome (see also Major Criteria, Track I, B)	2
2. Indirect effects	
Early:	
Unexplained changes in family, social, and business relationships; complaints about wife, job, and friends	3
Spouse makes complaints about drinking behavior, reported by patient or spouse	2
Major family disruptions: separation, divorce, threats of divorce	3
Job loss (due to increasing interpersonal difficulties), frequent job changes, financial difficulties	3
Late:	
Overt expression of more regressive defense mechanisms: denial, projection, etc.	3
Resentment, jealousy, paranoid attitudes	3
Symptoms of depression: isolation, crying, suicidal preoccupation	3
Feelings that he is "losing his mind"	2

were incarcerated for public intoxication for three-month periods had a total yearly alcohol intake and a total time available for drinking that may have been less than that of the "normal" drinker. Yet these men reported withdrawal signs and symptoms upon cessation of each drinking spree. Thus, there is in some cases an apparent persistence of the "alcohol addiction memory." The conditions that cause withdrawal signs and symptoms are not as yet fully understood.

Thus, where the practitioner has a patient whose drinking pattern consists of intermittent or recurrent drinking and in whom the appropriate diagnostic criteria are satisfied, the condition should be diagnosed as alcoholism (with the qualification as to pattern added if it seems important).

Alcoholism: Recovered, Arrested, or in Remission

Since alcoholism is relapsing and chronic, there are very few authorities who claim a complete cure. But there are many patients who, after a time of complete sobriety, have reordered their lives in a rehabilitative way and are completely able to perform complex and responsible tasks. There are also a few patients who have returned to "social" drinking or who have infrequent "slips" but who still function as rehabilitated persons.

Although these diagnostic criteria are not devised as a guide to prognosis, it is the opinion of the committee that a history of alcoholism in the past, followed by a significant recovery, should be taken into account as a guide to treatment, employment, and restoration of rights and privileges previously denied because of active alcoholism. Some members of the committee believed that total abstinence would not, in the future, turn out to be an absolute, final necessity for recovery from alcoholism. However, it was agreed that total abstinence, as a measure of recovery, arrest, or remission, was usually more easily measurable, definitive, and generally accepted than a change from "dependency" to "social" drinking. Thus, the committee agreed that the following considerations should determine the diagnosis of recovered, arrested, or remitted alcoholism:

Duration of abstinence
Concurrent active treatment program
Concurrent A.A. attendance with full participation
Concurrent self-administered and professionally guided deterrent medication
Resumption or continuation of work without absenteeism
No traffic violations
No substitution of other drugs

Although the committee did not choose at this time to assign definitive time values for any of these considerations, the recovery or remission gains in its validity with a progressively longer time. For abstinence alone to be the criterion, without other therapeutic activity, there needs to be a longer time period than if abstinence is combined with other criteria.

Alcohol Use

Diagnostic terms that define conditions that fall short of alcoholism are necessary because of the effects of alcohol on behavior. Although the term *alcohol abuse* has wide currency, we prefer *alcohol use*, accompanying this term with a description of effect. This leaves the term "abuse" for such situations as child abuse, animal abuse, or self-abuse, where there is an animate object of the abuse, and does not anthropomorphize alcohol, which, after all, is a chemical (the "neutral spirit"). The term *misuse*, we believe, also carries an unnecessary moral implication.

Alcohol Use with Inebriation

Intoxication may be mild, moderate, or severe, or may lead to coma. Although alcoholics are frequently obviously intoxicated, mere intoxication is not sufficient for the diagnosis of alcoholism. Indeed the physician should be cautious in making a diagnosis of alcohol intoxication on the basis of a staggering gait, slurred speech, other neurological signs, and an odor of alcohol on the breath. In such cases, one must be sure to rule out diabetic acidosis, hypoglycemia, uremia, impending or completed stroke, and other causes of cerebral impairment. An alcohol breath test, determination of blood alcohol level, or serum osmolality measurement may assist in making a diagnosis of alcohol intoxication. A history from the patient and from family members or friends is usually helpful but must in itself be subject to evaluation. Alcohol intoxication must be

thought of in any person in coma; *in addition*, barbiturate and other sedative intoxication must be investigated: cross-dependence and cross-tolerance are common.

Alcohol Use with Pathological Intoxication

In some individuals a small amount of alcohol will evoke violent, aberrant behavior. Pathological intoxication is an idiosyncratic response to alcohol and is separate from alcoholism.

Alcohol Use: Reactive, Secondary, or Symptomatic

Reactive, secondary, or symptomatic alcohol use should be separated from other forms of alcoholism. Alcohol as a psychoactive drug may be used for varying periods of time to mask or alleviate psychiatric symptoms. This may often mimic a prodromal stage of alcoholism and is difficult to differentiate from it. If the other criteria of alcoholism are not present, this diagnosis must be given. A clear relationship between the psychiatric symptom or event must be present; the period of heavy alcohol use should clearly not antedate the precipitating situational event (for example, an object loss). The patient may require treatment as for alcoholism, in addition to treatment for the precipitating psychiatric event; one may be able to confirm the diagnosis only in retrospect.

Alcohol and Anxiety

The effects of alcohol on the rising slope of the absorption curve parallel the four stages of anesthesia, and thus excited or uninhibited behavior may be shown with mild inebriation. But it also has been documented that, with large doses over a prolonged period of time, alcohol produces anxiety. Whether this bimodal effect occurs as a regular result of any amount of alcohol is currently being investigated. The progressive rise of anxiety with continued heavy drinking is responsible for many of the effects listed as minor criteria.

Cross-Dependence

Cross-dependence (or "cross-addiction") may begin iatrogenically or spontaneously with the use of any of the sedative class of drugs, barbiturates, or "minor" tranquilizers in an attempt to control the anxiety generated by heavy alcohol use or in the mistaken impression that pharmacological control of the anxiety will stop the alcohol use. Such cross-dependence is so common that it must be investigated in any person suspected of alcoholism.

In addition, the life-style of persons who seek pharmacological "highs" is associated with heavy alcohol use *pari passu* with other psychoactive chemical materials. Such persons are at risk of alcoholism, and patients being investigated for the diagnosis of alcoholism should also be evaluated for use of these materials.

Treatment programs for the use of other drugs engender a significant proportion of "instant alcoholics" who, having relinquished the other drugs, turn to alcohol and experience an unusually rapid onset of dependency. Thus, patients in this category should also be screened for alcoholism, and attempts should be made to prevent its onset.

Persons at High Risk of Alcoholism

Epidemiological and sociological studies show that the following factors indicate high risk for the development of alcoholism. There is not complete agreement on the extent of risk for each factor.

- A family history of alcoholism, including parents, siblings, grandparents, uncles, and aunts (2).
- A history of teetotalism in the family, particularly where strong moral overtones were present and, most particularly, where the social environment of the patient has changed to associations in which drinking is encouraged or required (2).
- A history of alcoholism or teetotalism in the spouse (2) or the family of the spouse (3).
- Coming from a broken home or home with much parental discord, particularly where the father was absent or rejecting but not punitive (4).
- Being the last child of a large family or in the last half of the sibship in a large family (3).
- Although some cultural groups (for example, the Irish and Scandinavians) have been recorded as having a higher incidence of alcoholism than others (Jews, Chinese, and

Italians) the physician should be aware that alcoholism can occur in people of any cultural derivation (5–7).

- Having female relatives of more than one generation who have had a high incidence of recurrent depressions (8).
- Heavy smoking: heavy drinking is often associated with heavy smoking, but the reverse need not be true (9).

Recording the Diagnosis

If alcoholism as defined above is present, the diagnoses should be stated in this order:

Alcoholism: intermittent use, recurrent use, steady use (early, moderately advanced, far advanced)

Psychiatric diagnosis

Physical diagnosis

If major criteria or a sufficient number of minor criteria are not met, the diagnosis should be:

Suspected alcoholism; psychiatric diagnosis; physical diagnosis

Other diagnoses that can be made:

Alcohol use: reactive, secondary, or symptomatic; psychiatric diagnosis; physical diagnosis

Alcohol use with inebriation

A description of the physical diseases associated with alcoholism and their diagnosis will be the subject of a separate communication.

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APPENDIX 1
Criteria Committee, National Council on Alcoholism

Chairman: Samuel C. Kaim, M.D., Director, Staff for Alcoholism and Related Disorders, Department of Medicine and Surgery, Veterans Administration

Henry Brill, M.D., Director, Pilgrim State Hospital

Luther A. Cloud, M.D., Associate Medical Director, Equitable Life Assurance Society of the United States

David H. Knott, M.D., Ph.D., Director, Alcohol and Drug Dependence Clinic, Tennessee Psychiatric Hospital and Institute

Charles S. Lieber, M.D., Professor of Medicine, Mount Sinai School of Medicine, and Chief, Section of Liver Disease and Nutrition, Bronx Veterans Administration Hospital

William M. McIsaac, M.D., Ph.D., D.Sc., Director, Texas Research Institute of Mental Sciences

Jack H. Mendelson, M.D., Professor of Psychiatry, Harvard Medical School, and Director, Psychiatry Service, Boston City Hospital

James Rankin, M.B., B.S., Director, Institute for the Study of Addiction, Addiction Research Foundation, and Associate Professor of Medicine, University of Toronto

Anthony Reading, M.D., Assistant Director, Psychiatric Liaison Service, Johns Hopkins University School of Medicine

Richard S. Shore, M.D., Director, Bureau of Alcoholism, San Francisco Department of Public Health

Harold N. Willard, M.D., Associate Professor of Internal Medicine, Yale University

Steven J. Wolin, M.D., Clinical Associate, Laboratory of Alcohol Research, National Institute on Alcohol Abuse and Alcoholism (Saint Elizabeths Hospital)

Ex Officio: Irvin E. Hendryson, M.D., Professor of Surgery and Assistant Dean, University of New Mexico School of Medicine

Staff: Frank A. Seixas, M.D., Medical Director, National Council on Alcoholism

APPENDIX 2

Whiskey Equivalents of Consumption of Wine and Beer

Equivalents are based on:
0.8 quart = one-fifth gallon
32 ounces = 1 quart
Whiskey contains 43 percent ethyl alcohol
Fortified wine contains 20 percent ethyl alcohol
Table wine contains 12 percent ethyl alcohol
Beer contains 4 percent ethyl alcohol

Person's weight (pounds)	Whiskey (quarts)	Fortified wine (quarts)	Table wine (quarts)	Beer (quarts)	Beer (12-oz. bottles)
220	1.0	2.0	3.6	11.0	29
200	0.9	1.9	3.2	9.7	26
180	0.8	1.7	2.9	8.6	23
160	0.7	1.5	2.5	7.5	20
140	0.6	1.3	2.2	6.5	17
120	0.5	1.0	1.8	5.4	14

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PROJECT DESIGN

Establishing a central consulting service

In organisations where medical facilities exist, it would be desirable to establish a consulting service as an additional component. The central consulting service can preferably be situated at the industrial medical centre. A consultant psychiatrist from a general hospital may be coopted into the programme. The physician in-charge of the industrial medical centre, the welfare officer or the social worker and a clinical psychologist, along with representatives of the labour unions would form the rest of the team.

The central consulting service would ensure the cooperation of the management and labour unions, educate the work force along with their families, and supervisory personnel. It would also maintain the administrative liaison between other community facilities (e.g., the Halfway Home) and the general hospital, and periodically evaluate the cost effectiveness of the programme.

Identification of the problem drinker

Many of the earlier industrial programmes relied on the signs and symptoms of alcoholism for the identification of the problem drinker. However, training the work supervisors to detect such signs and symptoms have been found to be impractical. Moreover, the supervisors are usually reluctant to label an employee as an alcoholic, and often do not report their findings. In most instances, problem drinking will manifest itself in impaired work performance and absenteeism. Apart from this, simple biochemical evaluations and self rating questionnaires are available for early detection of problem drinking with high reliability and validity.

Referral and documentation

Once the problem drinker is identified, a series of corrective interviews are conducted motivating him to seek help on their own. When the offer is accepted, he may be evaluated by the clinical psychologist, social worker and the physician at the central consulting service, to assess his motivation, factors maintaing the problem drinking and the extent of physical morbidity. He and his family are informed about the method of treatment and their cooperation sought. The welfare officer is approached for necessary assistance regarding leave from the job for detoxification.

Detoxification and management during the early phase

Two to three weeks of admission on an average is required during this phase. Detoxification, initiation of counselling for the individual and his family, identification and treatment of major psychiatric disorders and medical complications are conducted at this stage. At the end of the hospital stay, the individual is either returned to the factory for resuming active duty immediately, or send to the halfway home if prolonged care is required.

Follow up and maintenance of abstinence

It is known that frequent follow ups results in a higher probability of success in abstinence of alcohol. The most efficient follow up may be conducted at the work place. Those who drop out from the follow up may quickly be identified and their families contacted by the social worker. The problem drinkers are to be regularly followed up for at least one year, since the maximum relapses are seen to occur in this time.

The halfway home

A portion of the problem drinkers would have deteriorated sufficiently to warrant additional, more technical and graded care for rehabilitation. They are to be referred to the halfway home after discharge from the hospital, where they may have to stay for three to six months.

Personnel

The medical officer of the industry's medical centre would primarily assist in the integration of the programme in the existing medical service of the industry, and provide supportive medical management from time to time. He would also initiate training of other personnel and health education programme which are integral parts of the project. In course of time the industry's medical team would assimilate sufficient technology to maintain the project on their own.

The clinical psychologist provides the important therapeutic management and gives the continuity to the programme at it's various stages, and at different locations of treatment. Once the project is started the emphasis of the educational and motivational programmes shift to the families of the problem drinkers. In addition, development of documentation systems, counselling the individuals and their families, carrying out follow up of cases and periodic evaluation of the programme are to be dealt with by the psychologist.

The social worker primarily brings about attitudinal changes at the beginning of the programme, through meetings of labour unions and the management, and meetings of family groups. This is essentially a programme of health education. Apart from this the social worker can facilitate the liaison with section supervisors in the identification of cases and maintain liaison with the halfway

home. During follow up he conducts home visits in order to detect and motivate dropouts, and keep track of the public opinion of the programme among the different categories of workers in the industry and their families.

Additional staff are recruited as and when required according to the necessity of the moment and availability of facilities.

CONCLUSION

Employee Assistance Programme for Alcoholism have been able to successfully control alcohol related loss of productivity among the industrial workers. This method also has an inherent advantage in that the population catered for is immobile and thus available for easy identification and follow up. Finally the cost effectiveness is considerable in terms of productivity to the industry, relief of problems to the family, reduced expences related to consumption of alcohol, and management of alcohol related medical problems. However, in most programmes the referral rates tend to be high in the early phase of the operation, when it is easy to identify the most troublesome and chronic problem drinkers, but decline once these employees have been treated.

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Problem drinking in industries varies between 5 to 15 percent, according to various surveys conducted in India [1]. In recent years, professional attention has increasingly been directed to the workspot as a potential locus for identifying the problem drinker. Most problem drinkers have jobs. They can be identified relatively early by the evidence of impaired work performance and other simple tools for early detection [2]. Occupationally oriented programme, offering help rather than dismissal, yield the highest reported rates of successful recovery from problems related to alcohol.

In developed countries, the Employee Assistance Programme for Alcoholism (EAPA) has been introduced, based on recognition of the adverse effects of problem drinking on productivity, social consciousness on the part of the management and specific awareness of the drinking problem within the industry. It has been very sucessful in organisations of large or medium size, employing more than 1000 workers [3]. The work place is viewed not merely as an agency of referral but also as an active force in the rehabilitation process.

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INDUSTRIAL ALCOHOLISM AND LOSS OF PRODUCTIVITY : A STRATEGY FOR INTERVENTION

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(e) two and one half grammes of a preparation, mixture, extract or other material containing not less than one-fifth of one *per cent* of morphine or a salt of morphine or any proportion of diacetylmorphine or an ester of morphine or a salt of an ester of morphine;

(f) 5 grammes of barbitone or a salt of barbitone or a preparation, mixture, extract or other material containing any proportion of barbitone;

(g) 5 grammes of cannabis (*Amended, 46 of 1978, s.5*)

(ga) one half gramme of tetrahydrocannabinol either alone or contained in a preparation, mixture, extract or other material; (*Added, 46 of 1978, s.5*)

(h) 10 tablets or capsules containing any proportion of any dangerous drug,

shall, until the contrary is proved, be presumed to have had such dangerous drug in his possession for the purposes of trafficking therin.

Detention order.

4. (1) Where a person is found guilty of a relevant offence and the court is satisfied that in the circumstances of the case and having regard to his character and previous conduct it is in his interest and the public interest that he should undergo a period of cure and rehabilitation in an addiction treatment centre, the court may, in lieu of imposing any other sentence, order that such person be detained in an addiction treatment centre.

(2) A person in respect of whom a detention order is made shall be detained in an addiction treatment centre for such a period, not less than six months and not more than eighteen months from the date of such order, as the Commissioner may determine, having regard to the health and progress made by such person and the likelihood of his remaining free from addiction to any dangerous drug on his release, and shall then be released.*

(3) Before a detention order is made in respect of any person, the court shall consider a report of the Commissioner on the suitability of such person for cure and rehabilitation and on the availability of places at addiction treatment centres, and if the court has not received such a report it shall, after such person has been found guilty, remand him in the custody of the Commissioner for such period, not exceeding three weeks, as the court thinks necessary to enable such a report to be made.

(4) When a court makes a detention order, it may, where the circumstances of the offence so warrant, order that no conviction shall be recorded.

(5) The Commissioner shall, in his report under subsection (3), inform the court whether or not a detention order has previously been made in respect of the person to whom the report relates.

Supervision order.

5. (1) The Commissioner may order that a person released from an addiction treatment centre shall, for a period of twelve months from the date of his release, be subject to supervision by such organisation or person as he may specify and shall while under such supervision comply with such requirements, including requirements as to medical examination, as he may specify.

(2) The Commissioner may at any time vary or cancel a supervision order.

* The periods in subsection (2) have been reduced to 4 months and 12 months respectively.

British Journal of Addiction 80 (1985) 153–162

Alcoholism and the Nature of Outcome

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Summary

A great deal of previous work has been directed to conceptual and methodological issues relating to descriptions of 'outcome' in studies of career, natural history, and treatment of alcoholism. There is still no consensus even on basic measures of drinking behaviour which would allow comparison between reports, and the 'dynamics' of relationships between variables remains obscure. This paper uses data from a 10–12 year follow-up of 68 interviewed male alcoholics to explore simple and partial correlations between outcome variables, relating both to the total follow-up period and the 12 months prior to interview. In several instances significant correlations are found between outcomes in different domains, but the levels of correlation which are reached suggest that outcome cannot be conceived as a simple unitary dimension, while the pattern of partial correlations begins to reveal the underlying dynamic of relationships. Outcome may need to be conceived not just in terms of end points or scores, but in terms of the processes in which the individual becomes caught up.

Introduction

Research on alcoholism treatment and on the career and natural history of the alcoholic share a fundamental concern with definition and measurement of 'outcome'. But it is now generally recognised that what may once have appeared a relatively straightforward and unitary concept in reality poses many complex questions.

These questions have over recent years been widely debated [1,2] but have not as yet achieved sufficient clarification for different research workers commonly to employ comparable field measures, and secondary analysis therefore poses many difficulties due to the disparate nature of the data which is collated [3,4]. This is not to undervalue the research advances which have been made. But if there are to be larger steps forward (both theoretical and practical) it is urgent that a wider consensus be reached on basic matters relating to the meaning and measurement of outcome. Outcome is a conceptual and methodological issue in its own right, and to continue with studies which explore the significance of predictor variables or the efficacy of different treatments without the nature and measurement of outcome first being better resolved, is to build on insecure foundations.

Turning briefly to some account of those advances on which we can build, it is now generally accepted that rather than employing drinking behaviour as the sole outcome measure, measures of other domains such as social adjustment and mental and physical health should in addition be recorded. [2,5,6]. Measurements of degree of dependence on alcohol have begun to be employed [7,8]. There is a recognition also of the possible disjunction between these different measures, with debate over the extent to which amelioration in drinking is predictive of change in other areas [5,6,9,10]. Early studies often tended to use measures which compounded drinking and social adjustment [11], whereas the recent trend is to keep the basic measures of different elements of outcome distinct and explore their correlations [2,7]. Attention has increasingly been given to refinement in measures of the drinking dimension itself with an acknowledgement that continuous as well as categorical descriptions may have to be explored, and with 'normal drinking' thus a possible outcome [9]. It is evident that the range and complexity of possible drinking outcome patterns cannot be easily be subsumed under such headings as 'drinking' and 'abstinent'. There are some aspects of outcome which can be recorded meaningfully in terms of 'present status' — a cross sectional view of the present. Others may be recorded in terms of a relatively short time-base, while career studies often also seek to integrate data relating to a particular dimension over a long period of time. A relatively constricted 'window of observation' pertaining only in the recent past can have advantages relating to accuracy of recall but a restricted

sampling of fluctuating behaviour (eg. drinking behaviour) can on the other hand be too narrow and potentially misleading [7,12].

It should also be admitted that the word 'outcome' itself in some ways invites misunderstanding. In the classic sense the 'outcome' of a fixed time-base clinical trial has meaning, but in studies of career and natural history 'outcome' is never complete until the subject's eventual death — we are otherwise observing only an arbitrary segment of a continuously unfolding story.

On the basis of previous work we thus have, as it were, a map which outlines many of the questions which have to be further explored, but the details of the mapping are still in many areas vague. Furthermore, the current tendency to deploy multiple outcome measures though representing an important advance, is still really evidence only of a catch-all empiricism — we have not so far been able at all closely to approach a dynamic understanding of the way of any relationship between different aspects of outcome or to disentangle the nature of intervening processes. Our map lacks the roads and railways.

A preliminary report on the characteristics and overall long-term outcome of the cohort we will discuss in this paper has been published elsewhere [8]. Here our aim is not to give an 'outcome report' in the conventional sense, but to use these data to illustrate and examine some of the basic issues which are posed by this type of research. Conceptually we are particularly interested in the significance of degree of dependence on alcohol [13] as mediator between drinking behaviour and other variables. Whether our findings would generalise to other patient groups is a question only to be resolved by further exploration.

Method

Sample

The present study originated in 1968–70 when the Addiction Research Unit conducted a controlled trial in which 99 married, male alcoholics were randomised between 'treatment' and 'advice' only. These patients were originally followed-up 12 months [14] and 24 months [15] after their intake to the study. Between March 1980 and April 1982 efforts were made to contact the surviving members of this cohort. It is to data from this 10–12-year follow-up that this paper refers. Of the original 99 subjects, 18 had died and 68 of the remaining 81 were interviewed.

Overall strategy of the enquiry

Data were collected by a semi-structured enquiry, and by self-completion questionnaires. Two distinct periods were examined seperately — firstly the 12 months prior to interview and secondly the entire follow-up period of 10–12 years (mean 11.3 years). These time periods will subsequently be referred to as the '12 month' and the 'entire follow-up' periods. Guidelines were developed for scoring all individual items where relevant. Information relating to illnesses, hospital admission and criminal offences were obtained from extensive record searches. Data was also obtained wherever possible independently from collaterals, but this information will not be discussed in the present paper.

Data relating to the 12-month period

1 *Drink behaviour*

Three measures of drinking behaviour were used.

(a) *Categorisation by frequency and quantity.* The drinking behaviour of each individual was assessed by determining the number of weeks out of 52 the patient spent in any of four categories. The highest level of drinking on any day of the given week decided the category to which the entire week was assigned. The four categories of drinking were defined thus:-

(i) Weeks containing any day on which the patient exceeded 10 pints of beer or its equivalent (200gm absolute alcohol).

(ii) Weeks containing any day where 5 pints of beer or its equivalent (100gm) was exceeded, but not more than 10 pints (200gm).

(iii) Weeks containing any day where up to 5 pints of beer of its equivalent (100gm) consumed, but this level was not exceeded.

(iv) Weeks of total abstinence.

(b) *Summary drinking score (SDS).* The information categorised above was condensed into a continuous score. Points were awarded as follows:- 0 was given for each abstinent week out of the 52. 1 point for each week at the ⩽ 5 pints of beer (or equivalent) level.

2 points for each week at the >5 ⩽10 pints of beer (or equivalent) level.

3 points for each week at the >10 pints of beer (or equivalent) level.

(c) *Alternative drinking category.* Subjects were tricotomised into a 'Good' group, an 'Equivocal' group and a 'Bad' group. This summary classification was originally employed by Orford and Edwards [14] and is included here for the purpose of comparison. In essence, outcome was considered 'Good' if the patient reported five or fewer weeks containing any episode of 200 gm (10 pints) intake or more. Outcome was considered 'Bad' if the patient had 26 or more weeks containing any 100gm (or more) per day drinking (five pints). Between these two extremes all other cases were considered 'Equivocal'.

2 *'Troubles' associated with alcohol abuse*

This was a ten-point scale developed in the original study [14]. It consisted of the following equally weighted items relating to adverse experiences over the previous 12-month period:-

Suffered morning shakes
Morning drinking
Inability to stop drinking till drunk
Vomited or had nausea in the mornings
Passed out when drinking
Experienced alcoholic hallucinations
Lost time (even half-a-day) from work
Drank secretly
Pawned own or wife's possessions or household articles
Got family into debt of £50 or more.

These ten items fall into two categories. The first six items may be described as 'dependence' symptomatology while the remaining four are social problems experienced as a result of alcohol abuse.

3 *Social adjustment*

A four-item scale gave points related to marriage, criminality, employment and accommodation. For each of the following one point was given (i) if the patient was married for the last 12 months regardless of whether or not it was the same marriage as in 1968/70; (ii) if he had not been in prison during the last 12 months; (iii) if he had been employed for at least 11 months of the previous twelve; (iv) if he currently lived in his own home (either owned or council rented) as opposed to living in lodgings, a hostel or being of no fixed abode.

4 *Mental health adjustment*

This comprised five items. A point was scored respectively for absence of depression, absence of drug problems and absence of suicide attempt, all within the last 12 months. A point was scored unless the patient had a Purpose-in-Life (PIL) score more than 1 standard deviation below the 'normal' range [16] and similarly unless he had an EPI score on the N sub-scale greater than 1 standard deviation above 'normal' [17].

5 *Socio-medico assessment*

Addition of the previous two scales created a nine-point summary of the individual's socio-medical status. A point was scored for each of the following:-

No imprisonment in the last 12 months
No attempted suicide in the last 12 months
No problems with drugs in the last 12 months
Married for last 12 months (minimum)
Had worked 11 months out of the preceding 12
No evidence of depression in last 12 months
Has an N score on the EPI less than 1 sd. above normal
Lives in his own or rented accommodation
Has a Purpose-in-Life score greater than 1 sd. below normal

Entire Follow-Up Period

1 *Drinking behaviour*

For each year (or part year) of the follow-up period the number of months spent in each of the three categories, Abstinent, Social Drinking and Troubled Drinking, was recorded. Social drinking was defined as untroubled drinking for at least three consecutive months and not exceeding five pints on any drinking day.

Abstinence was the time spent in total sobriety. 'Troubled' subsumed any drinking outside these two categories.

2 *The 'Total Drinking Days' (TDD)*

For each year or part year the number of months spent in troubled drinking were calculated and averaged. Each individual could thus receive a score between 0 and 12. A more detailed examination of these data will be given in a forthcoming paper.

3 *Mental health*

This was assessed by a three-item score relating to any episode of depression, suicide attempt or drug abuse. As for the twelve-month score a point was scored for the absence of each of these problems.

4 *Physical health*

The three items contributing to this category were (i) reported brain damage, (ii) peptic ulcer, (iii) any major chronic or life threatening diseases including cirrhosis. This information largely derived from hospital notes, rather than interviewer report.

Table 1. Distribution of subjects across weeks within each alcohol consumption category (N = 68). (For definition of categories, see text page 154. Columns sum to ≈ 100% with rounding errors. Percentages in brackets.)

Weeks out of 52 in defined category		Greater than 10 pints	Greater than 5 pints but less than 10 pints	5 pints or less	Abstinence
0	N	37	47	49	19
	%	(54)	(69)	(72)	(28)
1–13	N	10	13	7	14
	%	(15)	(19)	(10)	(21)
14–26	N	3	0	3	6
	%	(4)	0	(4)	(9)
27–39	N	3	0	1	4
	%	(4)	0	(2)	(6)
40–52	N	15	8	8	25
	%	(22)	(12)	(12)	(36)

5 *Severity of Alcohol Dependence Questionnaire (SADQ)*
This self-completion questionnaire was used here to measure the worst ever experience of psycho-physiological dependence during the entire follow-up period [18].

Results

The N of the tables listed below varies from 59–68 due to missing data.

1 *Twelve Month Follow-up: Raw Scores*
(a) *Drinking behaviour categorised by weeks spent in each of four categories.* For each of the four drinking levels the distribution of subjects by number of weeks out of the previous 52 spent within the particular level is given in Table 1.
(b) *Summary drinking score (SDS).* Results by this summarising technique are present in Table 2. Subjects appear to split fairly evenly within the four bands, with 25 per cent scoring zero.
(c) *Three-way drinking categorisation.* Forty per cent of patients had a 'Good' outcome, 13 per cent an 'Equivocal' outcome and 47 per cent a 'Bad' outcome by this definition. The correlation between categorisation by this approach and the SDS was 0.9 ($p < .001$, one-tailed).
(d) *Troubles associated with drinking.* The rates of endorsement for the individual items of this scale are given in Table 3. When the individual scale scores were examined the distribution was as follows: 48 per cent of patients (32) scored 0 Troubles; 23 per cent scored 1–3 Troubles; 29 per cent (19) four or more Troubles. The results suggest that upwards of 50 per cent of subjects had experienced one or more Troubles during the previous 12 months. For those subjects experiencing any Trouble at all, the mean count was 4.2 and the median 4.
(e) *The adjustment score.* For Social Adjustment and Mental Health rates of endorsement for individual items are shown in Table 4. The distribution for Social Adjustment scores was 0, 3 per cent; 1, 16.4 per cent; 2, 25.4 per cent; 3, 32.8 per cent; 4, 22.4 per cent (n = 67). As regards mental health, Table 4 suggests that only small percentages of subjects had shown overt evidence of impairment as indicated by depression, drug problems or suicide attempt while larger numbers showed maladjustment as judged by scores on the EPI N Score and Purpose-in-Life inventory. Complete data on Mental Health Adjustment was available on only 59 subjects and corrected scores were therefore calculated when only one item was missing (n = 65) the missing item being replaced by the mean score: the distribution was then 1, 4.6 per cent; 2, 10.8 per cent; 3, 18.5 per cent; 4, 32.3 per cent; 5, 33.8 per cent. Distribution for the summary Psychosocial Adjustment Scale is given in Table 5, which suggests a skewing toward better outcome with relatively few subjects (13.2%) scoring 4 or less on this 10-point scale.

Table 2. Drinking for 12 months prior to interview: Distribution of subjects within categories defined by Summary Drinking Score (N = 68).

Score	%
0	25
1–52	25
53–104	22
105–156	28

Table 3. Troubles with drinking experienced during the 12 months prior to interview (n = 67). The endorsement of the individual items comprising the scale.

Item	N	%
1 Morning shakes	25	37
2 Morning drinking	23	34
3 Inability to stop	23	35
4 Vomited or morning nausea	20	30
5 Passed out drinking	15	23
6 Lost time (even ½ day) from work	13	19
7 Drank secretly	11	16
8 Experienced alcoholic hallucinations	9	13
9 Got family into debt	6	9
10 Pawned own or wife's possessions	3	4

Table 4. Twelve months prior to interview: Social and Mental Adjustment. Percentage endorsement from individual items.

Scale	Item	% Endorsement
Social Adjustment	Married for last 12 months	70.6
N = 68	Not been imprisoned in last 12 mths	95.6
	Employed at least 11/12	51.5
	Own (or Council rented) home	35.3
Mental Health	No depression in last 12 mths	93.8
N = 65	No problems with drugs in last 12 mths	89.2
	No suicide attempts in last 12 mths	86.2
	'Normal' P.I.L. score	44.6
	'Normal' EPI score (N)	66.2

Table 5. Twelve months prior to interview: distribution of patients by Psycho-Social Adjustment Scores
Summation of scores on mental health and social adjustment scales (N = 68)

Score	N	%
0	0	0
1–2	1	1.5
3–4	8	11.7
5–6	16	23.5
7–8	23	33.9
9–10	20	29.4

2 *Twelve-month Period: Correlation Between Measures*
Table 6 shows the correlation matrix for the various measures of outcome discussed above: in this analysis we have employed only the one drinking measure (SDS) because it correlated highly with the other drinking measures. This table shows that consumption of alcohol, as expressed by the Summary Drinking Score, correlates significantly with 'Troubles' (.55) and with social functioning (−.30) over the same 12-month period. The correlation between drinking behaviour and twelve-month Mental Health Adjustment does not reach significance but there is a significant relationship between the 'Trouble' scale and impaired mental health (—.35) and between Troubles and impaired Social Adjustment (—.41). Mental health and social adjustment are significantly correlated.

Table 6. Twelve month follow-up: simple correlations between measures

	SDS	Troubles	Mental Health	Social Adjustment
SDS				
'Troubles'	*** 0.55			
Mental health	−0.16 N.S.	−0.35**		
Social adjustment	** −0.30	−0.41***	0.45***	

*** indicates $p < 0.001$; ** $p < 0.01$; * $p < 0.05$.
SDS is Summary Drinking Score.

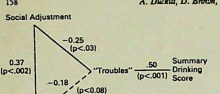

Figure 1. Twelve month outcome: partial correlations between four measures. One-tailed significance levels are quoted.

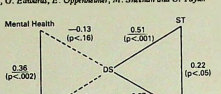

Figure 2. Twelve month outcome: partial correlations with Trouble Score split into Dependence Score (DS) and Social Troubles (ST). One-tailed significance levels are quoted.

In order further to understand these relationships a partial correlation analysis was carried out. The relationship between the four outcomes measures can then be schematically represented (Fig. 1). The analysis involves taking the partial correlation of each pair of variables controlling for the effect of all other variables, thus showing the direct relationship between the given pair. [21,22].

This diagram reveals the position of 'Troubles' as a mediating variable. Thus the relationship between Social Adjustment or Mental Health on the one hand and drinking behaviour on the other, is not a direct relationship as suggested by the simple correlation analysis, but a relationship that only exists through the variable "Troubles". The partial correlations for these two variables with SDS are −.07 and .03 respectively and are non-significant. They are omitted from the diagram. The lack of significant relationship between drinking and Mental Health that was seen in the simple correlation is again borne out in the partial correlation analysis. Thus the partial correlation analysis shows more clearly the subtle relationship of the links between distinct areas of functioning that was not apparent in the simple correlation. It is not the level of drinking *per se* which impinges adversely on adjustment, but drinking that leads to 'troubles with drinking' which thus impinges.

We do not have SADQ scores specifically for the twelve-month period but only 'worst ever' scores related to the whole follow-up. However among the items which comprise the Troubles score there are six items which may be taken as related to the dependence concept (see p.156). When the partial correlations are run with these six items as a Dependence Score (DS) and the remaining four items put into a Social Troubles (ST) score, the relationships are then as in Figure 2 with DS in a mediating position although its relationships with Mental Health and Social Adjustment do not reach significance.

3 *Entire Follow-up Period: Raw Scores*

(a) *Mental health*. Endorsements for each of the three items in the upper part of Table 7 relate to mental health over the entire period the of the follow-up. Roughly one-third of subjects (with overlap) had experienced difficulties in each one of the three areas of depression, drug abuse or suicide attempt, while one-third had experienced no difficulties of this type.

(b) *Physical health*. The lower section of Table 7 shows that 13 per cent of subjects had sustained brain damage, 13 per cent had contracted a peptic ulcer and 22 per cent had experienced a variety of other

Table 7. Entire Follow-Up Period: Mental and Physical Health.

	Item	% Endorsement
Mental Health	No depression in whole F.U. period	68.7
N = 67	No suicide attempt in whole F.U. period	67.2
	No drug abuse in whole F.U. period	68.7
Physical Health	No brain damage	86.6
N = 67	No peptic ulcer during F.U.	86.6
	No major health disorders	77.6

Table 8. Entire follow-up period: simple correlations between measures. Notation for significance as in Table 7. TDD is Total Drinking Data

	TDD	Mental health	Physical health	SADQ
TDD				
Mental health	0.06 N.S.			
Physical health	−0.07 N.S.	0.09 N.S.		
SADQ	−0.28*	−0.39***	0.01 N.S.	

*** indicates $p < 0.001$; ** $p < 0.01$; * $p < 0.05$ on a one-tailed test.

major illnesses during the entire follow-up period. However, 61 per cent had experienced none of these health problems.

4 *Entire Follow-up Period: Correlation Between Measures*

The simple correlations computed for the data on the entire follow-up period reveal that only the dependence measures (the SADQ) correlates with any of the other measures (Table 8). The SADQ shows a significant relationship with drinking behaviour summated over the follow-up period (the Total Drinking Data or TDD)*, and with Mental Health Adjustment for the same period. The physical health of this sample on the available measure appears to have no relationship with drinking or any other variable, but we are of course dealing with the physical health only of the survivors of the original sample.

The data was then examined using the same partial correlation technique described above, and again a

* Note 1: This relationship holds even when the QF element in the SADQ is dropped out. The relationship therefore is not explained by an overlap of items.

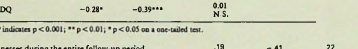

Figure 3. Entire follow-up period: partial correlations between four measures. One-tailed significance levels are quoted.

more illuminating picture of the relationship between variables emerges (Fig. 3).

The partial correlations in this case describes a simple chain. The link between drinking and dependence (SADQ) is weak but still apparent. The links between dependence, and impaired mental health is highly significant, and the relationship between mental and physical health just failed to reach significance.

5 *Correlations Between Measures for 12-Month and Entire Follow-Up*

Finally, the correlations between the 12-month outcome variables and the entire follow-up period variables were calculated (Table 9). Five simple correlations attained significance ($p > .001$) including

Table 9. Pearson correlation matrix showing the 12 month data against the entire 10 year data

12 Month Data	10 Year Data			
	Mental Health	Drinking Data	Physical Health	SADQ
Mental Health	0.52 (p < 0.000)	−0.07 N.S. (0.28)	0.08 N.S. (0.26)	−0.39 (p<.001)
Social Adjustment	0.38 (p < 0.001)	−0.35 (p < 0.002)	0.11 N.S. (0.19)	−0.17 (p<.09)
SDS	0.01 N.S. (0.47)	0.74 (p < 0.001)	0.11 N.S. (0.19)	−0.31 (p<.005)
Troubles	−0.19 (p < 0.06)	0.43 (p < 0.001)	0.06 N.S. (0.33)	0.26 (p<.02)

the correlations between long-term mental health and 12-month mental health .52; long-term mental health and 12-month social adjustment .38 drinking over the entire follow-up and drinking over the last 12 months (.67).

Discussion

The results section of this paper might be regarded as a sort of 'statistical essay' directed to an examination of the relationship between outcome variables. With such an essay it is proper to remember the possible shortcomings in the quality of the data. There must undoubtedly be a certain amount of 'noise in the system'. It could comfortingly be argued that for any significant relationships to be perceived through this 'noise' is evidence of their inherent robustness, but on the other hand there is a possibility that the correlations may be inflated by the individual who is willing to admit one socially undesirable fact being willing also to admit another. The relative crudeness of some of the scales we have employed should also be noted. For instance, our measures of drinking are very much 'summary' variables. They may fail to discriminate important differences in the *patterning* of drinking — a heavy weekend drinker would not be distinguished from a daily drinker and this may have implications for subsequent correlations with degrees of dependence. For the total drinking data as we focus on the amount of time spent drinking (rather than quantity consumed) on any occasion the effect would be to depress the score of the 'binge' drinker which would have the effect of slightly weakening the correlation between dependence and TDD (total drinking data) — if they are as heavily dependent as the consistent consumers. The correlation may be of a lower observed magnitude than perhaps it should be. It seems wise therefore to interpret these results with caution, and see them as pointing toward useful leads for further work rather than at this stage to firm conclusions.

Within this explanatory perspective, this discussion will focus on three issues — the measurement of drinking behaviour, the distinctiveness of and simple correlations between multiple outcome variables, and finally a consideration of the hints which emerge as to the complex underlying relationships between these variables.

The Measurement of Drinking Behaviour

Turning first to a consideration of 12-month drinking outcome, the data given in Table 1 make it apparent that any simple categorisation of outcome must be imposed on a very considerable real-life diversity of drinking patterns — the real picture is of very varied apportionment of time between different drinking levels. For instance, in relation to the heaviest drinking level 54 per cent of subjects spent no time at all in this band while 22 per cent spent 40–52 weeks at this level; the remaining 23 per cent were however spending some time in this category. The distributions for the intermediate levels of drinking (>5≤10 and ≤5 pint equivalents) suggests that drinking at these levels can occur as the dominant individual mode but that such behaviour is rather difficult to sustain, with only 12 per cent of subjects reporting 40–52 weeks at these levels. The distribution for abstinence again shows considerable variation, with 28 per cent of subjects spending no time in this category, 36 per cent spending 40–52 weeks at this level and 36 per cent spending some but lesser time abstinent.

The overall conclusion which thus emerges is that to conceptualise the integration of drinking outcome simply in terms of 'abstinent' or 'drinking' would be very inadequate. For analytical purposes it is necessary to develop some sort of summary descriptions, but the essentially empirical nature of present solutions to this problem must be admitted — we are segmenting drinking behaviour in terms of convenient cutting-points rather than along the boundaries of any naturally occurring typology (if such exists). The Summary Drinking Score (SDS) employed in this presentation appears though to provide a useful continuous measure; that categorisation by levels on this score should correlate highly with the Good/Equivocal/Bad (see p.154), might be seen as supporting its validity.

As already mentioned fuller discussion of the data for drinking behaviour over the entire follow-up will be given elsewhere. Here though the high correlation (.74) between the measures for drinking behaviour relating to the entire and twelve-month periods should be noted. This suggests a considerable stability in group behaviour over time, but with only 45 per cent of variance accounted for it is also a reminder that a twelve-month 'window' would not at all completely reflect behaviour over the longer period. This conclusion is in broad agreement with recent major outcome studies [2,7,12].

Conceptually the measures which we employed to describe aspects of long-term outcome reflect between them a number of different approaches to integration of outcome over time. The TDD score is an example of a deliberate crude attempt to summarise fluctuating behaviour and data which could not otherwise be easily handled statistically. Measures of mental and physical health essentially tot-up the occurrence of events, while the 'worst ever' SADQ score reflects 'integration' in terms of maximal experience of a given variable over time.

Similar diversity can be found in the basis of measurement over the twelve month period. When looking at patterns of correlation the fact that we are employing conceptually rather varied approaches to integration should be born in mind.

Simple Correlations between Multiple Outcome Measures

The results at several points confirm the existence of significant correlations between outcome variables, as regards the 12-month period (Table 7). The relationships which are displayed are in accord with what would intuitively be expected — the positive correlation for instance between SDS and Troubles and the negative correlation between SDS and Social Adjustment. At this level of analysis it would certainly not be possible to maintain that the way in which patients drink is unrelated to what otherwise happens to them but the degree to which variables are independent also deserves emphasis: there are evidently different dimensions of outcome, although they may correlate. Furthermore, SDS does not correlate significantly with Mental Health. The inadequacy which would result from employing only one dimension is underlined, and the conclusion to be drawn from our results are in this respect much in accord with the findings of previous investigators [2,5,6].

The picture which emerges for the entire period (Table 8) is that the drinking measure (TDD) agains fails to correlate significantly with the measure of Mental Health, or in this instance Physical Health — a meaningful Social Adjustment scale could not be constructed for this longer time period.

The Underlying Relationships Between Variables

In the introduction to this paper we referred to our present understanding of 'outcome' in terms of analogy to a map which is still lacking much detail on lines of communication. In Figures 1,2 and 3 we have perhaps some very tentative indication as to how the exercise in mapping might be further approached — outlines no doubt as indistinct as any early exercise in cartography. Whatever the shortcomings in detail the important conclusion may though be that there is some sort of intelligible dynamic to be unravelled in these relationships. Figure 1 for instance raises the question why drinking itself (SDS) does not bear directly on Mental Health and Social Adjustment, but only through the mediating relationship with the conglomerate of Troubles. Further analysis (Fig. 2) hints that a more central position in the network of relationships may be held by dependence (DS) but give the imperfect and post-hoc nature of the dependence measure which is being employed the conclusion can only be very tentative. Turning to the data for the entire period (Fig. 3) there is a suggestion that a dependence measure (SADQ) may hold a mediating position between drinking behaviour and adverse consequences. There is congruence between the shorter and longer term findings.

The Way Forward

As stressed at the beginning of this paper any further advance in the understanding of outcome must be highly dependent on design of a reliable and valid basic set of measures, and preferably such as to commend themselves very generally to research workers and allow more comparability between studies.

Equipped with such tools there might be some possibility of taking further the sort of 'mapping' which can begin to show the underlying relationship between variables. A true understanding of the nature of outcome is probably still quite considerably beyond our grasp. A categorisation of outcome which is close to the natural events may have to approach the unravelling of diverse *processes* of outcome — rather than dealing only in artificially constructed scores. For example, the person who is heavily dependent, or who has suffered severe impairment in some sector of adjustment (or who has sustained multiple disadvantages, and be caught up in a *process* which can itself be conceived as an *outcome*. In these terms outcome would be categorised not just on the basis of empirical scores but also in the terms of the dynamic career path along which the individual is moving [19]. We need further to delineate the nature and characteristics of these 'outcome pathways' and capture the sense of movement.

Appendix

Shows the distribution of Psycho-Medical scores for the entire follow-up period.

	Score	%	N
Mental Health	0	3	
	1	23.9	
	2	38.8	
	3	34.3	N = 67
Physical Health	0	0	
	1	10.4	
	2	28.4	
	3	61.2	N = 67

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British Journal of Addiction 80 (1985), 163–171

Drug Use and Religious Affiliation, Feelings and Behaviour

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Summary

The present study examined the relationship between religious affiliation, intensity of religious feelings, frequency of church attendance on the one hand, and on the other, drug use among a sample of adolescent students (N = 2,066). Six drug-use measures were employed: alcohol use; cannabis use; non-medical and medical drug use; hallucinogenic use; and polydrug use. The findings indicate that, religious-affiliation of students was insignificantly related to drug use. The only exception to this rule was for alcohol use, in which case non-affiliated students used less frequently than did Protestant or Roman Catholic students. Church attendance exhibited a stronger negative effect on drug use than did religiosity; however, the effect of the latter had greater impact among females than among males. Overall, the impact of both variables increased as the drug examined moved towards the upper end of the licit-illicit drug continuum. Finally, many of the results varied according to students' gender and age.

Introduction

It seems intuitive that an individual's religious beliefs and behaviours serve as one of many mechanisms of social control. Indeed, social-learning theory would postulate this to be the case; that is, connections to conventional institutions, such as the church, should provide exposure to and reinforcement of normative behaviour [1]. This hypothesis, much to the chagrin of many, was not confirmed by Hirschi and Stark [2]. They found no relationship between frequency of church attendance and delinquent behaviour among a sample of adolescents. This finding, in violation of conventional wisdom, kindled further research in the area. Burkett and White [3] later confirmed Hirschi and Stark's findings; however, they suggested that religion may differentially affect the various components of delinquent behaviour. In particular, they found a moderately strong negative relationship between church attendance and the use of alcohol and marijuana. This differential effect has been confirmed more recently [4].

Most studies indicate that religious participation measured by frequency of church attendance is negatively related to both alcohol and marijuana use [3–7]. The exceptions to this rule are Kandel *et al.* [8] and Kaue and Patterson [9]. They found no relationship and a positive relationship, respectively. Other studies indicate that religious beliefs, in contrast to affiliation, are negatively related to drug use [5, 7, 10].

To date, several of these investigations have restricted their examination to alcohol use [5, 10]. Others, in addition, have examined marijuana use [6, 7, 11]. Few studies, however, have considered the impact of religious factors on other substance use, whether it be illicit use or use for medical purposes. In particular, one would expect that proscriptions against substances used for medical purposes should be less related to religious factors than the case of illicit-type drugs. Yet, few studies have entertained this hypothesis.

Moreover, despite the complexity and the number of confounding factors, only a handful of the studies employed multivariate techniques [6, 5, 10]. Certainly sophisticated multivariate techniques, in and of themselves, are not necessarily superior, for in the absence of theory results can become uninterpretable. At the same time, however, to disregard multiple independent variables, in which theory dictates or suggests a relationship, is also questionable. One of the factors occasionally omitted from analysis is gender, an especially important variable if one suspects differential socialization processes. Elifson *et al.* [4] for instance, found differential gender effects in explaining delinquency. They hypothesized that closeness to mother plays a major role in this difference and suggested that their results support Bardwick and Douvan [12], who contend that females are more likely to become strongly attached to

* The views expressed in this publication are those of the authors and do not necessarily reflect those of the Addiction Research Foundation.

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British Journal of Addiction (1985), 192, 355
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International Review Series: Alcohol and Alcohol Problems Research
6. India

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Summary

Indian society, which was once a model for abstinence is gradually becoming modernised. As a result of this, synthetic alcoholic beverage production and consumption have increased. However, if one surveys through current research activities from both a medical and sociological viewpoint a very dismal picture emerges. With the exception of the prohibition movement, very few scientific studies have been undertaken. On reviewing the research policies of Government and funding agencies, no consistent theme emerges. Training programmes are conspicuous by their absence, as are treatment facilities. Similarly, work on alcohol in the voluntary sector has a long history focused primarily on prohibition. However, in the 1980s some positive steps are being considered. These include the establishment of the 'Working Group on Alcohol and Drugs' and the policy to establish 'Advanced Centres'.

Introduction

The use of alcoholic beverages is not unknown to India, though both their production and consumption have been increasing rapidly since Independence [1]. This is in addition to illicit distillation which is carried on as a parallel economy within the country and whose amount has been calculated as almost being equal to that of legal production [2]. However, if one looks at the historical trends in research, a very dismal picture emerges. Briefly stated, in an historical perspective, India has had no mainstreams of ideas either in medical or sociological research on alcoholism.[3] The historical trend during this period, if any, is reflected in the preoccupation with prohibition policies, which were interwoven within the freedom movement. These were finally enshrined as a directive principle in the Constitution of India, Article 47, which lays down broad guidelines in terms of how and what the State should do, so as to reduce and ultimately eradicate the use of alcohol and other intoxicant drugs.

Since Independence the issue has cropped up time and again leading to the appointment of various commissions. It was envisaged that these look at the reasons why prohibition was not enforced and impediments, if any, to its implementation. The most prominent among such commissions was the Tek Chand Inquiry team on prohibition [4] appointed by the Planning Commission. It submitted a voluminous report, covering each State of the Federal Republic, identifying the type of beverages that were being used, both legal and illegal. It also took into account much hearsay evidence on the alleged adverse social and public health issues in arriving at its recommendations. However, it neither commissioned any research nor identified any areas in which future research even with a broad framework of social and public health policy should be carried out. It piously stated that alcohol consumption had still not reached alarming proportions, that the local taxes were an impediment to enforcing total prohibition, and that these could be overcome by tapping alternative sources for state financial resources. It also suggested that in those states which were finding enforcement difficult due to economic reasons, 50 per cent of the loss in revenue would be compensated by the federal government.

However, except in two states where prohibition was effective right from Independence, this thread was not picked up by the state governments. The issue remained alive until 1976, when it was still a part of the stated government policy, but then desapperated from political commitment, following the brief experience of the Janata Government between 1977–80. This government had, for the last time, at a federal level, indicated its intention to introduce nationwide prohibition within five years by progressively and systematically reducing sales outlets. Since the government did not last its stipulated 5 years due

Table 1. Selected studies in general population on alcohol and other drugs

Sr. No	Investigator(s)	Area of study and year	Type of study	Sample size	Sampling procedure	Funding status	Highlights
1.	Dube and Hasda[3]	Agra (1969	Mental morbidity	16,725	—	F	Prevalence of mental illness among the adult population was assessed. Information about alcohol and drug use was obtained.
2.	Deb and Jindal[6]	Punjab (1973	General population survey	1,251 above 15 yrs	Stratified random sampling of two villages	N.F.	Attempt to correlate alcohol consumption with socio-economic parameters especially impact of green revolution.
3.	Singh and Lal[7]	Punjab (1976–77)	General population survey	6,999 above 10 yrs	Random sampling four villages	N.F.	Assessed prevalence and patterns of alcohol and drugs and socio-demographic correlates.
4.	Mohan *et al*.[8]	Border districts of Punjab (1975–77	General population survey	3,600 above 15 yrs	Random sampling 24 villages	F	Described in detail, prevalence and pattern of alcohol, narcotics and other drugs and also studied characteristics of drug users in depth.
5.	Sethi and Trivedi[9]	Lucknow (1979	General population survey	2,415 above 10 years	Random sampling 8 villages	N.F.	Alcohol was the commonest drug followed by cannabis "drug culture" into rural areas also.
6.	Verma *et al*.[10]	Chandigarh-Punjab (1979	General population survey	1,031 above years	Random sampling rural semi-urban and urban areas	N.K.	Difference in prevalence rate of alcohol consumption in urban and rural population, increased alcohol use was associated with extra farm income in rural areas.
7.	Mohan *et al*.[11]	Ajmer District Rajasthan (1979–80	General population survey	4,670 above 15 yrs	Random sampling of 24 villages	F	Assessed drug use in a traditional community and studied associated pattern Psycho-social sequelae on W.H.O
8.	Mohan *et al*.[12]	Bangalore, Delhi, Ranchi & Dibrugarh (1981–85	General population survey	14,000 above 15 years	Purposive sampling in rural, urban, tribal and industrial population	F	Basically an intervention study on alcohol and tobacco. Assessed prevalence and pattern of alcohol, tobacco and other drugs also.

N.K. — Not known F — Funded N.F. — Not funded

to political dissension within it, the policy could not be implemented. It still remains a point of speculation as to whether these policies could have been implemented had the Government served its full term of office. In retrospect it is fairly obvious that governmental concern about reduced availability, or policy development carried very little conviction with the intelligentsia economic planners and health functionaries.

The present scene

(a) *Funding agencies*

In India the major national funding agencies are:

(i) Indian Council of Social Science Research (ICSSR)
(ii) Indian Council of Medical Research (ICMR)
(iii) Indian National Science Academy (INSA)
(iv) Council for Scientific and Industrial Research (CSIR)
(v) Indian Agriculture Research Institutes

These funding agencies support research on an *ad hoc* basis in their respective areas. Some of them also have established specific centres for research under their control in identified areas of national importance. The ICMR under it has many National Institutes (e.g. the National Institute on Nutrition) and Advanced Centres (e.g. the Advanced Centre in Biological Psychiatry). The CSIR has similarly developed a string of laboratories to carry out long-term research. The INSA supports basic research in all fields including medicine. More recently the Government of India has also constituted a Department of Science and Technology (DST) to supervise, oversee and co-ordinate research in space, oceanography and other frontier areas. In all the above funding agencies, *ad hoc* projects are sanctioned based on approval by peer group review and in addition, research is commissioned on a long-term basis or for specific problems.

Unfortunately if the research policies of these funding agencies are reviewed, it becomes evident that they have had no coherent research direction. The ICMR and the ICSSR, should have developed joint research policies and programmes. However, this was not implemented, as it was not considered a priority area with immediate social and public health consequences. The only ray of hope is the 'Working Group on Alcohol and Drug Dependence,' which the Government has now constituted to formulate long term research activities under the Central Prohibition Committee, which still has to give its recommendations. It is hoped that this group, being multidisciplinary, will make formal recommendations regarding continuous research on policy planning, social and public health aspects and would provide requisite funding. The earlier review on sociological aspects of alcohol research refers only to prohibition [4].

(b) *Research activities within the present framework*

Proposals for *ad hoc* grants in alcohol research have originated mainly in the late seventies and early eighties. Most of the activity has been confined to the post-doctoral thesis work in departments of psychiatry across four major centres of training and some departments of sociology in other universities. Those have mostly been surveys of a specific target group, either in terms of psychiatric morbidity or with regard to drug abuse in general, and where alcohol has been included as one of the incidental drugs. Only two studies (one post-doctoral thesis and one project specifically funded for research on alcohol) have been carried out over the past 37 years. The research publications in the area have been included in the bibliography and the funded studies summarised in Tables 1 and 2. Obviously these studies do not in any way reflect the national picture or a co-ordinated effort. They are listed, as they may mark the beginning of sustained interest in this area in time to come.

Work on alcohol in the voluntary sector has had a long and varied history. The All India Prohibition Council (AIPIC) is the longest active organisation in this field. The origin of the group had a lot in common with the philosophies of the political movement, and some of its activities were aimed at enforcing prohibition and increasing abstinence through picketing. However, research was never one of its aims. The work by this organisation, spanning over 50 years, is the only lasting influence in the alcohol movement in this country. Indeed, it encompasses the whole of a review chapter on alcohol for the Encyclopaedia of Social Work [4].

Other voluntary organisations (such as Alcoholics Anonymous (AA)) which have flourished in the West, have had a very chequered history in India. They never really caught on in an organised fashion. The only organisation in addition to the above mentioned groups is the recently funded private organisation, the TTK Ranganathan Foundation in Tamil Nadu. Time alone will show if its ambitious programmes develop over the years.

Future directions

Hopefully, with increasing urbanisation and rapidly expanding alcohol production, alcohol research will become a priority issue. Reference has already been made to the 'Working Group on Alcohol and Drugs' under the Central Prohibition Committee. Another promising possibility would be the development of an 'Advanced Centre on Alcohol and Drugs' which has been recommended to the Indian Council of Medical Research.

Many reasons can be considered when speculating about the causes for lack of research, policy and funding

Table 2. Selected studies on students on alcohol and other drugs

Sr. No.	Investigator(s)	Area of study and year	Sample size	Sampling procedure	Funding status	Highlights
1.	Dube *et al.*[13]	Agra (1977)	564	Random sampling. School students. Universe not defined	N.K.	One-third of the sample used alcohol and cannabis while one-fourth reported use of barbiturates, sedatives and minor tranquilizers.
2.	Sethi and Manchanda[14]	Lucknow (1977)	748	Random sampling. Under-graduate and postgraduate medical students. Universe undefined	N.F.	Half of the students reported use of minor tranquilizers while 43% used alcohol.
3.	Varma and Dang[15]	Chandigarh and Raipur Rani (1978)	570	Sampling and universe not defined. School, University students.	N.F.	Alcohol was the first preferred drug. Drug use was more common among urban group.
4.	Singh and Singh[16]	Patiala (1979)	217	Cluster sampling. Medical students.	N.F.	Comparatively high prevalence rate of every drug. Besides alcohol and tobacco, use of hard drugs like amphetamines, barbiturates was reported.
5.	Mohan *et al.*[17]	Delhi (1979)	217	Cluster sampling. High school students	N.F.	One-fourth of the students reported use of alcohol and tobacco while one-tenth used cannabis and other drugs.
6.	Mohan *et al.*[18]	Delhi (1981)	2,032	Stratified random sampling. High School students	F	One-tenth of the students were using alcohol and other drugs.
7.	Mohan *et al.*[19] (Multi-centered study).	Bombay, Madras, Delhi, Jaipur, Hyderabad, Sagar, Varanasi (1976	26,161	Cluster sampling. College University students.	F	A multi-centered study using standardised questionnaire. Alcohol and tobacco commonly used drugs.

N.K. — Not known F — Funded NF — Not Funded

The first is its close association with the political and moral movement reflected in the prohibition approach. The second major reason is that it did not appear as an issue of immediate or remote concern in national health policy: it was always perceived to be a social evil. Lastly perhaps, the scientific community has lacked both the expertise and funding from the concerned agencies. It is our hope that this picture will soon change.

In one sense the Indian experience reflects a larger global concern of the health planners both at national and international levels as they tend to focus more on immediate problems, e.g. communicable diseases, nutrition, and infections, rather than simultaneously planning preventive activities, in problems such as alcohol, road traffic accidents, and industrial safety, which will bear fruit in decades to come. This blind spot persists despite World Health Assembly resolutions and various reports that are periodically issued from the Division of Mental Health, WHO Headquarters. Perhaps this indifferent response by national governments is reflective of WHO's own lack of conviction in the area of alcohol related problems.

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Family Therapy: Development, Issues, and Approaches

THE DEVELOPMENT OF FAMILY THERAPY

Family therapy has gradually gained widespread acceptance as a psychosocial intervention technique since its recorded beginnings nearly 30 years ago. Four recent reviews serve as a valuable social history on the development of family therapy. Haley's (1971a) chronological account covers important events in the field and the evolution of the theoretical bases of family therapy. Fox (1976) focuses on important issues--theories, techniques, ethics, assessment, outcome, and training--and the individuals associated with them; other sections of the review are devoted to prominent figures such as Murray Bowen and Theodore Lidz. Guerin (1976) presents a similar developmental history but couches it in the context of the geographical regions associated with different theoretical concepts. Stanton (1979) presents a comprehensive and updated review of this field. The reader with a particular interest in the historical development of family therapy will find these sources excellent supplements to the brief review presented in this report.

Historically, accounts of treating whole families in therapy began to appear in the mental health literature in the early 1950s. These accounts reflect the influence of such pioneers in family therapy as Frieda Fromm-Reichmann (Haley 1970), Nathan Ackerman (1958, 1966a, 1966b), and Gregory Bateson and his colleagues (1956). Fromm-Reichmann's work in the late 1940s pointed to a greater improvement in schizophrenic children when their mothers were included in the therapeutic process. This led others to speculate about the role of the father as well as the mother in the development of mental and emotional problems. Bateson and his colleagues soon began to include both parents in their clinical research on the causes of mental and emotional problems. By this time, Ackerman was already quite experienced in working with families. In the early 1930s, he incorporated into clinical practice his belief that many emotional and mental problems originated in and could be treated within the family. His first writings on the subject were not published until 1958, but knowledge of his work through other channels greatly influenced the practice of many prominent family therapists.

The influence of these diversely oriented practitioners and researchers enabled two broader and interrelated theoretical developments to further the growth of family therapy. These are learning theory and systems theory.

The principles of learning theory (Bandura 1969; Hawkins et al. 1966; Wolpe 1958) are viewed as particularly relevant to family therapy because of their emphasis upon changing behavior, establishing a similar set of operations for behavioral change, and evaluating therapy outcomes. A vital aspect of this learning theory orientation is that it permits each family member to monitor how his or her behavior has changed and how this change affects the behavior of another family member (Patterson 1971; Patterson et al. 1968; Stuart 1969).

The concerns of systems theory--e.g., homeostasis, communication patterns, deviation processes--were adapted to the therapeutic process by Hoffman (1971), Jackson (1957), and Watzlawick and his colleagues (1967). In applying these concerns to family therapy, the primary emphasis has been on helping family members understand that no member acts in isolation, that the actions of each member affect the actions of other members. Helping family members realize and understand this interdependence of behavior among themselves has been a major goal of the systems-oriented family therapy. This therapeutic process seeks to counter and interpret the situation which most often brings a family to the therapy, that is, the symptoms of the one ". . . identified patient, whom the family labels as 'having problems' or 'being the problem'" (Minuchin 1974).

Other theoretical movements have influenced family therapy also, but seemingly to a lesser extent than learning theory and systems theory (Haley 1971b). Accounts of these other movements appear in Fox (1976), Haley (1971a), and Guerin (1976).

FAMILY THERAPY ISSUES AND APPROACHES

Currently, there are many family therapy approaches and techniques, but there is, as yet, no universally accepted definition of family therapy. Still, there are grounds of common agreement. One, obviously, is that the therapist should focus on the family rather than on the individual. Further, each family member is to be equally considered in the therapeutic process. Less universal, but quite common, is the practice of avoiding medical terms such as treatment, patient, mental illness, and therapy. This is done to reduce identification with the medical model of treatment and its accompanying designation of one individual as the source of the family's problems.

Family therapists seem to agree, therefore, that if therapy is to be successful, an "identified patient" cannot be the focus of treatment. This leads to numerous important questions concerning the process involved in the identification of one family member as "the problem" or the "deviant" one--a process that takes place before therapy. Because the family therapist can only evaluate and help the family deal with the results of the process, rather than its development, it is essential for the therapist to understand the conditions most conducive to the development of the designation of a deviant individual in the family. A number of theoretical assertions have been suggested to help therapists understand these conditions or issues:

1. It is helpful if the family is viewed as a system. When this occurs, the problem or deviant behavior of any one family member is not viewed as an isolated act but may be viewed as either caused by the behavior of other family members or as resulting in changes in the behavior of other family members (Jackson 1957; Watzlawick et al. 1967).

2. The deviant behavior of one family member should not be considered as a random or inexplicable set of occurrences but as a behavior that fulfills a function. For example, a child's deviant behavior might function to draw the family's attention away from the family's feeling of being scorned by the larger community (Vogel and Bell 1960).

3. What may be considered as "deviant" behavior by one family may not be considered so by another family.

4. The causes and effects of deviant behavior in a family should not be viewed in isolation; several levels of the family system may be involved. Hoffman (1971) suggests that amplifying behavior on one level of the system (e.g., a child's defiance) may inhibit further deviance on another level (e.g., tension between parents that may break up the family). He and others (Buckley 1968; Nett 1968) believe it is unfortunate that therapists sometimes fail to view some deviant behavior as potentially beneficial on at least some level (such as a child's deviant behavior functioning to reduce tension, keeping the family together).

Most therapists agree that there is a deviance process similar to that described above, although different therapists use different terminologies in speaking of the process. And most agree that involving the whole family in the therapeutic process is the most efficient way of solving an individual's problems. Following are some examples of several major family therapy approaches used to counter and reverse the deviance process and engage the family's support in therapy.

Multiple Impact Therapy

This approach is a brief, usually 2-day, intensive study and treatment of a family in crisis conducted by a team of clinicians (e.g., a psychiatrist, psychologist, and social worker). Treatment is based on the assumptions that crises are times when families are most receptive to therapy and that greater progress

can be made in the early rather than in later stages of the difficulty (Caplan 1964; Macgregor 1962; Macgregor et al. 1964; Parad 1965; Ritchie 1960).

The interviewing procedures are somewhat unique. Interviews are held with the entire family unit and also with individual family members, both privately and in an overlapping interview. Team members focus on obtaining a family history and interventions are based on this material. In the overlapping interview, a team member who has been talking privately with a family member terminates this interview and joins another conference, either alone or accompanied by the family member s/he has been seeing. In this way, differences of opinion or interpretation between different family members are sometimes aired and resolved. Where there is an "identified patient," his or her communicative behavior may be shaped by being involved in gradually enlarged groups until s/he is comfortable speaking in the presence of the entire family.

A more recent but similar approach based on Milton Erickson's work has been encouraged by Jay Haley (1973, 1976). Sometimes termed "strategic therapy," the approach rests on a communications systems orientation. Intervention during crisis is considered desirable, therapy is brief (though longer than 2 days), and the entire family is seen (though perhaps in different groupings). The focus of therapy, however, is on presenting symptoms (rather than family history), and specific intervention strategies are based on identified symptoms.

Structural Family Therapy

This approach, like strategic therapy, is based on a systems-oriented family therapy (Guerin 1976) and is one of the most widely used techniques. It is the focus of this report because it has recently been used, apparently with some success, with families of heroin addicts (Stanton 1978a; Stanton et al. 1978; Stanton and Todd 1978).

Minuchin (1974), who has been most instrumental in developing structural family therapy, describes it as ". . . a body of theory and techniques that approaches the individual in his or her social context." It is based on three assumptions: (1) that the context of an individual's behavior affects inner processes; (2) that changes in context produce changes in the individual; and (3) that the therapist's behavior is significant in any movement toward change in the family structure. Minuchin considers the third assumption especially important, and stresses the therapist's intimate role in changing or "restructuring" the family's transactional patterns, alliances, subsystems, sensitivity to the individual member's actions, sources of support, and so on.

Minuchin and his colleagues have found these techniques especially effective when working with families they term "disorganized" (Minuchin and Montalvo 1967; Minuchin et al. 1967). Children or

young adults in these families are often the "victims" in the family's shifting alliances, and structural family therapy techniques reportedly help identify and change such subgroupings for the benefit of all, including the person the family has chosen to be the "victim."

Other Systems-Oriented Approaches

In one view, any therapist who ventures into the family area becomes, by implication, a "systems-oriented" therapist. Some approaches, however, are more grounded in systems theory than others. Particularly notable in this regard are those techniques and theories which involve the analysis of a family's communication problems and behaviors.

Although it is difficult to differentiate clearly the several approaches employing communications analysis in family therapy, several individuals have emerged as advocates of this set of techniques. A brief description of the ideas of each of these individuals follows.

Gregory Bateson. Although Bateson is clearly a leader in this field, several researchers/practitioners who have either worked with Bateson or relied on his concepts could lay claim to being on an equal plane in the application of systems and communications principles to family therapy (e.g., Don Jackson, Jay Haley, Paul Watzlawick, and John Weakland). Their clinical research with families led to two concepts which remain important in the theory and practice of family therapy today, namely, the concepts of double bind (Bateson et al. 1956; Weakland 1960) and family homeostasis (Jackson 1957).

The importance of these concepts for family therapy, more than the specific therapeutic techniques derived from them, lies in the way they require the therapist to view the family. The double bind concept emphasizes the disturbed communication patterns present in a family and calls for the therapist to be aware of these patterns and, especially, to make the family aware of them. The concept of family homeostasis requires that the family be viewed as a system which, when its balance is threatened, will take the necessary steps to recover or maintain that balance. Viewing the family from this perspective causes the therapist to radically reorient his/her approach to family therapy, to be aware of changes in the family system as well as the causes of those changes, and, most importantly, to make the family think of itself as a system.

Virginia Satir. In the late 1950s and early 1960s, Satir was associated, at the Mental Research Institute (MRI) in California, with a number of individuals from the Bateson group, notably Jay Haley and Don Jackson. She believes, quite simply, ". . . that by observing and learning to understand communication in a family we can discover the rules that govern each individual's behavior" (1971). Her technique involves viewing the family as

an open system which has developed its own rules about how changes may occur: (1) within individual family members; (2) between family members; and (3) between family members and the demands of the social environment (Satir 1967). She believes that the therapist helps the family to uncover these rules, make them explicit, and analyze how they affect the operation of their family system.

The goals of Satir's therapeutic approach are related to this analysis of family communication. Three changes in the family system are sought in the following ways:

> First, each member of the family should be able to report congruently, completely, and obviously on what he sees and hears, feels and thinks, about himself and others, in the presence of others. Second, each person should be addressed and related to in terms of his uniqueness, so that decisions are made in terms of exploration and negotiation rather than in terms of power. Third, differentness must be acknowledged and used for growth.
>
> (1971)

Satir believes that when these changes are able to be achieved, communication within the family will lead to appropriate outcomes. These "appropriate outcomes" are defined by Satir (1971) as:

> . . . decisions and behavior which fit the age, ability, and role of the individuals, which fit the role contracts and the context involved, and which further the common goals of the family.

Murray Bowen. Bowen has utilized systems theory and communications analysis in a somewhat different manner than have Bateson and Satir. First, Bowen believes that an "undifferentiated family ego mass" exists in varying levels of intensity in all families. This "conglomerate emotional oneness" (Bowen 1961, 1966, 1973) is, in its more intense forms, debilitating for a family. Its effects may be relieved by encouraging differentiation of self among the family members, that is, helping each member of the family to see themselves as individuals who are a part of many systems, including but not limited to, the family system.

Second, Bowen asserts that family problems are the result of a multigenerational transmission process. Intervention in this process may be accomplished by an analysis of current family interaction, as well as through historical analysis.

Finally, Bowen cautions that the identified patient in any troubled family may be involved in a very complex communications pattern. This individual may be "triangled," that is, forced to play the role of the mediator of communication between the parents. S/he may become the scapegoat and receive only negative communication or may remove himself or herself from family communication in order to survive as an individual.

Guerin (1976), in summarizing Bowen's approach, notes that:

> The Bowenian model is cautiously idealistic and optimistic about the human potential for growth and change. It is strongly based on a philosophy of free will. Education at its best is seen as a combination of the implicit knowledge of experiences, solidified and reproduced by cognitive appreciation of its form.

Existential Approaches to Family Therapy

Laing (1969) and Laing and Esterson (1964) suggest a helpful and unique way of considering families in which one or more individuals report emotional difficulties, although they offer no specific techniques.

The approach was derived from studies of families with a schizophrenic child. It involves reformulating the behavioral bases of such families. Ordinarily, the behavior of these families is considered bizarre or senseless because family interactions are seldom considered. However, Laing points out that the behavior may make sense if it is viewed in the original family context; there may be a good reason, however unspoken, for the seemingly bizarre acts of the family members.

Like Bowen, Laing considers the study of several generations of the same family an important diagnostic tool, especially where there is an "identified patient." Considering the identified patient's behavior in the context of other family members' behavior is believed to be especially useful. The patient's behavior that seems so inappropriate in most social contexts may come to be viewed as a necessary means of coping when considered in the family context.

SUMMARY

Each of the broad classes of techniques may be used in resolving difficulties faced by family units. The family structure, the problem at hand, and the particular skill and training of the therapist make up the variables that are considered when choosing a particular therapeutic modality or technique.

Haley (1971a) makes the further point that a family therapist, once he or she gains experience, will begin to view these techniques of family therapy ". . . not . . . as a method of treatment--one more procedure in a therapist's armentarium . . . but as a new orientation to the arena of human problems."

There is evidence that this new orientation is taking hold, that family therapy is being adapted to other than specific mental health problems. It is now being used in several other areas, including drug abuse, corrections, and alcohol abuse.

family therapy services in the drug treatment field. However, they suggest that several family therapy approaches (e.g., structural and multifamily therapy) are effective in treating drug abuse clients.

Family Therapy with Alcohol Abusers

The literature contains many positive claims about the effectiveness of family therapy with alcoholics. As in other fields, such claims are based mostly on clinical impressions which have been supported by a few recent empirical studies. These studies will be discussed after a brief presentation of major theoretical orientations related to the use of family therapy in the alcoholism field. It can be noted that the theoretical considerations and studies are limited to alcoholism; there appear to be no attempts yet to focus on concurrent use of alcohol and other drugs.

THEORETICAL ORIENTATIONS

The use of family therapy with alcoholics is a relatively recent development. During the 1950s, the prevailing theories on alcoholism were not compatible with the psychosocial concepts underlying the emerging field of family therapy. Alcoholism was then viewed as a disease which absolved the patient of any responsibility for his or her behavior. Treatment reflected such theories of causation as biochemical sensitivity and oral dependency (Davis et al. 1974).

In the late 1960s many investigators began to believe that environmental factors were important contributors to alcoholism. The behavioral model gained in prominence and family therapy was introduced into the field. Alcoholism began to be viewed as a symptom of larger family problems (Steinglass 1976; Steinglass et al. 1971).

Since that time, no standard definition of family therapy has been adopted in the field. Many definitions appear in the alcoholism literature, and they reflect diverse opinions about who should be included in family therapy and/or which method of intervention should be used and in which setting.

There are also various different theoretical concepts related to the drinking process; these generally incorporate a systems theory approach, and alcoholism and family treatment are viewed in the context of that system. Among the most influential theorists and/or practitioners on this subject are Bowen, Ewing, Fox, Steinglass, Davis, and their associates.

In Bowen's (1974) framework, "excessive drinking occurs when family anxiety is high." This excessive drinking heightens the anxiety of family members who are dependent upon the drinker; they, in turn, "react by anxiously doing more of what they are already doing." Bowen states that this "process of drinking to relieve anxiety" and "increased family anxiety in response to drinking, can spiral"; the result may be "functional collapse" or the development of a "chronic pattern." In his view the goal of family therapy is to reduce the level of spiraling anxiety so that family functioning patterns can be examined and improved. Bowen believes that any "significant" family member who can "cool" the anxious response," or their own anxiety, "can make a step towards deescalation"; thus, family therapy sessions may be limited to one or two family members without the drinking member necessarily being present.

Ewing and Fox (1968) view alcoholism as an established part of rigid family interactional patterns which maintain family homeostasis. All family members strongly resist changes in drinking patterns--including abstinence--because the changes threaten the family "status quo." Steinglass (1976) also notes that alcoholism may serve as a stabilizing factor in the family, one which produces "extremely patterned, predictable, and rigid sets of interactions." In his view, these interactions reduce uncertainties not only about family life but also about the family's relationship to society. Thus, the goal of family therapy is to increase understanding about the role of drinking in the family so that interpersonal relationships may be improved. Treatment is focused on nurturing family growth, rather than on a reduction in drinking, and the entire family is viewed as "the patient."

Davis and associates (1974) include aspects of behaviorism in their theoretical approach. They view alcoholism as having certain adaptive consequences which all family members reinforce in ways that maintain the drinking habit. In this framework, the goals of family therapy are to discover the adaptive functions and reinforcements of drinking, to help the family members use this adaptive behavior during periods of sobriety, and to assist members in learning adaptive alternative behaviors.

UTCOME STUDIES

...ere is very little published research on the effectiveness of family therapy with alcoholics. Most of this research has been conducted by specialists in alcohol rather than by family therapists.

Most of the research based on family therapy outcomes with alcoholics appears in two reviews by Steinglass (1976, 1977). The 1977 Steinglass review includes only 10 studies. All of these support the use of family therapy with alcoholics. However, these studies are so limited in number, comparability, and methodological rigor that one cannot draw any firm conclusions about the effectiveness of family therapy with alcoholics. For example,

outcome measures ranged from highly subjective measures such as social a[illegible] marital satisfaction to measures of abstinence from alcohol. The use of abstinence as an outcome measure is especially controversial because of existing research which indicates that some alcoholics are able to drink socially after receiving treatment (Ewing 1974; Pattison 1968; Pattison et al. 1968).

The studies included in the Steinglass reviews are further limited by the nearly universal failure of the researchers to use comparison groups or to include many female alcoholics in their samples. The failure to include female alcoholics in outcome studies may introduce a bias that has serious implications for treatment. Meeks and Kelly (1970), for example, have argued that:

> . . . wives of alcoholics seemed better able to shift the focus to the family unit and to view their own behavior within the framework; husbands, with their masculinity and competence at stake, may have a greater need to keep the alcoholic wife in the sick role. When the husband is the alcoholic he may have less difficulty relinquishing the role of identified patient.

A large-scale and as yet unpublished study of family therapy outcomes supports the claims of sex differences in treatment for alcoholics. These differences were found by Williams (1972) in his evaluation of the Hospital Improvement Project at the Center for Alcoholics in Avon Park, Florida.

In that study, 44 percent of 647 patients offered family therapy chose to participate in that treatment. Only 17 percent of the total 647 completed the 8 sessions (initially in the office and later at the client's home) that were intended. Intact families were far more receptive to the treatment than other families; about three-fourths of the patients living with a spouse and children received the therapy. Also more likely to participate in the family therapy were patients of "middle class and above" social status. While nonwhite patients were as likely as white patients to accept the therapy initially, nonwhites were less likely to complete all three home sessions.

Participation in the family therapy appears to contribute to full-time employment and increases in attendance at Alcoholics Anonymous among patients at followup (i.e., 6 to 12 months after discharge); these findings were more characteristic of male than of female patients. The family therapy also seemed to influence the likelihood of abstinence at followup; this finding was more characteristic of patients who completed all four therapy sessions. At followup, a majority of the males showed significant changes in "gains in self-awareness"; these changes were not found in the majority of females, even though females were judged to have a "less severe" degree of impairment on psychiatric formulation measures at the time of intake.

Data from two small-scale studies raise the question of whether many alcoholics hold as positive a view of family therapy as professional proponents of the method.

Price and Curlee-Salisbury (1975) obtained attitudinal data from 51 male alcoholics after their discharge from alcoholism treatment at the Veterans Administration Hospital in Indianapolis. These men rated eight treatments they received on eight "helpfulness" dimensions; treatment included such interventions as group therapy, individual counseling, lectures, and family counseling. Of the treatments, family counseling received among the least favorable ratings on "worth," "therapeutic benefit," and "pleasantness," and was not ranked highly on the remaining five dimensions.

Similar results have been reported by Hoffman et al. (1975-76). They compared attitudes toward treatment among two groups of male alcoholics who had previously completed a 6-week Alcoholics-Anonymous-oriented program where they received the six types of treatment (detoxification, lectures, group therapy, individual counseling, work therapy, and family therapy). In rating treatments, a significantly higher percentage ($p=0.03$) of the "successful" group rated family therapy as "most helpful";[1] however, the percentages of "most helpful" responses were quite low in both groups (22 versus 7 percent in the "successful"--N=37--and "unsuccessful" groups--N=46). In terms of the "total" group, family therapy was as likely to receive a "least helpful" (14 percent) as a "most helpful" (13 percent) rating. Treatments that received the highest percentages of "most helpful" ratings from the total group were group therapy (54 percent) and individual counseling (26 percent).

While one obviously cannot generalize the findings from the two studies on attitudes toward family therapy to the large universe of alcoholics, these studies do suggest significant optimism in this area.

SUMMARY

Reports based on clinical impressions suggest that various types of family therapy are effective in the treatment of alcohol abuse. Positive claims have been made by professionals for a systems approach using concurrent therapy, conjoint therapy, and marital-couples therapy. Two small-scale studies on client attitudes raise the question of whether alcoholics view family therapy as positively as do clinicians.

Empirical studies provide limited support for the clinical impressions. These studies generally are based on small samples, lack

[1]These differences in group ratings may be related to statistically significant group differences in marital status and educational backgrounds. Compared to the "successful" group, the "unsuccessful" group was less likely to be married at the time of the study (41 versus 73 percent) and had fewer mean years of education (9.5 versus 11.8 years).

comparison groups, focus on male alcoholics, and use a variety of therapies and outcome measures. The research findings cannot be generalized to all treatment programs and all alcoholic clients.

with and without cirrhosis, there is a strong positive correlation between hepatocyte size and portal pressure (10). However, as a group, cirrhotics have a higher mean portal pressure associated with a larger mean hepatocyte size than noncirrhotics (10, 11). Elevated portal pressures were found in 49.4% of alcoholic patients without cirrhosis, and 23.6% of patients with cirrhosis have pressures below 10 mm Hg (10).

It should be noted that while the hypothesis of hepatocyte expansion followed by sinusoidal compression appears attractive as a mechanism to increase hepatic resistance and thus portal pressure, this postulate does not exclude the contribution of a number of other factors, such as fibrosis, compression by ascites, or portal blood flow, in determining portal pressure. In fact, the correlations observed suggest that in some cases the mechanism of portal hypertension is not related to sinusoidal compression; this certainly appears to be the case in our sample of patients with nonalcoholic liver disease. Our data do not allow us to conclude on these other mechanisms.

The mechanism postulated here to play an important role in portal hypertension, based on hepatocyte expansion, could provide a possible explanation for the observation that in some patients portal pressures are reduced within a short period of hospitalization (26, 27). Although, obviously there is no information derived from human studies on the time it takes for enlarged hepatocytes to return to normal after withdrawal from ethanol, data in rats have shown that nonomegaly subsides with a half-life of approximately 4 days after discontinuing alcohol administration (28). These results support a causal relationship between hepatocyte enlargement, compression of the sinusoids and portal hypertension.

These data also suggest that further investigation of the mechanisms leading to hepatocellular expansion induced by ethanol might result in new approaches to treatment of portal hypertension, one of the leading causes of mortality in alcoholic liver disease.

Acknowledgment: The invaluable assistance of Dr. R. Colapinto in performing the transjugular liver biopsies is gratefully acknowledged.

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To Moise Jacob Best Wishes always. Joe Vidins

0270-9139/85/0503-0408$03.00/0
HEPATOLOGY
Copyright © 1985 by the American Association for the Study of Liver Diseases
Printed in U.S.A.
Vol. 5, No. 3, pp. 408–414, 19

Sinusoidal Caliber in Alcoholic and Nonalcoholic Liver Disease: Diagnostic and Pathogenic Implications

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Portal hypertension in alcoholic liver disease has been attributed to an increased resistance to blood flow either of sinusoidal or of postsinusoidal origin. The former should be accompanied by sinusoidal compression while the latter is expected to result in an increased or a normal sinusoidal diameter. Patients with alcoholic liver disease showed a marked reduction ($p < 0.001$) in relative sinusoidal area ($995 \pm 135\ \mu m^3$; $n = 19$) when compared to nonalcoholic patients with normal liver histology ($6,100 \pm 389\ \mu m^3$; $n = 6$), or to patients with nonalcoholic liver disease ($6,242 \pm 467\ \mu m^3$; $n = 19$). Hepatocyte surface area was significantly increased in patients with alcoholic liver disease when compared to hepatocytes from normal biopsies ($663 \pm 32\ \mu m^2$ vs. $301 \pm 28\ \mu m^2$; $p < 0.001$). Patients with nonalcoholic liver disease had hepatocyte surface areas within the normal range ($327 \pm 14\ \mu m^2$). There was a significant inverse correlation between hepatocyte size and sinusoidal area ($r = -0.63$; $p < 10^{-4}$; $n = 44$), indicating that larger hepatocytes were associated with sinusoidal compression. In the alcoholic patients, portal pressure correlated inversely ($r = -0.77$; $p < 0.01$) with sinusoidal area only after the sinusoidal area was markedly reduced to areas below 20% of normal. Such a threshold was not reached in patients with nonalcoholic liver disease, in whom no correlation between sinusoidal area and portal pressure was observed. Rats fed chronically with a diet containing 35% of calories as ethanol, in which liver enlargements of 36 to 42% were observed relative to controls fed an isocaloric carbohydrate diet, had a significant reduction in both extracellular space and blood space per unit liver weight. Data presented support the hypothesis that hepatocyte expansion and compression of the sinusoidal space appear to be important determinants in the development of portal hypertension in alcoholic liver disease. In addition, the striking difference in the observable sinusoids in alcoholic and nonalcoholic liver disease should provide an added criterion in the histological differentiation of the two conditions.

The increased hepatic resistance to portal blood flow that occurs in intrahepatic portal hypertension has been classified as: (a) presinusoidal; (b) sinusoidal, and (c) postsinusoidal in origin (1). In alcoholic liver disease, portal hypertension has been frequently attributed to compression of the venous outflow by expanding, "regenerative" nodules (2–5) or by sclerosis or occlusive lesions of the terminal hepatic vein (central vein sclerosis) (6–8, Miyakawa, H. et al., *Gastroenterology* 1983; 84:1385, Abstract). Recent studies have suggested that in alcoholic liver disease hepatocyte expansion and a reduction in hepatic extracellular space may play important roles in the genesis of portal hypertension (9–11). According to this hypothesis, sinusoids should be compressed, while in the case of a postsinusoidal block, such as the one produced by occlusion of the venous outflow, this should not occur. Although recently the concept of portal hypertension in alcoholic cirrhosis resulting exclusively from postsinusoidal resistance has been questioned (12, 2), to our knowledge no studies have been conducted to determine sinusoidal caliber in alcoholic liver disease and to compare it to the sinusoidal caliber in nonalcoholic liver disease or histologically normal liver.

We report studies in which we show that in alcoholic liver disease there is a dramatic reduction in sinusoidal area, which is not seen in biopsies of patients with nonalcoholic liver disease or in nonalcoholics with normal liver histology, in line with a sinusoidal mechanism for portal hypertension in alcoholic liver disease.

Received June 8, 1984; accepted December 18, 1984.
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NH-29-31

MATERIALS AND METHODS

Human Studies

A total of 44 patients with liver biopsies were entered into the study; 38 of these had portal pressures measured. Nineteen of these patients were consecutive admissions for alcoholic liver disease (14 men, 5 women; mean age 53 ± 2.8 years) in whom alcohol as an etiological factor (more than 80 gm per day) was assessed on the basis of personal interviews. In this group, according to histological diagnosis, 12 patients had cirrhosis with or without hepatitis, 2 had alcoholic hepatitis and 5 had fatty livers. Another 19 patients were consecutive admissions for nonalcoholic liver disease (6 men, 13 women; mean age 46 ± 3.8 years). Alcohol abuse in the patients classified as having nonalcoholic liver disease was excluded on the basis of personal interviews. In addition, the majority of these patients had histological characteristics which clearly distinguished them from the usual abnormalities found in alcoholic liver disease. According to history, laboratory tests and biopsy data, 9 patients had chronic hepatitis of different etiologies, 1 had primary biliary cirrhosis, 2 had idiopathic portal hypertension and 7 had cirrhosis with positive viral or immunologic markers. For comparison, we included 6 nonalcoholic patients (3 men, 3 women; mean age 51 ± 6.2 years) in whom biopsies had been performed to rule out the following conditions: metastatic tumors, 2 patients; Gilbert's disease, 1 patient; ulcerative colitis, 1 patient; unexplained moderate increase in transaminase, 1 patient, and unexplained pruritus, 1 patient. All of the biopsies in these cases were reported as normal. These normal biopsies were retrospectively collected from the pathology records of the Toronto Western Hospital, in a consecutive manner, until six patients were identified. Data for portal pressure were not available in these subjects.

All percutaneous biopsies in this study were obtained with a Menghini needle, while the transjugular biopsies (12–14) were obtained as previously described (10). In the 19 patients with alcoholic liver disease, only one biopsy was performed by the transjugular route. In the nonalcoholic patients, 9 had transjugular biopsies, and 10 had percutaneous biopsies. All biopsies were fixed immediately with 10% formalin and processed for light microscopy. A variety of staining techniques thought to be of value in the diagnosis of alcoholic liver disease were used. Morphometric data presented in this paper were obtained from hematoxylin-eosin stained slides. All biopsies were examined and reported by one of us (A. M.). Written, informed consents for the biopsy and the pressure measurements were obtained. The study was approved by the Joint University of Toronto-Addiction Research Foundation Committee on Ethics in Human Experimentation.

Determination of Sinusoidal Caliber

Sinusoidal area, as a function of sinusoidal caliber, was measured by quantitative digital-image analysis using a MOP-3 modular system (Carl Zeiss, Inc., Eching, West Germany). Micrographs were taken at 400× magnification with an Orthoplan microscope fitted with a Vario Orthomat camera (Leitz, Wetzler, West Germany). Four or five prints (10.5 × 16.5 cm) from different areas of each biopsy were taken, and the area of the biopsy included in each print (45,900 μm^2) was calculated with the use of a Leitz stage-calibration micrometer, 2 mm in length, with 10 μm divisions. All prints were made keeping a constant negative-to-positive magnification factor. Areas in the micrographs were determined by adjusting the total print area measured by the digital computer to the 45,900 μm^2, and sinusoidal area was measured within this total area. Final sinusoidal area, expressed in μm^2 per 45,900 μm^2 total area, corresponds to the average sinusoidal area in the complete set of prints from each specimen in which all the sinusoidal areas were measured. The areas photographed were selected so as not to include portal areas, terminal hepatic veins or areas of fibrosis.

Determination of Hepatocyte Size

In our earlier studies, we estimated hepatocyte surface area by counting the number of nuclei in hepatocytes per field and dividing this number into the total area of the field (9, 10). In this study, we determined if the use of computerized surface analysis would yield a better system for this measurement. This was not borne out, since, even in the liver biopsies reported as normal, the computer method required the selection of specific cells with clearly defined boundaries and nuclei. We found that surface areas determined by the computer method, as seen in normal biopsies or in biopsies of patients with nonalcoholic liver disease, presenting small hepatocytes with often well-defined boundaries and nuclei, correlated weakly ($r = 0.55$) with the method of counting the nuclei. In individuals with alcoholic liver disease in whom hepatocytes were frequently irregular, with imprecise borders, the tracing of the cell contours was virtually impossible for the majority of cells. Therefore, this method did not appear adequate. Thus, the method of counting nuclei provided a better estimation of relative cell enlargement. While this method could be improved by measuring cell surface areas by nuclear counts while correcting total areas by subtracting the computer-derived sinusoidal area, we found that correction for sinusoidal area altered the results of cell size by only 3% in alcoholics and by 17% in nonalcoholics or in normals. Since we found that involving such an elaborate method was not warranted, as the interpretation of the results was not influenced by this correction, we have presented our data without correction for sinusoidal area.

As in any study in which tissues have to be processed for histological observation, there is the possibility of introducing artifacts. However, it is usually assumed that the relative difference in size of cells seen histologically reflects the difference in size *in vivo*. Since this study was a comparative one, the actual size of hepatocytes and sinusoids *in vivo* is not critical for the interpretation of the results.

Pressure Determinations

Portal pressures were determined in 19 patients (9 alcoholics and 10 nonalcoholics) by direct measurement

of portal vein pressure, using the Chiba needle (23-15.0 DCN, Cook-Bloomington Inc., Markham, Ontario, Canada) under fluoroscopic guidance (15, 16). In all of them, intrahepatic (interstitial) pressure was also determined during the same procedure. In 10 patients (9 nonalcoholic and 1 alcoholic), pressures were determined by measuring wedged hepatic vein pressure via the transjugular route (17). In nine alcoholic patients, only intrahepatic pressures were determined as previously described (9, 10, 18). Zero pressure was adjusted by placing the transducer at the midaxillary line at the same level as the Chiba needle, with the patient in the supine position. The same reference point was used for wedged hepatic vein pressures. Pressures were recorded using a pressure transducer (Gould Statham SP1405, Gould, Oxnard, Calif.) and a recorder (Statham SP2009, Gould).

It is accepted that wedged hepatic pressure and portal vein pressure correlate very well (17, 19). We have further examined the correlation between intrahepatic pressure and portal vein pressure in the 19 patients in whom we measured both pressures. In these patients, following anesthesia of the skin and the intercostal muscles with 2% xylocaine, the needle position was verified radiologically to ascertain the optimal position of entry. A small skin incision was made at this point with a scalpel. With the patient apneic at end expiration, the Chiba needle was inserted parallel to the X-ray table. The needle obturator was then removed, and the needle was connected to the pressure transducer and the whole system was filled with saline. Intrahepatic (interstitial) pressure was then measured and recorded. The needle was then advanced further into the parenchyma, and a small amount of Hypaque-M, 60%, (Winthrop, Aurora, Ontario, Canada) was injected through a three-way stopcock attachment until a branch of the portal vein could be identified fluoroscopically. The injection was then stopped, and intraportal pressure was measured after fluoroscopic disappearance of the contrast medium. This procedure was performed twice in each patient by slightly modifying the angle of needle insertion. As shown in Figure 1, intrahepatic (interstitial) pressures correlate very strongly ($r = 0.92$) with portal vein pressure in the same patient. This confirms previous reports showing similar correlations between intrahepatic pressures and either wedged hepatic vein pressure ($r = 0.93$ and $r = 0.85$) (18, 20) or portal vein pressure measured at surgery ($r = 0.93$) (21).

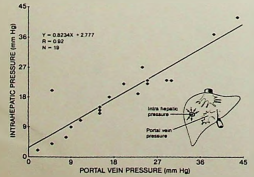

FIG. 1. *Relationship between intrahepatic pressure and portal pressure.* See "Materials and Methods" for details on procedures.

ANIMAL EXPERIMENTS

Four-week old female Sprague-Dawley rats (Charles River, St. Constant, Quebec, Canada) were fed a liquid diet containing 35% of calories as ethanol for 30 days. Control animals were pair-fed a diet in which ethanol was replaced isocalorically with carbohydrate (22). The day before sacrifice all animals received control diet *ad libitum*.

The vascular space of the liver was determined by measuring the amount of blood in the liver, using the spectrophotometric method described by Dahlberg (23). The animals were sacrificed by decapitation, and the blood was collected in heparinized tubes. This procedure assumes an equal degree of exsanguination from the liver in both the ethanol and control groups. The hematocrits were not significantly different in the two groups of rats. The liver was removed, weighed and a portion was homogenized in 24 volumes of 50 m*M* Tris buffer (pH 7.4) containing 1 m*M* EDTA and 10% v/v glycerol. The homogenates were centrifuged at 105,000 × *g* for 1 hr, and after removing the fatty layer, the clear supernatant was collected. A sample of blood from each animal was homogenized in 61 volumes of the same buffer, and centrifuged at 43,500 × *g* for 40 min to obtain a clear supernatant, which was diluted a further 11 times with buffer. The liver and blood supernatants obtained were scanned in a Kontrondual-beam recording spectrophotometer between 280 and 700 nm, using buffer in the reference cuvette. Dahlberg (23) reported that supernatants of blood and tissues containing blood had absorbance peaks at 540 and 580 nm, and that the heights of these peaks were proportional to the amount of blood present. The current experiment confirmed these results for the liver. Different amounts of blood were added to homogenates of perfused livers, which contained no blood, and after centrifugation, the absorbances of the supernatants at 540 and 576 nm were determined. Linear plots were obtained, indicating a direct proportionality between the amount of blood present and the absorbance at either of these wavelengths. The recovery of blood added to liver homogenates was measured and found to be 107 ± 5%; there was no significant difference in recovery in liver homogenates from ethanol-treated and control rats. The blood space of the liver was calculated by dividing the absorbance (at 540 nm) of the supernatant from a known amount of liver by the absorbance of the supernatant from a known amount of blood from the same animal.

Hepatic extracellular space was determined using [^{3}H] methoxy-inulin (New England Nuclear Canada, Lachine, Quebec, Canada; specific activity 238 mCi per gm). Each rat was lightly anesthetized with ether, and a dose of 2 μCi per 100 gm body weight (in 0.2 ml of a solution containing 5% w/v cold inulin in 0.154 *M* NaCl, pH 7.4) was injected into the femoral vein. After 5 min, the animal was sacrificed by exsanguination from the abdominal aorta. The blood was collected and centrifuged to yield serum. The liver was removed, weighed, and a portion was homogenized in 9 volumes of 0.55% w/v inulin in distilled water. The liver homogenate was centrifuged at 43,500 × *g* for 60 min. It was found that this procedure released all of the [^{3}H]inulin from the tissue, since the same results were obtained after complete tissue digestion. Aliquots of the liver supernatant and of serum were counted for ^{3}H. The inulin space (milliliters per gram of liver) was calculated by dividing the amount of ^{3}H in the liver (disintegrations per millisecond per gram) by the concentration of ^{3}H in serum (disintegrations per millisecond per milliliter) per 0.94, where 0.94 is the correction for serum water (24).

STATISTICS

Results are expressed as means ± S.E. Statistical significance ($p < 0.05$) was determined using Student's t test. Regression analyses were performed using a preprogrammed Radio Shack (Division of Tandy Corp., Barrie, Ontario, Canada) TRS80 microcomputer.

RESULTS

Table 1, in which sinusoidal areas are presented, shows that sinusoids are markedly compressed by 80 to 84% in the livers of patients with alcoholic liver disease, when compared to either patients with nonalcoholic liver disease or to patients with normal liver biopsies. In only 1 of 19 patients with nonalcoholic liver disease was the sinusoidal area compressed to values within the range found in alcoholic liver disease. Since there were 13 women and 6 men in the nonalcoholic liver disease group, the sinusoidal area between these two subgroups was compared and found to be not significantly different (women 6,680 ± 561; men 5,464 ± 632; not statistically significant). Data also showed that hepatocyte surface areas were markedly increased by 72 to 87% in alcoholic liver disease, when compared to those in nonalcoholic liver disease or in biopsies reported as normal. In nonalcoholic liver disease, hepatocyte surface areas were not significantly different from those of patients with normal liver histology. Average portal pressures in the patients with alcoholic liver disease were elevated (17.7 ± 2.3 mm Hg) and were not different from those in patients with nonalcoholic liver disease (21.0 ± 2.7 mm Hg).

TABLE 1. PORTAL PRESSURE, CELL SIZE AND SINUSOIDAL AREA IN PATIENTS WITH NORMAL BIOPSIES, NONALCOHOLIC LIVER DISEASE (NALD) AND ALCOHOLIC LIVER DISEASE (ALD)

	Normals (6)	NALD (19)	ALD (19)
Sinusoidal area (μm^2/45,900 μm^2 total area)	5,100 ± 389 (4,183–6,826)*	6,242 ± 467 (2,137–9,908)	995 ± 135 (301–2,422)
Cell surface area (μm^2)	361 ± 26	327 ± 14	563 ± 32
Portal pressure (mm Hg)	—	21.0 ± 2.7	17.7 ± 2.3
Differences	Normals vs. NALD	Normals vs. ALD	NALD vs. ALD
Sinusoidal area	NS†	$p < 0.001$	$p < 0.0001$
Cell surface area	NS	$p < 0.001$	$p < 0.0001$
Portal pressure	—	—	NS

* Range.
† NS, not statistically significant.

Figure 2 shows the striking difference in the appear ance of sinusoids in biopsies from patients with alcoholi and nonalcoholic liver disease. A marked reduction i sinusoidal space is seen in the former, where sinusoid are hardly visible. Also seen in Figure 2 is the larger siz of hepatocytes in the liver of patients with alcoholic live disease.

We have previously reported a good correlation ($r =$ 0.74 to 0.79) (11) between hepatocyte surface area an

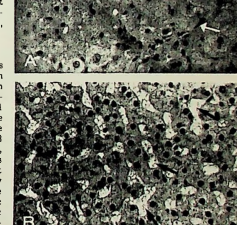

FIG. 2. *(A) Light microscopy from a liver biopsy of a patient wit alcoholic liver disease (× 400). (B) Light microscopy from a liver biops of a patient with nonalcoholic liver disease (× 400).* Note the gre reduction in the calibers of sinusoids (*arrows*), in alcoholic liver disea as compared to those in nonalcoholic liver disease. Hepatocytes i alcoholic liver disease are markedly enlarged.

portal pressure in alcoholic liver disease, irrespective of the presence or absence of cirrhosis. We have reconfirmed this observation ($r = 0.66$; $p < 0.01$, data not shown). In nonalcoholics, however, no such correlation was found ($r = 0.32$; not statistically significant) (Figure 3).

We have analyzed the interrelation between sinusoidal area and cell surface area in the complete patient sample. As shown in Figure 4, a good inverse relationship was observed between these two parameters ($r = -0.63$; $p < 10^{-6}$; $n = 44$), indicating that smaller sinusoidal areas were associated with larger hepatocytes. Further analysis of the data (curve in Figure 4) shows that, within the normal biological variation in cell size, sinusoidal areas can vary markedly, while a strong inverse correlation ($r = -0.79$, $p < 0.001$) between cell size and sinusoidal area is found in the pathological range of sinusoidal areas below 900 μm^2.

For the alcoholic patients, the relationship between portal pressure and sinusoidal area was found to be conspicuously exponential in nature, and by computer iteration it could be best described by two linear functions in relation to sinusoidal areas either greater or smaller than 900 μm^2. Figure 5 shows the strong negative correlation ($r = -0.77$; $p < 0.01$) between sinusoidal area and portal pressure for livers in which sinusoidal areas were smaller than 900 μm^2. No correlation between sinusoidal area and portal pressure was found for livers presenting larger sinusoids ($r = -0.17$; not statistically significant).

In nonalcoholic liver disease in which all sinusoidal areas exceeded 2,000 μm^2, no correlation was observed between sinusoidal area and portal pressure ($r = -0.15$, not statistically significant; $n = 19$) (data not shown).

In the group of nonalcoholic liver disease patients, 10 of 19 patients had transjugular biopsies, while the rest had percutaneous biopsies. Since the methods of obtaining the biopsy specimen could conceivably have different effects on liver morphology, we compared sinusoidal areas and cell surface areas in these two subgroups. No statistically significant differences were observed (sinusoidal areas: 5,786 ± 647 and 6,675 ± 671 μm^2; cell surface areas: 373 ± 26 and 391 ± 18 μm^2, for the transjugular and percutaneous biopsies, respectively). Portal pressures in these two subgroups were not significantly different (24 ± 3.8 and 19.8 ± 3.4 mm Hg, respectively).

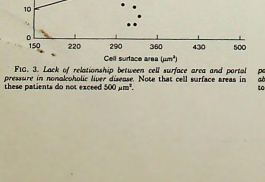

FIG. 3. *Lack of relationship between cell surface area and portal pressure in nonalcoholic liver disease.* Note that cell surface areas in these patients do not exceed 500 μm^2.

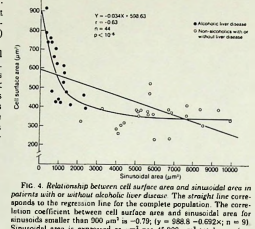

FIG. 4. *Relationship between cell surface area and sinusoidal area in patients with or without alcoholic liver disease.* The *straight line* corresponds to the regression line for the complete population. The correlation coefficient between cell surface area and sinusoidal area for sinusoids smaller than 900 μm^2 is -0.79; ($y = 988.8 - 0.692x$; $n = 9$). Sinusoidal area is expressed as μm^2 per 45,900 μm^2 total area (see "Materials and Methods").

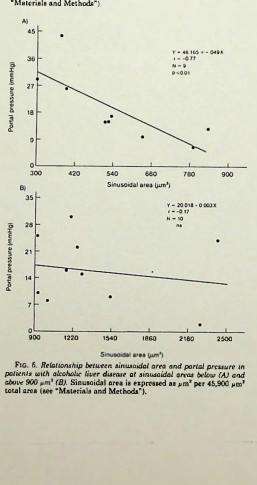

FIG. 5. *Relationship between sinusoidal area and portal pressure in patients with alcoholic liver disease at sinusoidal areas below (A) and above 900 μm^2 (B).* Sinusoidal area is expressed as μm^2 per 45,900 μm^2 total area (see "Materials and Methods").

TABLE 2. EXTRACELLULAR (INULIN) SPACE AND VASCULAR (BLOOD) SPACE IN LIVERS OF RATS FED CHRONICALLY WITH ETHANOL AND IN CONTROLS

	Control (B)*	Ethanol (7)	% change	p
Extracellular Space				
Body weight (gm)	117 ± 3	99 ± 4	−15	< 0.01
Liver weight (gm)	5.23 ± 0.17	6.29 ± 0.30	+20	< 0.01
Liver/body weight (gm/100 gm)	4.47 ± 0.12	6.33 ± 0.08	+42	< 10^{-6}
Total hepatic extracellular water (ml/100 gm body weight)	0.43 ± 0.02	0.47 ± 0.02	—	NS†
Hepatic extracellular water (ml/gm liver)	0.097 ± 0.01	0.075 ± 0.01	−22	< 0.01
Vascular Space				
Body weight (gm)	144 ± 5	131 ± 6	—	NS
Liver weight (gm)	6.28 ± 0.25	7.73 ± 0.45	+23	< 0.02
Liver body weight (gm/100 gm)	4.35 ± 0.11	5.91 ± 0.23	+36	< 10^{-6}
Total hepatic vascular space (ml/100 gm body weight)	0.16 ± 0.01	0.15 ± 0.01	—	NS
Hepatic vascular space (ml/gm liver)	0.036 ± 0.002	0.026 ± 0.002	−27	< 0.01

* Numbers of animals.
† NS, not statistically significant.

DISCUSSION

Data presented indicate that a marked compression of the sinusoidal area exists in the liver of patients with alcoholic disease, when compared to that in patients with normal biopsies or in patients with nonalcoholic liver disease. The difference in sinusoidal areas is so striking that this observation might be used as an additional criterion in the histological differentiation of alcoholic liver disease from that of other etiologies, especially chronic hepatitis.

We have previously shown that hepatocellular enlargement induced by chronic alcohol consumption leads to a compression of the extracellular space in the liver, and we have hypothesized that this compression could increase the resistance to blood flow, thus contributing to portal hypertension (11). However, our previous studies have not included the measurement of hepatic blood space in animals or a determination of the actual sinusoidal areas in humans. The present data were in line with our earlier postulate, in that chronic alcohol consumption was observed to result in a reduction in the hepatic vascular compartment in animals and in a striking decrease in the observable sinusoidal area in patients with alcoholic liver disease. These observations strongly suggest that in alcoholic liver disease portal hypertension is unlikely to be of postsinusoidal origin, but agree with the postulate that the increase in liver resistance to portal blood flow may have a sinusoidal origin (1, 2).

We observed a strong inverse relationship between sinusoidal area and portal pressure for relative sinusoids areas smaller than 900 μm^2. Such a correlation, however, was not observed in livers presenting larger sinusoids, thus suggesting that the sinusoids must be compressed by about 80% before they become important contributors to increases in portal pressure. Previously, we have also reported that hepatocytes must also exceed a threshold volume before affecting portal pressure (10, 11). It is conceivable that a correlation between two factors does not necessarily indicate causality between them. Nevertheless, it is of interest that in rats where chronic alcohol treatment results in hepatocyte enlargement without hepatitis or cirrhosis, those animals with very large hepatocytes have a reduction in the hepatic extracellular space and elevated intrahepatic pressures (9). We have also shown that hepatocyte enlargement produced isometrically in perfused rat livers results in a decrease in hepatic extracellular space and in an increase in both portal pressure and hepatic resistance (25). These results support a causal relationship between hepatocyte enlargement, compression of the sinusoids and portal hypertension.

Classically, alcoholic liver disease has been categorized as fatty liver, alcoholic hepatitis and cirrhosis. The small number of patients without cirrhosis in the present study did not permit the comparison of cell sizes and sinusoidal area within these categories. However, in previous studies, we have observed that in alcoholic patients, both

The International Journal of the Addictions, 16(4), 749-758, 1981

Clinical Note

Phencyclidine Ingestion: Drug Abuse and Psychosis

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Abstract

Phencyclidine (PCP) is a popular illicit drug often misrepresented as some other hallucinogenic substance and distributed in widely varying dosage forms and strengths. Users of hallucinogenic drugs may present with unintentional PCP overdoses. Toxicological laboratory analyses are essential to establish the diagnosis. In nine admitted overdose patients, the consciousness level ranged from alert to comatose on presentation, and all showed a prolonged recovery phase with agitation and toxic psychosis. Severe behavior disorder, paranoid ideation, and amnesia for the entire period of in-hospital stay are characteristic. In very high dose patients, shallow respiratory excursions and periods of apnoea and cyanosis coincided with generalized extensor spasm and spasm of neck muscles. Excessive bronchial secretions, gross ataxia, opisthotonic posturing, and grimacing occur. PCP toxic psychosis should be considered in drug-abusing patients presenting with schizophrenic-like symptoms, psychosis, or other bizarre behavior, whether or not they admit to taking PCP.

INTRODUCTION

Phencyclidine is a drug better known as PCP, a designation which is derived from its chemical name 1-(1-phenylcyclohexyl)piperidine. It is structurally related to the clinically useful anaesthetic agent ketamine and was introduced into clinical trials in the late 1950s as an intravenous anaesthetic with potent analgesic activity (Greifenstein and DeVault, 1958; Luby et al., 1959; Meyer et al., 1959). Despite the findings that it was an effective anaesthetic agent for superficial surgery (Greifenstein and DeVault, 1958), clinical testing was discontinued in 1965 because of a high incidence of adverse effects including a complex spectrum of sensory and cognitive effects characterized by alteration in body image with feelings of depersonalization, delusional and illusional experiences, a sense of isolation sometimes associated with intensification of dependency feelings, disorganization of thought, drowsiness, apathy, and euphoria. Repetitive motor behavior, anxiety, and depression were encountered occasionally. The duration of these effects after PCP anaesthesia ranged from a few hours to 4 d, and patients generally experienced amnesia for events which occurred after they regained consciousness (Greifenstein and DeVault, 1958). The effects of an acute dose of PCP have been likened to a sensory deprivation syndrome (Luby et al., 1959; Meyer et al., 1959).

In 1967 a PCP-containing tablet known as the "PeaCe Pill" appeared in San Francisco and within a year this drug was widely available in the eastern United States under the name "hog" (Londgren et al., 1969). Since that time

it has become a common drug on the illicit market throughout North America, being found frequently in material alleged to contain some other drug(s) (Baselt et al., 1972; Brown and Malone, 1973; Marshman and Gibbins, 1970; Schnoll and Vogel, 1971). The drug is therefore often ingested unintentionally.

During the past 4 years our Emergency Department has seen numerous mild PCP intoxications. Patients presenting with mild impairment were observed in a quiet room in the Emergency Department and the "talking down approach" was very effective in controlling the manifestations of toxic psychosis. Diazepam (10 mg orally) was used to sedate some patients. The majority of patients were discharged. The minority of patients who did not respond over a period of 8 h to the above management were admitted. The nine admitted patients comprising this report can present (to the unsuspecting physician) a bewildering clinical picture easily misdiagnosed as a primary psychosis. The clinical characteristics and course of nine patients admitted for PCP overdose are presented in Tables 1, 2, and 3. PCP was detected qualitatively by gas chromatographic analysis in the urine and/or blood of all nine patients (Marshman et al., 1976), although only *three* patients reported use of this drug.

CASE REPORTS

Patient 2

A 33-year-old male allegedly took tetrahydrocannabinol (THC), 1 g, 2 d prior to presenting in a catatonic, mute state at another hospital's Emergency Department. On examination at the time of transfer to the Clinical Institute, he was found to be alert with roving eye movements but showed no nystagmus, normal pupils, decreased response to pain sensation, catatonic rigidity, increased deep tendon reflexes, and flexor plantar responses. His heart rate was 88/min, blood pressure was 140/90 mmHg, respiratory rate and temperature were normal, and he showed excessive salivation. The catatonic mute state lasted 4 h and was followed by a period characterized by staring into space, making clicking noises with his tongue, echolalia, inappropriate monosyllabic answers, and euphoria. He was confused, disorientated, agitated, and hallucinating; he frequently assumed bizarre postures and showed a short attention span and profound sleep disturbance. Urine and blood samples on admission were positive only for PCP. He was given diazepam (10 mg) intravenously, q6h.

By the sixth day he was orientated in time and place and his mental state had improved significantly. At the end of 9 d in hospital he had completely recovered and showed no evidence of toxic psychosis. He was amnesic for the entire period of in-hospital stay. Urine and blood remained positive for PCP for nine consecutive days.

Table 1

Patient	1	2	3	4
Age/sex	26/M	33/M	16/M	18/F
Drug use history	Hallucinogens, antidepressants, multiple street drugs	MDA, THC, mescaline, amphetamine, heroin, PCP	Heroin, THC, cocaine, hashish, LSD, PCP, diazepam	LSD, PCP, amphetamine
Drug allegedly taken prior to admission	LSD, mescaline, imipramine, amphetamine, LSD	THC (1 g)	PCP, heroin	PCP
Drug ingestion time prior to admission	2 d	2 d	2 d	3 d
Duration of toxic psychosis before complete recovery	3 d	9 d	2 d	5 d

Patient 8

A 20-year-old male allegedly took 31 tablets of THC as a suicidal attempt. Soon after this he became violent and then fell unconscious. He was immediately brought to the Emergency Department and on examination he was unconscious, responded to painful stimuli, and showed normal pupils, generalized rigidity, and normal plantar response. His blood pressure was 150/100 mmHg, heart rate was 120/min, temperature was normal, respirations were shallow with intermittent laryngeal spasms, apnoea, and cyanosis. Investigations on admission included: blood gases (room air), pH 7.32, PCO_2 50 mmHg, PO_2 88 mmHg, actual bicarbonate 25.5 mEq/L. Blood taken on admission was positive for phencyclidine. During the period of coma, he received oxygen and intravenous fluids. Approximately 5 h after his initial presentation he was drowsy and just able to repeat words said to him. Tone and rigidity increased intermittently. After 14 h he was awake with slurred speech, echolalia, copralalia, and decreased response to pain sensation. He manifested quiet periods alternating with periods of uninhibited, inappropriate behavior consisting of agitation, kicking, and thrashing around in bed, screaming and singing, confusion, disorientation, hallucinations, and paranoia. He would stuff his mouth with food until he vomited. General supportive care included maintenance of minimal stimuli so as to lessen agitation, hyperactivity, and violence. Hypersalivation and increased

Clinical Histories of Nine Cases of Phencyclidine Toxic Psychosis

5	6	7	8	9
17/M	18/F	18/M	20/M	18/M
Amphetamine, heroin, LSD, PCP	LSD, THC, marijuana, hashish, mescaline	LSD, amphetamine, heroin, marijuana, PCP	LSD, amphetamine, heroin, opium, marijuana	Marijuana, LSD, mescaline, THC
PCP (6 tablets)	Mescaline (5–7 g)	Cocaine, amphetamine	THC (31 tablets)	THC
3 d	1 h	3 d	1–2 h	5–6 h
4 d	4 d	8 d	11 d	13 d

bronchial secretions required frequent suctioning. Chlorpromazine (100 mg) orally, q6h, was given to control his psychotic behavior.

On the fifth day he was depressed and suicidal and over the next 2 to 3 d he continued to be destructive and irrational. Thereafter, gradual improvement was noted. By the tenth day his speech was slow and deliberate and although he was unable to initiate conversation, he was polite and cooperative. On the twelfth day he had completely recovered, showing no evident psychotic signs and chlorpromazine was discontinued. He remains amnesic to the entire period of toxic psychosis.

DISCUSSION

The wide discrepancy between the patients' description of drugs abused and his/her actual street drug use is consistent with the findings of various street drug analysis programs. For example, during the period 1971–1976 PCP was the drug most commonly encountered in the street drug analysis program of the Addiction Research Foundation of Ontario. The samples had been voluntarily submitted to the Ontario Addiction Research Foundation for qualitative analysis by people not associated with law enforcement. Approximately 22% of all drug-containing samples (N = 294) examined by the laboratory contained PCP; of these, 26% were combinations of PCP with some other drugs, commonly

Table 2

Clinical Manifestations of Nine Cases of Phencyclidine Toxic Psychosis

Patient	1	2	3	4	5	6	7	8	9
Agitation	+	+	+	+	+	+	+	+	+
Confusion and disorientation	+	+	+	+		+	+	+	+
Hallucinations		+			+	+	+	+	+
Delusions					+	+			
Staring into space		+	+	+			+	+	+
Short attention span	+	+	+						
Alterations in communicative ability:									
Difficulty in verbalizing						+	+	+	+
Slurred speech	+					+		+	+
Echolalia	+	+					+	+	+
Catatonic mute state		+	+						
Behavior disorder		+		+			+	+	+
Paranoid ideation	+				+	+		+	
Depression	+					+		+	+
Amnesia for period of psychosis		+	+	+			+	+	+

LSD. Only 11% of the PCP-containing samples were alleged by the submitting physician (or patient) to contain PCP; the remainder were described as THC, mescaline (or peyote), MDA (i.e., methylene-dioxyamphetamine), LSD, psilocybin, cocaine, and less frequently as some other drug or drug combination. Tablets, powders, and capsules in a wide range of colors were the most frequently encountered dosage forms, and in some cases it was evident that capsules intended for legitimate pharmaceutical preparations had been diverted or emptied of their original contents and PCP had been introduced for street sale. Some of the street drug preparations had the form of yellow brown gummy materials or crystalline chunks, forms which suggest "illicit" synthesis. Occasionally the drug was encountered in solutions or in admixture with mushroom material (either decaying or dried) or green leaf material, sometimes marijuana. Quantiative assays of a random sample of these products revealed PCP contents ranging from 2.2 to 9.9 mg for tablets and 0.4 to 81.0 mg for capsules.

With widespread availability and the variation in dose level of PCP, it is not surprising that the recent literature contains several clinical reports of acute states of intoxication associated with nonmedical ingestion of the drug, involving not only teenagers and adults (Burns et al., 1975; Eastman and Cohen, 1975; Kessler et al., 1974; Liden et al., 1975a, 1975b; Lin et al., 1975; Marshman et al., 1976; Reed et al., 1972; Reynolds, 1971; Stein, 1973; Tong et al., 1975) but also young children whose ingestion of the drug was accidental (Burns et al., 1975; Liden et al., 1975a, 1975b; Lin et al., 1975). Despite the "street" impression that PCP is a benign recreational chemical, several recent reports of PCP-associated deaths confirm its status as a drug of substantial risk (Burns et al.,

Table 3

Abnormal Physical Signs in Nine Cases of Phencyclidine Overdose

Patient	1	2	3	4	5	6	7	8	9
Level of consciousness on admission	Drowsy	Alert	Alert	Alert	Alert	Coma	Coma	Coma	Coma
Intermittent apnoea and cyanosis	+		+			+		+	+
Excessive bronchial secretions						+		+	+
Blood pressure on admission	140/100	140/90	140/90	130/90	130/90	190/100	130/90	150/100	140/90
Nystagmus	+					+	+		+
Visual disturbance						+			+
Ataxia	+		+			+	+		+
Catatonic signs		+	+						
Neck rigidity								+	
Generalized rigidity and opisthotonic posturing						+	+	+	
Grimacing and trismus						+	+	+	+
Athetotic movements	+						+		
Decreased response to pain		+				+	+	+	+
Autonomic changes:									
Hypersalivation		+	+				+	+	+
Lacrimation			+			+		+	

1975; Eastman and Cohen, 1975; Kessler et al., 1974; Lin et al., 1975; Reed et al., 1972; Reynolds, 1971). The clinical manifestations of PCP toxic psychosis seen in our patients (Table 2) are consistent with previous reports (Burns et al., 1975; Liden et al., 1975b; Stein, 1973; Tong et al., 1975). The abnormal physical findings (Table 3) in all nine cases are typical of intoxication with moderately high (Patients 1-5) to very high (Patients 6-9) doses of PCP.

When present, coma lasted 4-6 h, and two of these patients had shallow respirations during the period of coma. Periods of apnoea and cyanosis occurred which coincided with neck muscle spasms (including laryngeal spasms) and generalized extensor spasms. Pooling of excessive bronchial secretions interfered with normal ventilation in unconscious patients, but in alert patients these secretions were easily expectorated and constant spitting was a characteristic feature.

Even in the presence of a normal respiratory rate, PCP overdose patients must be closely watched in the first 12-18 h for apnoea and cyanosis which may be associated with localized or generalized muscle spasm. Unless the history suggests very recent ingestion of a large number of tablets or capsules, gastric lavage is contraindicated in the alert patient as it may induce laryngeal spasm and aspiration of emesis. Respiratory acidosis, which occurred in two of the nine cases, was treated by adequate ventilation and by frequent suction of excessive secretions. Intubation and ventilatory assistance were not indicated in any of our cases.

Nystagmus, transient photopsia and blurred vision, gross ataxia, and other motor system abnormalities (Table 3) observed in these patients were consistent with previous reports (Burns et al., 1975; Eastman and Cohen, 1975; Liden et al., 1975a, 1975b; Stein, 1973; Tong et al., 1975). Opisthotonic posturing and generalized rigidity were present in three patients who were comatose, but seizures did not occur (cf. Burns et al., 1975; Liden et al., 1975a). Although opisthotonus has been previously noted in an adult patient (Liden et al., 1975a), it has been more commonly reported in children (Burns et al., 1975; Liden et al., 1975a, 1975b). Decreased response to pain was observed in most patients during the alert state.

Our treatment approach in all patients support the observations that interaction with staff causes exacerbation of the drug-induced problems (Stein, 1973; Tong et al., 1975) and that avoiding even minimal stimuli to the patient lessens severity of the toxic psychosis. Patients uncontrolled by symptomatic treatment should receive medication. Oral or intravenous diazepam was useful in reducing agitation and muscle spasm in Patients 2, 4, 6, and 7. Chlorpromazine was used in the management of more severely psychotic cases (Patients 5, 8, and 9).

Phencyclidine is a commonly available street drug frequently mislabeled as some other substance and marketed in a wide range of doses. The staff in

Table 3

Abnormal Physical Signs in Nine Cases of Phencyclidine Overdose

Patient	1	2	3	4	5	6	7	8	9
Level of consciousness on admission	Drowsy	Alert	Alert	Alert	Alert	Coma	Coma	Coma	Coma
Intermittent apnoea and cyanosis	+		+			+		+	+
Excessive bronchial secretions						+		+	+
Blood pressure on admission	140/100	140/90	140/90	130/90	130/90	190/100	130/90	150/100	140/90
Nystagmus	+					+	+		+
Visual disturbance						+			+
Ataxia	+		+			+	+		+
Catatonic signs		+	+						
Neck rigidity								+	
Generalized rigidity and opisthotonic posturing						+	+	+	
Grimacing and trismus						+	+	+	+
Athetotic movements	+						+		
Decreased response to pain		+				+	+	+	+
Autonomic changes:									
Hypersalivation		+	+				+	+	+
Lacrimation			+			+		+	

Emergency Departments should be trained to consider PCP toxicity in patients presenting with schizophrenic-like symptoms, delirium, psychosis, or in fact in a young drug user with any form of bizarre behavior. The slow recovery is largely the result of slow elimination of PCP (half-life approximately 15 h) (Marshamn et al., 1976). Toxicological laboratory findings were particularly valuable in facilitating diagnosis when the patient presented with psychosis and/or abnormal neurological and systemic manifestations.

ACKNOWLEDGMENTS

We are indebted to Miss Naomi Hunchuck and Miss Cathy Van Der Giessen for their help in the preparation of this article.

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SCIENTIFIC SECTION

REVIEW ARTICLE

Use of drugs with dependence liability

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The term addictive as used by the popular press frequently confuses the more precise concepts of acute and chronic tolerance, physical dependence and withdrawal, and psychologic dependence. Serious physical dependence on psychoactive drugs is rare and is easily managed. In contrast, psychologic dependence, the most important reason for persistent drug use, is much more common and is difficult to treat. Some tactics are available — for example, confrontation and discussion with the patient about how a drug is not going to be effective over long periods. Treating the symptom of a complex problem should, of course, not be expected to solve the problem. The most important tactic is to prescribe dependence-associated drugs only when clearly indicated, when the problem is responsive to drug therapy and for the shortest period necessary, without the option for renewing the prescription. Many problems related to drug use long after the period of expected benefit is past can be avoided by far more restrictive drug prescribing. Barbiturates and nonbarbiturate sedative hypnotics (e.g., ethchlorvynol, glutethimide, meprobamate, methaqualone and methyprylon) should not be prescribed for insomnia, acute reactive anxiety, chronic anxiety neurosis or depressive illnesses, since the safer and equally effective benzodiazepines, which are less associated with dependence, are available.

Dans la presse profane l'expression toxicomanogène confond souvent les notions plus précises de tolérance aiguë ou chronique, d'assuétude et de sevrage, et dépendance psychique. Une assuétude sérieuse aux médicaments psychoactifs est rare et se traite facilement. Par opposition, la dépendance psychique, la cause la plus importante d'utilisation persistante des drogues, est beaucoup plus fréquente et difficile à traiter. Quelques tactiques sont disponibles — comme, par exemple, de susciter une confrontation et une discussion avec le patient pour lui expliquer comment un médicament ne pourra être utile pendant de longues périodes. On ne devrait pas s'attendre à ce que le traitement des symptômes d'un problème complexe puisse résoudre le problème. La tactique la plus importante consiste à ne prescrire les médicaments capables de produire de la dépendance que quand ils sont parfaitement indiqués, quand le problème répond au médicament et pour la plus courte période nécessaire, sans possibilité de renouveler la prescription. Plusieurs problèmes reliés à l'utilisation des médicaments pour des périodes outrepassant la durée prévue d'effet bénéfique pourraient être évités par une prescription plus restrictive des drogues. Les barbituriques et les sédatifs non barbituriques (e.g., ethchlorvynol, glutéthimide, méprobamate, méthaqualone et méthyprylone) ne devraient pas être prescrits pour l'insomnie, l'anxiété aiguë réactionnelle, la névrose d'angoisse chronique ou la dépression, alors que les benzodiazépines, des médicaments plus sûrs, tout aussi efficaces et moins associés à un état de dépendance, sont disponibles.

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Knowledge in two areas is important for the proper use of drugs with dependence liability: first, an exact appreciation of the meanings of terms often applied to "addictive" drugs (acute and chronic tolerance, physical dependence and withdrawal, and psychologic dependence); and second, the clinical pharmacology of psychotropic and analgesic drugs, including their proven indications, proven duration of optimal efficacy, dosages, toxicity and true physical liability.[1] In each of these areas there is often considerable misinformation. For example, oxycodone (as in Percodan®) is commonly prescribed as a drug with a lesser risk of producing dependence than other opiates, yet the risk of physical dependence with this drug is no different from that of morphine or meperidine.

Terms applied to addictive drugs

Acute tolerance

Acute tolerance is the adaptation to a drug's effect after a single administration of the drug. For example, after alcohol or diazepam ingestion a greater effect may be observed during the ascending phase of the drug's concentration–time curve than at the peak concentration or during the descending phase.[2] Such acute receptor site tolerance or adaptation to the drug may be viewed as a therapeutic advantage or as an unwanted side effect. The drowsiness and sedation produced by benzodiazepines used

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for preoperative sedation, endoscopic procedures[3] or treatment of seizures are beneficial therapeutic effects, but when benzodiazepines are used as anxiolytic agents the acute drowsiness is an unwanted side effect.[3]

Chronic tolerance

Long-term administration of barbiturates, benzodiazepines, ethanol, nonbarbiturate sedative hypnotics and opiates is associated with decreased effects of the same dose or a need to administer larger and larger amounts of the drug to obtain the same pharmacologic effects. Early in therapy repeated ingestion of diazepam may lead to excessive sedation. If treatment is continued at the same dose the sedation abates even though the blood concentrations of diazepam and its active metabolite, desmethyldiazepam, greatly exceed those measured during the early part of therapy, when drowsiness was most evident.[4] Similar results have been observed with single- and multiple-dose flurazepam therapy.[5] Another practical consequence of the development of chronic tolerance has recently been illustrated during the use of diazepam for gastroscopy. The intravenously administered dose of diazepam necessary to produce sufficient relaxation for passage of a gastroscope varied 22-fold among patients, and those who had been using benzodiazepines required larger doses for relaxation than those who had not.[2] Chronic tolerance or adaptation at the drug's receptor site exceeds the relatively small changes in drug half-life or clearance caused by enzyme induction during long-term administration.[1]

Physical dependence and withdrawal

Physical dependence is a physiologic state of adaptation to a drug following the development of chronic tolerance that results in a characteristic set of withdrawal symptoms ("abstinence syndrome") when administration of the drug is stopped.

Physical withdrawal is the unmasking of the adaptive neurophysiologic or biochemical changes, or both, that have occurred during long-term drug administration. The withdrawal reaction presents as a hyperadrenergic state, with anxiety, agitation, tachycardia, mild hypertension, hyperacuity of all the senses, hyperreflexia and decreased seizure threshold.[4] The withdrawal reaction is most prominent for drugs that can be taken frequently, are associated with extensive adaptation (tolerance), are taken by a route whereby absorption is rapid and produce a response that is closely associated with the drug taking. In addition, the drug must remain in the body long enough to permit the adaptation associated with chronic tolerance to develop (as with alcohol). On the other hand, the drug must be eliminated from the body at a rate greater than that of the corrective biochemical and neurophysiologic processes that reverse the drug-induced changes associated with chronic tolerance. All these criteria will determine the severity and likelihood of physical withdrawal in relation to the development of chronic tolerance.

The physical dependence syndrome can be treated by readministration of the same drug or a drug with similar pharmacologic properties (e.g., barbiturates and alcohol). What contribution the need to prevent the withdrawal reaction plays in persistent drug taking is not known and has certainly been overemphasized. The requirement to take a drug to prevent the symptoms of withdrawal appears late, only after physical and psychologic dependence are well established.[3] The classic characterization of the alcohol, barbiturate or opiate addict is often generalized to all abusers of these and other agents without any firm basis. There is considerable evidence that persistent drug taking is the result of many more subtle factors than the need to prevent withdrawal symptoms.[4]

Psychologic dependence

Psychologic dependence can be associated with repeated consumption of virtually any drug, and is characterized by little or no tendency to increase the dose of the drug and by satisfaction of a psychic drive without necessarily the development of physical dependence (Table 1). Other terms used for psychologic dependence are behavioural, psychic or emotional dependence and habituation. In the management of patients the predominant role of psychologic dependence must always be considered. Evidence for this comes from two principal sources. First, many patients who have taken "addictive" drugs for long periods do

Table 1—Drugs with physical or psychologic dependence liability or both

- Central nervous system stimulants
  - Amphetamines
    - Dextroamphetamine sulfate (e.g., Dexedrine®)
    - Methamphetamine hydrochloride (e.g., Methedrine®)
  - Amphetamine congeners
    - Chlorphentermine hydrochloride (e.g., Pre-sate®)
    - Diethylpropion hydrochloride (e.g., Tenuate®)
    - Mazindol (Sanorex®)
    - Methylphenidate hydrochloride (e.g., Ritalin®)
    - Phentermine (e.g., Ionamin®)
- Central nervous system depressants
  - Barbiturates
    - Amobarbital (e.g., Amytal® sodium)
    - Barbital sodium
    - Pentobarbital sodium (e.g., Nembutal® sodium)
    - Phenobarbital sodium (e.g., Luminal®)
    - Secobarbital sodium (e.g., Seconal® sodium)
  - Nonbarbiturate sedative hypnotics
    - Chloral hydrate (e.g., Noctec®)
    - Ethchlorvynol (e.g., Placidyl®)
    - Glutethimide (e.g., Doriden®)
    - Meprobamate (e.g., Miltown®)
    - Methaqualone hydrochloride (e.g., Mequelon®)
    - Methyprylon (Noludar®)
  - Benzodiazepines
    - Chlordiazepoxide hydrochloride (e.g., Librium®)
    - Clonazepam (e.g., Rivotril®)
    - Clorazepate dipotassium (Tranxene®)
    - Diazepam (e.g., Valium®)
    - Flurazepam hydrochloride (Dalmane®)
    - Lorazepam (e.g., Ativan®)
    - Oxazepam (e.g., Serax®)
    - Triazolam (Halcion®)
- Analgesics
  - Non-narcotic
    - Acetylsalicylic acid
  - Narcotic
    - Alphaprodine hydrochloride (Nisentil®)
    - Anileridine (e.g., Leritine®)
    - Codeine
    - Hydrocodone bitartrate (e.g., Hycodan®)
    - Hydromorphone (Dilaudid®)
    - Levorphanol tartrate (Levo-Dromoran®)
    - Meperidine hydrochloride (e.g., Demerol®)
    - Methadone
    - Morphine
    - Oxycodone (e.g., Percodan®)
    - Pentazocine (e.g., Talwin®)
    - Propoxyphene napsylate (e.g., Darvon-N®)

not manifest any withdrawal signs or symptoms. Second, rehabilitation programs for individuals truly physically dependent that have relied on pharmacologic agents (e.g., methadone) as the only treatment modality have consistently failed. *Psychologic dependence is the most important reason for persistent drug use by patients.* The etiology of such dependence is complex, often being rooted in a matrix of familial, social and economic problems. Presumably the stimuli to take drugs are subtle, acquired, conditioned internal and external cues.

Prevention of physical and psychologic dependence

General principles

The reasons drugs are prescribed and individuals persist in taking them in situations in which therapeutic advantage is unclear are complex. Overall social traditions and attitudes do not discourage the use of drugs for coping with the stress and strain of normal life. Medical and popular advertisers of substances as diverse as alcohol, cigarettes and prescription and nonprescription drugs frequently promote the notion that the use of these agents is associated with lifestyles that are variously portrayed as glamourous, seductive or necessary, or all three. In a far more subtle way newspapers, by simply reporting patterns of drug use and abuse, may promote experimentation and more widespread drug use.

Should the widespread use of psychoactive agents cause concern? Excessive prescribing or use of any drug when it is not needed must be associated with increased health care costs and an increased frequency of adverse effects. For example, despite the safety and low physical dependence liability of benzodiazepines, these drugs share disadvantages with barbiturates.[8] They produce drowsiness and "mental clouding", interact with other psychotropic drugs, including alcohol, and can produce in some individuals psychologic dependence even after short-term use. The long-term behavioural and societal consequences of persistent administration of drugs that subtly modify central nervous system function is not known. Drug use may substitute for the learning of personally effective and flexible ways of solving problems and adapting to new stressful situations.

The most important tactic available to the physician is to prescribe dependence-associated drugs only when clearly indicated, when the problem is responsive to drug therapy and for the shortest period necessary, without the option for renewing the prescription. Of course, these tactics apply to all drug prescribing, but they may be less consistently applied when the problem is not readily responsive to the drug and the drug is "safe".

Patients with acute reactive anxiety, occasional insomnia, reactive depression or minor pain often do not require prescribed drugs since these common experiences are transient and not particularly serious. Persistent problems require full investigation and treatment directed at the cause rather than the symptom. In this respect the use of marital counsellors, psychologists and other community-based individuals, facilities and programs related to problem solving needs to be incorporated more commonly into the treatment choices of physicians.

Specific drugs (Table I)

Amphetamines and their analogues: Under the Canadian Food and Drugs Act, amphetamines and their analogues are "designated" drugs and may only be prescribed for narcolepsy, hyperkinetic disorders in children, mental retardation (minimal brain dysfunction), epilepsy, parkinsonism and hypotensive states associated with anesthesia. These restrictions have fairly effectively decreased the previous widespread abuse.[9] The effectiveness of the legislation presents a strong argument that physicians played a major role as the cause of the widespread misuse. These agents and their congeners (e.g., methylphenidate) should not be prescribed to patients unknown to the physician. Drugs such as methylphenidate and diethylpropion have recently been placed on Schedule G of the Food and Drugs Act. Their use will decrease as a result of this change.

Barbiturate and nonbarbiturate sedative hypnotics: Secobarbital- or amobarbital-dependent individuals can have a severe, life-threatening withdrawal reaction, in part because the plasma half-lives of the drugs are about 25 hours and physical dependence is unmasked when drug use is abruptly stopped. Phenobarbital is eliminated more slowly (half-life 87 hours) and is seldom associated with an important withdrawal reaction.[10] Other barbiturates and nonbarbiturate sedative hypnotics can produce physical dependence and have a narrow margin of safety. *Barbiturates and nonbarbiturate sedative hypnotics (e.g., ethchlorvynol, glutethimide, meprobamate, methaqualone and methyprylon) should not be prescribed for insomnia, acute reactive anxiety, chronic anxiety neurosis or depressive illnesses, since the safer and equally effective benzodiazepines, which are less associated with dependence, are available.* Individuals currently taking these drugs should have their management reviewed to establish the need for continued therapy.[1]

Benzodiazepines: Numerous benzodiazepine derivatives are available in Canada. These drugs are all similar structurally and in their pharmacologic actions. In general, a generic preparation of chlordiazepoxide or diazepam will suffice for most patients with insomnia, acute reactive anxiety, chronic anxiety neurosis or alcohol withdrawal. Many such patients do not require even a short course of drug therapy. Other conditions for which benzodiazepines are indicated include continuous seizures (e.g., diazepam); petit mal "absence attacks" (e.g., clonazepam); neuromuscular disorders, including backache and muscle trauma, cerebral palsy, tetanus and stiff man syndrome; and a variety of psychiatric problems in which their efficacy is unclear — for example, toxic psychosis, anxiety–depression and phobic disorders.[2]

For the physician prescribing benzodiazepines the following guidelines are useful:

- Determine the cause of or precipitating factors in insomnia and anxiety, and treat the primary problem (Table II). Decide if the drug is necessary.

- Prescribe chlordiazepoxide or diazepam rather than other products. Agents marketed specifically for insomnia (e.g., flurazepam [Dalmane®]) are not proven to have clinically important advantages.
- Older patients should start with half the usual dose. When morning drowsiness is a problem an agent with a short half-life, such as oxazepam (half-life 6 hours), should be considered. Similarly, a newly marketed benzodiazepine, triazolam (Halcion®), could be considered when a short duration of action is desired. The relative role for this new drug is not clear at present. Both oxazepam and triazolam are much more expensive than nonproprietary diazepam and chlordiazepoxide.
- Exercise special caution in prescribing standard doses for patients who are small or over 60 years of age or have liver disease.
- Warn the patient of the unpredictable and potentially serious interactions of benzodiazepines with alcohol, cold tablets, antihistamines, other tranquillizers, hypnotics and analgesics. Emphasize that such interactions are more predictable at the time drug therapy is started.
- Do not provide more than 2 weeks' supply of the drug initially, and do not allow for automatic prescription renewals. Reassess for continuation of medication, improvement of symptoms and maintenance of other supportive measures.

Non-narcotic analgesics: Table III summarizes recommendations that arise from reviewing analgesics with respect to effectiveness, toxicity and relative cost. Of particular note is the exclusion of combinations of acetylsalicylic acid with caffeine and small amounts of codeine (less than 30 mg). These agents are no more effective than acetylsalicylic acid or acetaminophen alone.[9] Propoxyphene alone and in combination is not included because of its questionable efficacy,[9] relatively high cost[10] and apparent risk of lethal overdose.[11]

Indomethacin, naproxen, phenylbutazone, oxyphenbutazone, other nonsteroidal anti-inflammatory drugs and combination products containing small amounts of acetylsalicylic acid must be avoided in individuals allergic to acetylsalicylic acid since these drugs all cause cross-reactions.[12] Acetaminophen is a reasonable alternative. Individuals who claim intolerance to a wide range of drugs may be sensitive to tartrazine, a frequently used colouring agent ubiquitous in medications.[13]

Narcotic analgesics: There are a large number of analgesic-containing narcotics (or opiates) available. Unfortunately the names are sufficiently confusing that few practitioners are likely to be able to keep them straight. Drugs such as hydrocodone (Dilaudid®), levorphanol tartrate (Levo-Dromoran®), Pantopon® (a mixture of morphine and all the opiate alkaloids) and oxycodone (Percodan®) can produce physical dependence and differ from meperidine and morphine only in potency.[11] Opiates administered orally are often not as effective as those administered parenterally because a large proportion of the ingested opiate is metabolized on the drug's "first pass" through the liver.[14] The dose of morphine required parenterally is approximately one seventh of the oral dose.

Narcotic analgesics are indicated for the control of moderate to severe acute pain, such as postoperative pain and chronic pain of terminal illness. Non-narcotic analgesics may be combined with narcotic analgesics to produce an additive effect. Chronic pain of terminal illness is best controlled by parenteral administration of medication at regular intervals that are sufficiently short (3 to 4 hours) to prevent the recurrence of pain. The usual dose range of morphine required for adequate pain relief is 5 to 10 mg every 4 hours; in small or elderly patients as little as 2.5 mg may be adequate. The initial drowsiness associated with the introduction of high-dose narcotic therapy is temporary, lasting for 48 to 72 hours. After this period the dose must be titrated for the individual patient; this is facilitated by the fact that the pain relief threshold is lower than the sedation threshold. In the terminally ill patient, tolerance and dependence on narcotics is not important. Excessive and unrealistic concern about the danger of addiction in patients with pain typically biases toward undertreatment with narcotics. Often an increase in the dosage requirements heralds a change in the disease status rather than tolerance.[11]

Management of physical dependence

Any drug, if perceived by a patient to be essential, can be associated with "dependence". However, such dependence is not synonymous with physical dependence. The management of physical dependence is relatively simple.[11,15]

Table II—Tactics for selection of a benzodiazepine

Indication	Drug	Typical dose*
Insomnia with day-time anxiety	Chlordiazepoxide or diazepam	25 mg at bedtime 5 mg at bedtime
Acute anxiety	Chlordiazepoxide or diazepam	25 - 50 mg 5 - 10 mg
Chronic anxiety	Chlordiazepoxide or diazepam	25 - 100 mg daily 5 - 40 mg daily†
Simple insomnia	Oxazepam	15 mg, increasing to 30 mg and then 45 mg, at bedtime

*Elderly or debilitated patients should receive lower initial doses — for example, 15 mg of oxazepam, 2 to 5 mg of diazepam or 10 mg of chlordiazepoxide.
†Initiate treatment with the lowest dose and increase the dose gradually according to effectiveness and side effects. The daily dose may be administered in two divided doses or even one dose, usually at night.

Table III—Guidelines for symptomatic relief of pain

Drug	Proven effective dose when given every 4 to 6 hours
Initial choice Acetylsalicyclic acid or acetaminophen	650 mg orally 650 mg orally
If above ineffective add codeine	32 mg, increasing to 65 mg orally
If above ineffective Codeine	120 mg orally
If above ineffective Parenteral narcotics (e.g., meperidine or morphine)	

The cessation of drug consumption in the physically dependent individual results in a drug withdrawal reaction, which is usually a self-limiting disturbance, seldom lasting longer than 2 weeks.[9] Various studies have been done to determine levels of drug consumption that are associated with sufficient physical dependence to result in a symptomatic withdrawal reaction. However, the general applicability of such studies is confounded by interpatient variations, the special populations studied and a failure to take the kinetics of the various drugs into consideration. As a consequence, only a general guideline can be given — namely, any patient who is taking on a long-term basis *three times the maximum recommended dose of a barbiturate (except phenobarbital), nonbarbiturate sedative hypnotic or narcotic analgesic* should be considered at risk for clinically important signs or symptoms of withdrawal. The spectrum of withdrawal potentially ranges from mild to life-threatening, and the severity cannot be predicted for a particular patient beforehand. Opiate withdrawal is usually not life-threatening, contrary to the perceptions of "addicts" and the popular press. On the other hand, alcohol,[20] barbiturate and nonbarbiturate withdrawal[11] can be life-threatening and may need to be managed in hospital.

Barbiturate and nonbarbiturate sedative hypnotics

Physical withdrawal is managed by giving phenobarbital (10 mg/ml) intravenously in a dose of 0.03 to 0.04 mg/kg per minute until withdrawal signs are controlled.[11] Vital signs, level of consciousness, short-term memory, nystagmus, dysarthria, coordination, ataxia and tremor are assessed hourly during the infusion. The infusion is terminated when the patient is able to sleep but is easily arousable. This loading with phenobarbital is a safe and efficacious treatment of barbiturate and mixed sedative withdrawal; no further treatment is required in most cases since phenobarbital has a long half-life, which ensures a slow decline in serum concentration. A loading dose of phenobarbital equal to or less than 120 mg generally indicates that the patient is not physically dependent and is unlikely to have a physical withdrawal reaction. Occasionally supplemental doses of phenobarbital given orally may be required in a patient with rapid metabolism of the drug, resulting in rapid decline of the serum phenobarbital concentration and development of signs of withdrawal.[11] Oral administration of the loading dose may be more convenient in some patients; 120 mg of phenobarbital can be given by mouth every hour until the desired clinical end point has been reached. Through titration of the drug's dose the physician can reduce his or her uncertainty concerning the adequacy of treatment and effectively manage drug seeking by the patient: clinical fluctuations during physical withdrawal are minimized, so that drug-taking behaviour is not reinforced. The total loading dose and the peak serum concentration may be objective indicators of the central nervous system's tolerance to the drug abused.

Benzodiazepines

Occasionally patients who have been taking benzodiazepines for a long time or who have been taking extraordinarily high doses manifest, when they stop taking the drug, symptoms and signs including insomnia, agitation, diaphoresis, decreased appetite, seizures, twitching, recurrence of depression and exacerbation of psychosis.[4] Case reports, unfortunately, have not distinguished psychologic dependence from physical dependence or the reappearance of the symptoms of anxiety, depression or insomnia for which the drug was prescribed. In addition, many of the allegedly dependent individuals were using a variety of other drugs with a far greater likelihood of causing physical dependence. Furthermore, diazepam and chlordiazepoxide are so slowly eliminated that it is difficult to accept that the physical dependence could be unmasked. Unfortunately, to simply stop taking the drug is not sufficient since it does not provide an alternative problem-solving behaviour.

Most indications for benzodiazepines — those for which the efficacy of these agents is proven — necessitate only 2 to 4 weeks of therapy, and such short-term therapy is not associated with clinically important dependence. At the commencement of a trial of benzodiazepine therapy the desired therapeutic goal and the duration of therapy should be identified.

Narcotics

The estimated number of users of narcotics in Canada increased over the period 1965 to 1974 from 4655 to 12 194 according to the bureau of dangerous drugs of the Department of National Health and Welfare.[21] Physicians must be extremely wary of patients not well known to them who seek analgesics by name (e.g., Dilaudid® and Percodan®). Such patients frequently have an "opiate-seeker's disease" such as Crohn's disease, kidney stone, back injury, migraine or "addiction". The physician must insist on objective evidence for the problem such as a previous medical report documenting the presence of the problem. Needle tracks should be looked for while the blood pressure is taken. The patient should be asked if he or she has received a prescription for a narcotic from another practitioner within the last 30 days (see sections 3[3] and 40 of the Narcotic Control Regulations).

Do not give even a small prescription. These patients usually go to many doctors. The physician can most effectively manage patients professing addiction by offering to refer the patient to a methadone clinic or to a hospital for management of withdrawal. A firm, consistent and sympathetic approach is essential since the ability of multidrug users to manipulate physicians will challenge the most astute, resolute and capable clinician.

Methadone is a long-acting opioid used as a substitute for abused opioids of shorter action. Methadone maintenance therapy is recommended when repeated attempts at withdrawal from opioid abuse are unsuccessful in persons with long-term narcotic dependence and entrenchment in the addict lifestyle.[22] No study has proven the efficacy of long-term methadone maintenance therapy without ex-

tensive counselling and other components of a rehabilitation program. Methadone withdrawal is generally recommended for persons with short-term opioid dependence and those under 18 years of age; motivation to become free of opioids is essential. To establish that a patient is physically dependent on a narcotic the physician should administer intravenously naloxone, 0.2 mg initially, then 0.4 mg in 5 minutes. The signs of narcotic withdrawal start in 1 to 2 minutes, the peak effect lasts 7 to 10 minutes and the duration of effects is 30 minutes. Morphine, 5 to 15 mg, can be given if the reaction is severe." If no signs develop, the patient is not physically dependent. In addition to the naloxone test, an assessment of psychologic dependence and motivation to stop taking the drug(s) is required. If the patient is physically dependent 10 to 20 mg of methadone is given initially and then in 12 hours, to a maximum of 30 to 40 mg/d. Similar doses are recommended for the management of narcotic addicts before and after surgical procedures. Only authorized individuals may prescribe methadone.

Management of psychologic dependence

For most patients, including those with an element of physical dependence, the main therapeutic challenge is to reduce the psychologic dependence upon drugs, identify the causes and consequences of the dependence and design a treatment plan that deals with each of these areas. Since the causes and consequences of drug dependence are multiple and are complex, the therapeutic strategies are accordingly multiple and, ideally, should be adapted to the individual patient. Nevertheless, it is usually difficult to provide the psychologically dependent individual with an alternative pattern of behaviour for problem-solving or support because the existing behaviour pattern has evolved over many years.

It is usually essential to have the individual stop taking the medication. This can be done in a variety of ways — for example, by tapering the supply. Such an approach often ends up as a time-consuming and usually pointless series of negotiations between patient and physician. One of the most successful tactics is to confront the problem directly. "Mrs. Smith, I am concerned that the sleeping pills you are taking aren't working anymore, and I think we should talk about having you stop taking them." Patients frequently acknowledge that the medication is taken out of habit rather than for its therapeutic effect. Many have thought about the problem and are relieved when the topic is approached. There is no scientifically valid evidence that patients will stop going to a doctor when prescriptions for such drugs are no longer given.

The next step is to stop giving the patient the prescription and to maintain a sympathetic but firm hand on the situation. In the discussion of the problem with the patient a full review of the level of alcohol consumption is important. Frequently alcohol is the most important substance of abuse. As few as four and six drinks per day

(one drink = one beer or 40 ml of spirits or 110 ml of wine) in women and men respectively can increase the risk of liver disease.

In the long course of increasing dependence on drugs there can be disintegration of many aspects of physical, mental and social function. The degree to which each type of function is affected must be assessed completely, and specific steps must be taken on the basis of the problems identified. Frequently personal encouragement, direct intervention or counselling, or a combination, is necessary because the patient's capacity to cope with the conventional demands of society is impaired. Psychotherapy should aim at replacing drug consumption by more effective problem-solving methods. Many communities have mental health, social service or self-help groups or assertiveness training programs that can be very helpful. In particular, groups that have focused on the special problems and requirements of women have become interested in this area. Since women much more commonly receive psychotherapeutic drugs, such a development is important.[34] Information about existing community groups for women can be obtained from local community information centres, provincial governments or the women's program directorate of the Secretary of State (15 Eddy St., Hull, PQ J8X 4B3). The federal government has published the "Directory of Canadian Women's Groups 1977", which is not generally available but can be obtained through the Secretary of State.

In general, few family practitioners are able to personally conduct the full range of support or counselling services that may be required. Psychiatric management may be necessary. However, coordination of the various treatment modalities may be carried out by the family physician or by another responsible person with interest and experience, such as a social worker, a psychiatrist or a public health nurse. The essential role of the coordinator is to be familiar with how to use the resources available in the community, be able to design and coordinate an overall treatment plan and be prepared to provide ongoing, long-term support to the patient. The most important thing that can be done by physicians is to be far more circumspect in the initiation and maintenance of therapy with psychotherapeutic drugs.

We are indebted to Cathy Van Der Giessen for her help in the preparation of this article and to Ms. M Holloway, pharmacy department, Toronto Western Hospital for her revisions of the tabular material.

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ALCOHOLISM

Alcoholism is a major problem in the country - urban and rural. Unfortunately, not enough importance has been paid to this problem. We are aware of the individual, family and social consequences, alcoholism is a problem for the family. It results in broken up families. In Bombay, one out of ten divorces are because of alcoholic husbands. We do not have sufficient data about the extent of the problem, especially in the unorganized and rural areas. It is estimated that there about 3 million alcoholics in the country (out of about 50 million Indians who consume alcohol). The Indian Council of Medical Research study in Bangalore, Delhi, Chiragaon and Rajkot showed that 10% of urban consumers of alcohol had become totally dependent on it. In the villages, the figure is even higher, going upto 30 - 40%. In industries, problem drinking, drinking leads to impaired work performance and decrease in productivity.

2. Alcohol Abuse is characterised by excessive use of alcohol which would result in impairment of occupational functioning. The economic costs of alcohol abuse are substantial, with loss of job, accidents, crime and other social problems. At the individual level, alcoholism leads to many diseases, including cirrhosis of liver and diseases of the nervous system; it is a risk factor in the causation of a number of diseases. Alcoholism causes extensive and severe dysfunction of the health, with an increased demand for health care services. The risk of death for those abusing alcohol or drinking heavily is two to four times that for the general population. Immoderate drinking accounted for a 10% loss of potential years of life.

3. Dependence: Social drinking is fairly common. If limited, there may not be a problem. But a time comes when the person becomes addicted to and dependent on alcohol. It is characterised by the development of tolerance, using increasing amounts to achieve the desired results and withdrawal symptoms occur when the person reduces or stops the intake of Alcohol.

Predisposing Factors: Some people are more prone to become problem drinkers. It has been said that there is an alcoholic personality - impulsivity, poor self-esteem, low ego strength during childhood and adolescence. Conduct disorders and childhood delinquency may predispose to alcoholism, as also minimal brain dysfunction. Alcoholism may be seen more commonly in certain families. Probably there are genetic factors also involved.

Precipitating Factors: Among the precipitating causes are loss of job, death of a spouse and situational changes. Improvement in income can sometimes lead to excessive drinking.

Alcoholism may be superimposed on a pre-existing psychiatric abnormality like depression or schizophrenia. In such cases, alcoholism may be periodic in nature and responds well to treatment of the primary disorder.

Intoxication: Acute intoxication is usually due to excessive quantities of alcohol being consumed over a short period (usually to blood levels are more than 200 mg per dl). The stage of intoxication may last even upto 12 hours after stopping drinking. That person may behave in disinhibited or violent manner and may have loss of memory for the period of intoxication (alcoholic blackout). In some people with pathological intoxication, the aggressive and other behavioural changes may occur after indulging in alcohol in quantities much smaller than the usual.

Withdrawal Symptoms: Stoppage of alcohol after prolonged drinking may result in withdrawal symptoms. These may be mild - tremors of the hands, nausea and vomiting, anxiety or irritability and insomnia. In severe forms, it may lead to convulsions (run fits or withdrawal seizures). The severest form results in delirium tremors, occuring within one week of the reduction or cessation of heavy drinking. The symptoms consist of insomnia, illusions, hallucinations (especially visual), restlessness and tremors of the hand. The person may have fever; dehydration may be present. There can be tachycardia, raised blood pressure and sweating. Disorientation, confusion and agressive behaviour may be seen. Pre-drinking factors in precipitating delirium tremors are pneumonia, head injury and liver disease.

Alcoholism in Industry: The adverse effects of problem drinking, on productivity, have been well recognised. The managements have become conscious of the problem and its social and economic consequences. Many programmes to help the employees give up excessive drinking have been instituted. Some of them have been successful and the work place, has often been used as an active force in the rehabilitation process.

Prevention: The emphasis will be on preventing excessive drinking before the problem arises. This will be mainly by education and awareness of the likelyhood of the problem. Courses will be conducted for the staff (along with their families and supervisory personnel if possible). The staff will also be trained to detect early signs and symptoms of problem drinking. Questionnires will be developed and administered to the staff.

Identification of problem drinkers: Problem drinking is often manifest by impaired work performance and absenteeism. Biochemical tests and self-rating questionnaires can reveal the problem and lead to early detection of the problem drinking. Alcohol-related disabilities are a major cause for medical consultations (with varied presentations of injury, sleeplessness, nausea and vomiting, etc). In such cases, a high degree of suspicion must be kept in mind, so that alcohol-related disabilities may be detected early intervening at a stage when the drinking behaviour is more amenable to treatment. Frank liver disorders, like cirrhosis of the liver and nervous disorders are late manifestations, when it would be more difficult to wean the person away from drinking.

A number of instruments and questionaires have been made to help in the diagnosis of alcoholism, among them being tast of the National Council of Alcoholism, U.S.A., the Michigan Alcoholism Screening Test and the Munich Alcoholism Test. They use the physical, social, interpersonal psychologic and laboratory criteria. There are many clinical indications of alcohol abuse.

One of the earliest indications of alcohol abuse is a growing concern or worry about personal drinking habits.

Laboratory findings: Enzyme studies can reveal alcohol abuse: serum gamma - glutamyl transpeptidase and serum aspartate aminotransferase are increased in alcoholics. The mean corpuscular volume of RBCs is raised in 70 - 95% of alcoholics. Serum High Density Lipoprotein Cholesterol level is raised in 50 - 60%. Increased concentration of serum bile acids may be a sensitive indicator of alcoholic liver disease.

Treatment: The treatment is multidimensional. The problem drinker must be motivated to seek help and further motivated to change the habit of drinking. Willingness to undergo treatment is important for the success of treatment. It is most successful in patients who have not reached an advanced stage of alcohol abuse.

Patient characteristics have a greater effect on the outcome than the kind of treatment given. The family should be involved in the treatment.

The treatment will be carried out by the consulting psychiatric service. The team will consist of

(i) Consultant psychiatrist
(ii) Clinical psychologist
(iii) Social worker

In induction, the Personnel Welfare Officer and other medical persons will be associated with the programme.

Comprehensive assessment: A good history (individual, family, occupational and social) is a requisite. The current status with respect to disabilities related to alcohol abuse must be determined. The psychosocial problems related to drinking and dependence on alcohol must be studied.

Counselling session: One or more counselling sessions may be beneficial. The assessment is reviewed with the patient and the interested family, emphasising the responsibility of the patient and the family to deal with the problem. The person, together with the family, should be helped to set goals with respect to alcohol use (abstaining from alcohol, etc), personal health, work and interpersonal relationships. The patient and members of the family should be given education material on alcohol.

Treatment of withdrawal symptoms: There will be need for treatment of withdrawal symptoms which could be expected to occur in an alcoholic. The general nutrition will have to be improved. Tranquilisers may help at this stage. Detoxification may be necessary. Psychotherapy (individual and group) may be required for the underlying emotional problems. Major psychiatric disorders, if present, will have to be dealt with appropriately. Behaviour and electrical aversion therapy may be useful.

Disulfiram: At later stages, disulfiram (Antabuse) may have to be given, after informing the person of the consequences of taking alcohol. Extremely unpleasant systemic reactions can arise after the ingestion of even small amounts of alcohol. Disulfiram is given in a dose of 200 - 500 mg daily (initial dose may be high, reducing to about 100 - 200 mg daily later on). Disulfiram leads to the accumulation of acetaldehyde (the product of the first step of oxidation of alcohol), as it blocks the activity of the enzyme, acetaldehyde dehydrogenase. Reactions, include flushing of the face, headache, palpitation, nausea and vomiting. With larger amounts of alcohol, cardiac arrhythmias, hypotension and collapse may occur.

Follow-up: Frequent and sustained follow-up is necessary for the success of abstinence. Those who drop out must be identified without loss of time and the families contacted. Relapses are common within the first year and so the follow-up must be carried out vigorously at least during this period. The social worker should liaise constantly with the person, the family, the management and personnel department to detect dropouts and bring them for consultation, counselling and other therapy as necessary.

Cost-effectiveness:

The programmes of preventing problem drinking, detection of alcoholism and treatment are highly cost-effective in reducing loss of productivity, boosting morale and preventing or decreasing expenditure on medical treatment of conditions arising out of excessive consumption of alcohol. Treatment of the problem drinkers can have beneficial effect on other staff in the industry.

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Experimenting with Family Treatment Approaches to Alcoholism, 1950-1975: A Review *

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The attention given family therapy approaches to alcoholism has been disproportionately low in relation to the magnitude of alcohol abuse as a clinical problem and its acknowledged impact on family life. Although the literature to date is limited and most studies should be characterized as pilot in nature, preliminary results have enthusiastically endorsed family therapy approaches to alcoholism.

This critical review assesses the existing experimental and clinical literature of the past 25 years. It also offers potential explanations for the reluctance of family therapists to engage this problem more actively.

CONSERVATIVE ESTIMATES indicate that at least 9 million adults in the United States abuse or are addicted to alcohol. Less conservative estimates range up to 15 million people. A stream of reports from both scientific and government sources have called attention to the fact that alcohol is once again the drug of choice of the American teenager. Financial estimates indicate that alcohol

* The preparation of this paper was supported by Grant No. R01 AA 01441 from the National Institute on Alcohol Abuse and Alcoholism. The author wishes to thank Ms. Lydia Tislenko for her assistance in the preparation of this paper.

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abuse exacts a staggering toll on American industry via absenteeism, interference with performance, and interference with sound judgment. These dramatic "tips of the iceberg" indicate that alcohol abuse in our culture carries with it staggering social and psychological consequences.

The pervasiveness of alcohol use and abuse in the United States is of such proportions as to guarantee that any mental health professional practicing in this country will be working with a significant number of patients whose use of alcohol has reached abusive proportions. For family therapists, who traditionally work with groups of two or more adults in conflict either with each other or with their adolescent children, the likelihood that one member of this group abuses alcohol becomes even greater. It seems clear, therefore, that treatment techniques for alcoholism should be of primary concern to the family therapist.

Whereas previously alcoholics were conceptualized as homeless, jobless, physically ravaged individuals with meager psychological resources, it is now clear that this "end stage" alcoholic is most unrepresentative of the patient population that abuses alcohol. A significant, if not major, proportion of the alcoholic population continues to function within nominally intact and stable family systems, a natural clientele for the family therapist. Therefore, whether or not the family therapist feels alcoholism per se is a condition appropriately treated by family therapy techniques, the symptom itself is so pervasive as to be virtually unavoidable. As we shall see in our review of the literature, however, alcoholism therapists have come relatively late to the family field, and family therapists have only recently begun to view alcoholism as an area of interest. This mutual disregard is frankly not at all surprising.

From the perspective of the traditional establishment in the alcoholism field, the priority issue has been the transformation of alcoholism from a moral problem into a medical problem. This conversion has been seen as a necessary prerequisite for the transfer of responsibility for alcoholism treatment from the judicial system into the medical establishment. With this goal in mind, the emphasis has been on the medical model. Alcoholism is viewed as a disease process with an etiology, a set of symptoms, a typical course, and a predictable prognosis. However, the medical model is designed primarily to describe disease processes as they affect an individual. Hence family therapy feels strange and foreign.

From the perspective of the family therapist, on the other hand, clinical interest focused on disturbed communicational patterns and structural dissonance within the family. Although these phenomena are hardly absent in the family with an alcoholic member, the abusive consumption of alcohol and its attendent behavioral and physical consequences appeared at first glance to be so overwhelming that it was hard to imagine successful treatment being achieved in any way other than intensive work with the individual who was doing the drinking.

Despite these obstacles, family therapy techniques have been used with increasing enthusiasm in alcoholism treatment. In recognition of this trend, the *Second Special Report to the U.S. Congress on Alcohol and Health* (27) called family therapy "the most notable current advance in the area of psychotherapy (of alcoholism)." We will review this development historically, paying attention both to theoretical trends and to those innovative experimental studies that have advanced the field. Although somewhat artificial, we can divide the existing literature into a four-step sequence that roughly builds one on another. Initial interest (I) in the "alcholic marriage," led to (II) experimentation with concurrent group therapy techniques. Later interest (III) in application of new family theory concepts to alcoholism, led to tentative use (IV) of more traditional family therapy techniques for alcoholism.

I. THE ALCOHOLIC MARRIAGE

Scattered reports concerning family factors in alcoholism had appeared in the literature prior to 1950, but the first concerted effort in this direction was a series of clinical reports about marriages between male alcoholics and their wives (2). The primary concern centered on the role of the wife in initiating and perpetuating her husband's drinking. A debate arose between a faction represented primarily by psychiatrists and psychiatric social workers who viewed the wife of the alcoholic as a person with severe, longstanding psychopathology antedating marriage, which led her to choose an alcoholic husband as a way of satisfying and stabilizing intrapsychic needs (4, 19), and a faction represented primarily by sociologists who explained the behavior of these wives as directly resulting from having to deal with the repetitive pressures and stresses placed upon the marriage by the husband's drinking (24, 25, 28, 29). In retrospect, this debate was probably artificial. The most recent review of this literature (13) concludes that no convincing evidence has emerged

suggesting a single personality "type" characteristic to wives of alcoholics, or a theoretical explanation of their behavior.

In any event, although interactional models were being proposed to explain behavior in an alcoholic marriage, most of the clinical data stimulating these ideas came from individually oriented therapy or research. For example, a clinician would be impressed with repetitive stories of inconsistent behavior on the part of his alcoholic patient's wife in which the wife is described as keeping the liquor cabinet well-stocked, pouring drinks for her husband, and making excuses for him in his work situation, at the same time that she is complaining bitterly about his excessive drinking and threatening to leave if he doesn't stop. Only rarely was this clinical data verified via a clinical interview with the wife as well (30, 45). Sociologists, on the other hand, obtained much of their data directly from wives, and had little opportunity to substantiate these reports via direct observation or collateral interviewing (24, 29).

However, these studies were important in providing a changing focus for therapy. Whereas earlier studies focused on family issues only from an historical perspective, the focus on the alcoholic marriage was a focus on the here-and-now. As long as the alcoholic individual was viewed in isolation and explanations for his or her abusive drinking were related only to individual psychodynamics or pathophysiology, the only logical treatment approaches would be individually oriented. If, however, questions were raised about the extent to which an interactional relationship between a husband and wife might either cause or perpetuate abusive drinking, then logic would dictate that a place had to be found for the spouse in the treatment plan. As Joan Jackson (25) has noted: "Once attention had been focused on the families of alcoholics, it became obvious that the relationship between the alcoholic and his family is not a one-way relationship. The family also affects the alcoholic and his illness. The family can either help or interfere with the treatment process" (p. 91). Jackson therefore concludes that significant family members must be taken account of, if not actively involved, in treatment in order to achieve success.

II. CONCURRENT GROUP THERAPY FOR ALCOHOLICS AND SPOUSES

In 1954, a project was instituted in the outpatient department of the Henry Phipps Psychiatric Clinic at the Johns Hopkins Hospital involving concurrent group meetings of male alcoholics and their wives (21, 22, 23). This project represented the first attempt to adapt the most successful psychological therapy approach to alcoholism, group therapy, to a family orientation. Nine male alcoholics and their wives were recruited and placed in two separate groups, one for the alcoholics, one for the wives. Thus, once they had volunteered for the study, husband and wife went their separate ways and entered into a group that developed its own schedule, therapy format, rules, and group process issues.

Despite the very small patient sample, this study was a pivotal one in the development of family techniques for the treatment of alcoholism. Although the specific results of the study were equivocal (marginal, but not convincing, improvement in most of the patients treated), ground was broken in a number of important areas that have subsequently become characteristic of family approaches. Perhaps the most important of these areas is the issue of outcome variables.

Most alcoholism treatment programs have focused almost entirely on a diminution of drinking as the sole outcome variable of merit. Although the wisdom of this approach has been questioned on occasion (34), the majority of treatment programs continue to be judged against a standard of percentage of patient population abstinent within a specified time frame. Gliedman, by including wives as potential clientele for the treatment program, significantly expanded the scope of appropriate outcome variables against which successful treatment was to be judged. Symptom reduction, for example, applied to the wife equally as well as it applied to her alcoholic husband. If symptoms such as depression are applicable for the spouse, then they must also be applicable for the identified alcoholic. Thus reduction in depression is added to reduction in drinking as an acceptable criterion for successful treatment. Secondly, the concurrent treatment of both members of a marriage naturally leads to an examination of marital satisfaction and marital interactional behavior as target criteria for therapeutic change.

Patients were therefore evaluated before and after treatement by means of four measures: (a) a drinking checklist to measure the severity of drinking; (b) a symptom checklist to indicate the amount of distress from psychological symptoms; (c) the mutual satisfaction or dissatisfaction experienced by the alcoholic husbands and their wives with each *other* during sobriety as contrasted with intoxication; and (d) a social ineffectiveness scale.

Within this widened perspective, Gliedman and his associates found that although there was some reduction in drinking behavior, the greatest changes in behavior resulting from the concurrent group therapy technique were in the areas of "marital milieu" (defined as satisfaction of alcoholic husband and his wife with each other) and "personal morale" (the alcoholic individual's satisfaction with himself). A significant change also was felt to occur in reduction in psychological symptomatology, especially irritability and depression, on the part of both alcoholic husband and non-alcoholic wife. The least change seemed to occur in the area of social effectiveness, which was judged to be poor at the start of therapy and showed little improvement as a result of the group experience. Gliedman's conclusion was that his concurrent group therapy technique was most effective in its ability to improve or elevate self-esteem in a patient group that tended to be demoralized prior to therapy.

Following the Johns Hopkins study, several clinical papers appeared in the literature describing group techniques for working with spouses of alcoholics (7, 30, 35, 37, 44). These papers indicated a growing interest in the development of techniques for changing the treatment focus from the alcoholic individual alone to the alcoholic individual in a marital context. It also reflected the conviction that the inclusion of the non-alcoholic spouse in the treatment of those alcoholic individuals who retained a stable marriage was a necessary prerequisite for successful therapy. Pixley and Stiefel (37), for example, state: "There is no question at this point that if psychotherapy is to be effective for a larger proportion of the alcoholic population the wife must also be treated" (p. 312).

The most ambitious study of concurrent group psychotherapy was carried out by Ewing and his colleagues (18) at the University of North Carolina School of Medicine. For a period of four years, starting in 1955, a program was established offering an optional concurrent group therapy program for spouses of alcoholic individuals already in treatment. Although the program was offered to both male and female alcoholics alike, only wives of male alcoholics volunteered for the program.

During the first 18 months of the program's inception, 32 still-married alcoholic men were accepted into the group therapy program offered by the Department of Psychiatry for alcoholism treatment. Of these 32 men, 16 wives volunteered to participate in concurrent group psychotherapy sessions. In contrast to the Johns Hopkins group, similar schedules were adopted for the husbands' group and wives' group. Both groups met weekly in different rooms in the same building; the basic therapy technique was described as "dynamically oriented group psychotherapy."

Long-term, follow-up data provided by Ewing's group is impressive on two scores. First is the finding of a significantly greater persistence in therapy for those male alcoholics whose wives were attending a concurrent group psychotherapy session. Second, long-term follow-up (a minimum of three years after the inception of group therapy) indicated significantly improved control of drinking and considerable improvement in marital harmony for those men engaged in concurrent group therapy with their wives, as opposed to men coming alone to the therapy program. The question of whether this improvement was due to the specific working through of marital issues in the therapy sessions or merely due to the increased longevity of treatment (because the engagment of the wives assisted in keeping their alcoholic husbands in treatment for a longer period of time) is raised but left unanswered by this study. However, since engagement of alcoholics in long-term therapy has in and of itself been a major obstacle to successful treatment, the results of the Ewing study have to be viewed as impressive.

Ewing's findings were strongly supported in a study carried out by Smith (39) at the University of Edinburgh. Despite the fact that the treatment program was radically different, (alcoholics were hospitalized for up to a six-week stay as opposed to being treated on an outpatient basis in the Ewing study), the institution of a separate therapeutic group for wives of alcoholic men led to a significantly greater rate of improvement as contrasted with men whose wives did not attend.

These clinical papers have therefore been by and large enthusiastic about the concurrent group psychotherapy technique. Although the emphasis remains on the effectiveness of the technique as an adjunct to the treatment of the alcoholic husband, wives are reported to be engaged in treatment for their own needs, having demonstrated independent issues of concern that could benefit from therapeutic examination.

III. THE ADAPTATION OF FAMILY THEORY TO ALCOHOLISM THERAPY

The studies discussed up to this point, although taking cognizance of family factors in alcoholism, were by and large adaptations of

existing individual and group therapy techniques. During this same time frame, however, a body of clinical theory dealing with family pathology, family concepts of symptoms formation, and family-oriented therapeutic interventions was being developed. Since most of these clinical thinkers were psychiatrists (Ackerman, Bowen, Jackson, Minuchin), their attention was naturally drawn toward new explanations for traditional psychiatric conditions such as schizophrenia, psychosomatics, and adolescent dysfunction. Although somewhat puzzling in retrospect, alcoholism and drug abuse were almost totally ignored both theoretically and clinically.

In the late 60s and early 70s, some cracks began to appear in this wall of indifference. The first marriage of family theory and alcoholism therapy appeared in Ewing and Fox's article "Family Therapy of Alcoholism" (17). Ewing and Fox adapted theoretical concepts associated with Bateson and Jackson's work with families, especially Jackson's notion of homeostasis in family systems. The alcoholic marriage is viewed as a "homeostatic mechanism" that is "established...to resist change over long periods of time. The behavior of each spouse is rigidly controlled by the other. As a result, an effort by one person to alter his typical role behavior threatens the family equilibrium and provokes renewed efforts by the spouse to maintain the status quo" (p. 87).

Specifically addressing marriages between male alcoholics and their wives, they suggest a process in which these two people strike an elicit and "implicit...interpersonal bargain," a marital "quid pro quo," to use Jackson's terminology, in which the male alcoholic's passive dependency needs implicitly encourage his wife's protective nurturing needs. A sexual bargain is also struck engaging an undemanding alcoholic husband in a behavioral pattern that complements the behavior of his sexually unresponsive wife. Both of these interactional pacts are played out within the context of a cyclical system in which the alcoholic marriage alternates between periods of sobriety and periods of intoxication. "By alternating between suppression of impulses and direct expression of them, he can maintain the conflict surrounding impulse gratification for a lifetime" (17, p. 91).

Ewing and Fox recommended family therapy for such families for two reasons: it increases the likelihood that a drinking problem will be acknowledged by a patient population (middle-class, gamma-type, male alcoholics) who are usually resistive to such self-labeling

procedures, and it stimulates motivation toward change within the alcoholic himself.

Based on their extensive clinical experience, Ewing and Fox conclude that "alcoholism can no longer be seen purely in terms of intrapsychic dynamics...It is the family emotional homeostasis which seems to perpetuate the drinking, and it is this behavior which must be changed if the drinking is to be controlled" (17, p. 91). Their therapeutic approach, concurrent group therapy, emphasized the need for reciprocal work with husband and wife in order to coordinate change in both halves of the homeostatic dyad. The corollary prediction was that working within an individual framework might increase the drive to change in the individual but would also increase the pressure toward resistance on the part of the spouse. Therapeutic efforts in one direction would therefore be countermanded by resistances in the other direction, minimizing the opportunity for a positive therapeutic outcome.

Steinglass and his co-workers (12, 40, 41, 42, 46) have incorporated many of the same concepts (homeostasis, marital bargain, complementary role functioning) in a more comprehensive interactional model of alcoholism developed in response to clinical observations of family interaction made during states of experimentally induced intoxication. These observations suggested that interactional behavior during intoxication is highly patterned and often dramatically different from the behavior predicted by the family during sobriety. As one example, a family that claimed drinking by their "identified alcoholic" caused depression, fighting and estrangement was observed to show increased warmth toward each other, increased caretaking, and greater animation when the "alcoholic" was permitted to drink.

The interactional model proposed by Steinglass is based on general systems concepts of family functioning. These concepts posit that families are operational systems obeying laws general to all systems, including the importance of organization, drive toward homeostasis, circularity of causal events, and feedback mechanisms as factors determining the quality of interaction between the component parts of the systems (in this case, members of the family plus alcohol).

Alcohol ingestion and intoxicated behavior is then viewed from the perspective of the extent to which, and manner in which, it affects the interactional life of the members of the family. Steinglass also

suggested that alcohol, by dint of its profound behavioral, cultural, societal, and physical consequences, might assume such a central position in the life of some families as to become an organizing principle for interactional life within these families. He labeled such a family an "alcoholic system." In such a system the presence or absence of alcohol becomes the single most important variable determining the interactional behavior not only between the identified drinker and other members of the family but among non-drinking members of the family as well.

This model implied that an intricate and delicate balance exists between drinking and the day-to-day functioning of the family. In fact, it was suggested that in certain instances alcohol might be unconsciously viewed by the family as a *stabilizing* rather than a disruptive influence on their interactional life. Although superficially disruptive, from a different vantage point, the abusive use of alcohol seemed to produce extremely patterned, predictable, and rigid sets of interactions that dramatically reduced uncertainties about the family's internal life and its relationship to the external society.

The opportunity to directly observe intoxicated interactional behavior led not only to unique theoretical proposals but also to quite different conclusions about therapeutic intervention. If, in fact, alcohol might be aiding "system maintenance," which in clinical terms means serving some important dynamic function in the interactional life of the family, then the first role of the therapist dealing with the drinking symptom in a family context is an appreciation of the relationship between alcohol and family life. In certain situations it seems clear that the identified patient's drinking behavior emerges *de novo* in a family situation at a time of stress or strain. In these situations the drinking behavior might well be viewed as a signal or symptom reflecting this stress or strain, and crisis intervention is called for. On the other hand, if alcohol consumption is part of an ongoing interactional pattern within the family system, then the traditional therapeutic intervention aimed at abstinence is totally inadequate to the task.

A logical extension of this theoretical model is to view family therapy not so much from the point of view of involving family members as a mechanism for improving treatment with the identified alcoholic but rather to view the entire family or the marriage itself as the patient. Therapeutic intervention becomes interactionally oriented rather than intrapsychically oriented, and goals for treatment center around an improvement in the functioning, flexibility, and growth potential of the family system as a whole rather than the more limited focus on reduction in drinking on the part of the identified alcoholic.

A paper by Davis, *et al.*, (12) expands on this theoretical model in two significant directions. First, it incorporates behavior theory, and second, it underscores the importance of focusing on maintenance factors rather than etiological factors at this very primitive stage of our understanding of chronic alcoholism. Pointing out that historically there have been two major premises underlying therapeutic approaches to alcoholism—the notion that excessive drinking is maladaptive and the belief in the existence of ultimate causes as explanations of why alcoholism develops—Davis notes that these premises have given rise to a wide variety of therapeutic approaches. These range from moralistic exhortations and aversive behavioristic approaches deriving from the maladaptive premise to the uniform psychodynamic or psychobiological approaches based on ultimate cause theories. Clinical experience, however, suggests that alcoholic behavior is more profitably thought of as a final common pathway. Incorporating behavioral concepts into the systems model allows for clinical diversity while at the same time suggesting new therapeutic strategies.

Davis *et al.*, postulate the following: that the abuse of alcohol has certain *adaptive consequences;* that these adaptive consequences are sufficiently reinforcing to serve as the primary factors maintaining the habit of drinking, regardless of what underlying causation there may be; and that the particular adaptive consequences or "primary factors" for each individual may differ and might be operating at a number of different levels including intrapsychic, intracouple, or the level of maintenance of homeostasis in a family or wider social system but that the final common pathway is the reinforced, chronic abuse of alcohol.

Two major implications for therapy are suggested. First, it is necessary for the therapist to determine the specific manner in which drinking behavior is serving an adaptive function for an individual or family. The maladaptive consequences are obviously readily apparent. Search for the adaptive consequences requires more clinical skill. Second, it is suggested that once the adaptive consequences of drinking have been ascertained, therapy may be structured around helping a patient to manifest the adaptive behavior while sober

instead of only during drinking and to learn effective, alternate behaviors.

Bowen (6), using similar concepts, also views alcoholism as potentially explainable in the language of family systems theory. Pointing out that alcoholism is one of the common human dysfunctions, Bowen contends that, as a dysfunction, alcoholism must "exist in the context of an imbalance in functioning in the total family system" (p. 115). In this context, every family member is seen as contributing to the dysfunctional behavior of the alcoholic member. In fact, Bowen would contend that the dysfunction of the alcoholic can only continue with the support of his or her family. Treatment that alters the behavior patterns of these other family members will, therefore, by definition, eliminate the necessary substratum for the existence of alcoholism. Bowen therefore states that "when it is possible to modify the family relationship system, the alcoholic dysfunction is alleviated, even though the dysfunctional one may not have been part of the therapy" (p. 117).

IV. FAMILY THERAPY TECHNIQUES

A. Conjoint Family Therapy

The previous section has reviewed the growing *theoretical* literature on family therapy for alcoholism. The literature reporting results of the use of conjoint family therapy for alcoholism has to date been limited to infrequent clinical papers describing case histories offered in support of the use of family therapy techniques. By conjoint family therapy, we are now talking about techniques involving conjoint interviewing of both members of a marital pair, or conjoint interviewing of two or more members of a nuclear or extended family. Of the limited number of reports on family therapy with alcoholics currently available, none has appeared in a journal of publication primarily addressed to family issues (e.g., *Family Process* or *Journal of Marriage and the Family*).

The state of the literature, however, is in all likelihood unrepresentative of the extent to which family therapy techniques are actually being utilized for the treatment of alcoholism. In many alcoholism treatment centers it is routine for therapists to insist on the inclusion of other family members in the initial evaluation, and conjoint interviewing techniques are often included as one option available to

the treatment team. However, on the other side of the fence, it is not yet the case that family treatment centers routinely view the treatment of alcoholism as within the scope of their expertise. Traditional family agencies will often refuse to work with families containing an identified alcoholic member, even when the families present themselves because of problems other than alcoholism. This is particularly true with agencies working with lower middle-class and lower-class families where a rapid referral to the "alcohol" center is the preferred disposition regardless of the nature of the presenting complaint.

An interesting study by Meeks and Kelly (31) evaluating the efficacy of family therapy techniques introduced during the recovery phase of the treatment of the alcoholic member of the family is the most representative and influential of the clinical studies of conjoint family therapy. Although only five families were treated and studied, as a pilot study this report is of considerable interest to us.

Meeks and Kelly adhere firmly to the theoretical orientation of the family therapist (in this case, Virginia Satir). Conjoint family therapy was begun following an intensive 7-week program of individual and group psychotherapy in a day treatment program. During this 7-week program, family members were seen separately from the "alcoholic patient." At the beginning of the aftercare phase, however, family members were seen in conjoint interviews only; the alcoholic member was never seen separately from his or her family during the aftercare phase. Families were seen for periods ranging from 10 to 12 months.

Therapy focused on interaction, communication, role performance, and redefinition of problems in family rather than in individual terms. Therapeutic goals were derived from Ackerman, and included achievement of a clearer definition of interactional conflicts, improved and more open communication about these conflicts, a greater understanding of intrapsychic determinants of interpersonal conflicts, and an improved level of complementarity in family role relations. Treatment evaluations included an interest in the drinking behavior of the identified alcoholic but focused more intensely on issues of improved family interaction and family equilibrium. Such issues as problem definition, communication, patterns of relating, and methods of problem-solving were included as possible variables indicating improved family functioning. The study also attempted to assess the extent of the family involvement

in the treatment process, the extent to which the therapists were able to remove the alcoholic member from the "identified patient" status, and issues of therapist involvement.

Although Meeks and Kelly underscored the exploratory nature of their report, they were enthusiastic about their experience. They concluded that techniques geared toward redefining alcoholism issues in family terms are quite profitable. The more drinking behavior can be seen as merely one aspect of family interaction, the greater the likelihood that the "alcoholic" member of the family will be able to shed his or her label and establish new patterns of interaction within the family.

Esser (14, 15, 16) reached similar conclusions in reports stemming from his experience with conjoint family therapy in the Dutch city of Haarlem. Once again the emphasis is on the recovery or aftercare phase of treatment. Family therapy is seen as potentially expanding the scope of treatment from hospitalization and clinical care for the identified alcoholic to a more sociotherapeutically oriented approach to the entire family. The family of an alcoholic is viewed as a "group under stress," but this stress is related as much to disturbed interactions as it is to the behavioral effects of alcohol. Restoration of communication, concentration on role conflicts, and the removal of the alcoholic from the role of the "identified patient" are again seen as the central issues that the therapist must approach.

These clinical reports can best be characterized as promising but unsubstantiated, enthusiastic but primarily impressionistic. They seem to reflect the level of optimism attached to family therapy for alcoholism—e.g., the *Alcohol and Health* (27) statement and Chafetz *et al.* (10)—but leave unanswered questions about the verifiable efficacy of these techniques.

B. Multiple-Couples and Multiple-Family Group Therapy Approaches

Multiple-couples group therapy is a particularly popular form of family therapy currently being utilized in alcoholism treatment. Its increasing popularity as a treatment modality in alcoholism treatment programs is perhaps related to the assumption that it represents the "best of all possible worlds." It retains, in format at least, a group therapy structure and is therefore attractive to many alcoholism therapists who have viewed group therapy as the treatment of choice. However, it also acknowledges and takes account of the importance of family factors in the exacerbation of alcoholism and is responsive to the growing feeling that alcoholism treatment is less effective if significant family members are not involved in therapy.

A growing body of experimental and clinical literature now exists concerning multiple-couples group therapy approaches to alcoholism. This literature includes traditional treatment outcome studies (9, 11), reports of experimental treatment techniques (42), and summaries of clinical experiences (5, 20, 38). We will examine three reports more extensively: an outcome study of multiple-couples therapy based on group techniques (9); an experimental study based on family therapy principles (42); and a clinical report of the extensive use of mulitple-couples groups in an operational alcoholism treatment program (20).

Cadogan (9) presented the first controlled study in the literature of multiple-couples group therapy in alcoholism treatment. Forty alcoholics (both men and women) and their spouses were recruited while the alcoholics were still inpatients at a traditional alcoholism unit and asked to volunteer for a "new and effective method of treatment" in which "an attempt would be made to improve family problem-solving patterns, to encourage the expression of feeling in marital communications and to develop a new awareness of the effect of their behavior on others" (p. 1188). The study group represented the first 40 couples volunteering for this new outpatient multiple-couples group therapy program. Subjects were then randomly assigned to one of two groups: an immediate treatment group or a waiting list in which they continued with the traditional treatment program but did not engage in the outpatient, multiple-couples group. Ultimately 20 couples were assigned to each group. Groups proved to be comparable in age, socioeconomic status, severity of alcoholism, and involvement with other treatment programs (especially AA).

The treatment group engaged in open-ended, multiple-couples group therapy sessions (90-minute sessions on a once-weekly schedule). The average group was composed of five couples, and membership was fluid with dropouts being replaced by newly interviewed recruits. Follow-up evaluation occurred six months after the couple was recruited for the study. Follow-up results were striking. At six months, nine alcoholic members in the therapy group remained abstinent, four were doing some drinking, and seven had relapsed

completely. Among the control group, however, only two were abstinent, five were drinking moderately, and 13 had demonstrated complete relapse.

Gallant, *et al.*, (20) have provided a report of the most extensive application to date of multiple-couples group therapy as an integral phase or component of an ongoing alcoholism treatment program. Their program, the New Orleans Alcoholism Clinic, comprises two integrated units, a 36-bed inpatient unit, and an outpatient alcoholism clinic. Gallant has been routinely assigning every discharged married patient who is returning home to live with his or her spouse to a multiple-couples therapy group in the outpatient clinic as the major form of ongoing treatment. These therapy groups, comprising four to seven couples meeting every two weeks for a two-hour session have a traditional alcoholism treatment goal of total abstinence for the alcoholic combined with an interpersonal goal of improvement in marital interaction. Treatment techniques combine both family therapy orientations toward analysis of interactional behavior and group therapy techniques of encouraging direct exchange and feedback between all members of the group.

Gallant has reported the results of 118 couples assigned to the Clinic's multiple-couples groups. Follow-up data were not systematically gathered, but most couples were contacted following treatment and drinking history and quality of family life were explored. (The follow-up period varied from two months to 10 months.) Fifty-three of the 118 couples were considered to be definite successes at the time of follow-up (either complete abstinence or no more than two brief drinking episodes and "reasonable" marital relationship), and 41 were considered definite failures (unhappy family life, frequent drinking episodes, or sobriety felt by the treatment team to be temporary and without satisfaction or contentment). Twenty-four couples were lost to follow-up. Based on these findings, Gallant, *et al.*, "conclude that marital-couples group therapy is the treatment of choice at this time for married alcoholic patients. The denial and projection mechanisms, exaggerated in the alcohol-marital problem, are more easily approached and treated in group" (20, pp. 43-44).

Steinglass and his colleagues have carried out work with multiple-couples therapy groups as part of their research studies examining interactional behavior in alcoholic families. An experimental treatment program was established at NIAAA's Laboratory for Alcohol Research in which couples with one or two alcoholic members were placed in an intensive, six-week, multiple-couples group therapy program. Although the treatment program was conceptualized primarily as an experimental model that permitted the establishment of a rich clinical field allowing for the examination of interactional behavior, the treatment process itself was highly unusual and proved to be quite fascinating in its own right. In contrast to Cadogan's work, where the emphasis was on the desirability of involving the spouse in a group process, the NIAAA group was firmly based in family therapy.

The experimental treatment program was divided into three phases: an initial two-week outpatient phase in which groups met for three sessions per week; a ten-day inpatient phase during which time three couples were simultaneously admitted to an inpatient facility; and finally, a post-hospitalization, three-week outpatient phase of two group meetings per week. Following the six-week intensive treatment program, groups reconvened at six-week intervals for follow-up sessions over a six-month, follow-up period.

The core of the program, and clearly its most innovative feature, was the hospitalization period. The hospital setting itself was a redesigned inpatient unit in a traditional state hosptial. This unit was described by the research team as a "simulated apartment setting" that was supposed to provide a homelike atmosphere for the couples, allowing them to reproduce as accurately as possible their usual interactional behavior. Of greatest importance, however, was the fact that alcohol was made freely available during the first seven days of the hospitalization period, and couples were asked to engage in their usual drinking patterns while they were on the ward. This last feature of the treatment program was an extension of the use of experimentally induced intoxication as a potential adjunct to therapy and was based on theoretical notions about the role alcohol can play in maintaining fixed interactional patterns within familes (these notions are discussed in Part III). The specific rational provided to the couples for this free availability of alcohol was that the therapist, by being able to directly observe intoxicated behavior, could gain a better understanding of the role that alcohol consumption was playing in the couples' lives.

The treatment program utilized a variety of techniques to examine patterns of interaction exhibited by each couple and to contrast the difference between interaction during sobriety and interaction during intoxication. These techniques included videotape recording and

feedback, role-playing techniques, use of one-way mirror observation, and feedback from observers, analysis of speech and communication patterns, emphasis on non-verbal behavior and postural analysis, and use of three-generational family genograms. All of these techniques have been used extensively by family therapists in more traditional settings.

The multiple-couples groups were conceptualized by the researchers as a societal system composed of three distinct elements or levels: individuals, couples, and whole group. This type of group was therefore viewed as an excellent vehicle for observing the relation between individual dynamics and intra-couple dynamics while at the same time having an opportunity to observe the couple's behavior in negotiating its position in a group of strangers (perhaps analagous to the relation between family and the outside society). However, the therapeutic target was always the couple, and individual dynamics or whole group behavior was viewed from the vantage point of its relation to each of the three couples.

Although this NIAAA program obviously represented a radical departure from traditional alcoholism treatment, it also is the purest example in the literature of an approach to alcoholism treatment based on family principles. Let us therefore summarize the main features that made this program unique: First, the program recruited middle-class, intact couples who displayed a substantial degree of economic and interactional stability despite the chronic abuse of alcohol by one of its members. Second, the program not only did not insist on the usual abstinence model of treatment, it actually suggested that intoxicated behavior can be utilized by the therapist as an adjunct to treatment. Third, instead of viewing the individual alcohol abuser as the "problem," therapy was directed at the couple. Fourth, both drinker and spouse enjoyed similar status as inpatients in a psychiatric hospital, and treatment goals and techniques were based on examining the relation between alcohol use, intoxicated behavior, and interaction. And last, the therapists insisted on improved family functioning rather than a reduction of drinking behavior as the primary target.

Although Steinglass and his colleagues have advised caution in recording outcome results from this experimental study, emphasizing the highly experimental, pilot nature of the program, it was found that couples responded quite positively to the treatment approach. Although only ten couples were treated, they all completed the study

despite its strenuous demands and reported a profound emotional impact deriving from the in-hospital experience particularly. The enthusiasm of patients for the therapeutic work was particularly impressive to the therapists in light of the fact that all couples had failed repeatedly in previous therapeutic efforts. The therapists were also most enthusiastic about the in-hospital experience as a mechanism facilitating the rapid clinical understanding of the relation between drinking behavior and interactional life for each of the couples involved.

Reports of six-month, follow-up data have not yet been completed, and it is therefore unclear at this time to what extent the intensity of this therapeutic experience was meaningfully integrated and had a lasting effect in changing the interactional patterns of the couples involved. In all likelihood this experimental study will be more valuable in suggesting directions for therapy rather than establishing a definitive therapeutive approach.

Two additional studies, although not focusing specifically on multiple-couples therapy, will be mentioned here because they also involved simultaneous work with spouses in a traditional hospital setting. Corder, *et al.*, (11) carried out a pilot project at a residential alcoholism treatment center. Wives of male alcoholics were included in a four-day, intensive workshop that followed a traditional, three-week inpatient program. The workshop program included group therapy and videotape and analysis of the sessions, didactic lectures, group discussions of "game-playing" and role-playing, recreational activities, and AA and Al-Anon meetings. A six-month follow-up performed on the pilot group of 20 alcoholics indicated a significant reduction in drinking for the experimental group compared with a control group that had gone through the traditional treatment program alone.

Paolino and McCrady (32) have been experimenting with the "joint admission" of a non-alcoholic spouse as a "guest" of the hospital. The couple lives on a psychiatric ward that includes patients of all diagnoses and ages over 12. The non-alcoholic spouse participated in ward activities as much as possible while retaining his or her job. The patient and spouse also participated in three types of weekly therapy groups: a group for *problem drinkers only*, a group for *spouses only* of problem drinkers, a group for *couples* in which one member is a problem drinker and the other member does not have a problem with alcohol. These groups all continued after

the couples left the hospital and were considered an essential pa
of the treatment program.

Paolino and McCrady conceptualized the goals of such a joi
admission as follows: (a) to give the staff the opportunity to obser
the couple's interactions; (b) to provide comprehensive feedback
the couple about their patterns of interacting; (c) to integrate th
spouse into the milieu so that the spouse has the same opportunity
the problem drinker to experience the closeness and caring of th
unit; (d) to integrate the spouse into the milieu so that the spou
may incorporate the approach to handling problems that is taught
the milieu. They plan extensive experimental studies based on th
treatment approach.

DISCUSSION

When compared to the magnitude of the problem, our review
revealed a remarkable paucity of studies dealing with family ther
for alcoholism. Although the literature is probably not representa
of actual therapeutic practices, it is nevertheless striking th
alcoholism, in comparison to such conditions as schizophrenia,
linquency, or psychosomatics, has engendered remarkably li
interest among family therapists. (Parenthetically, a similar aver
seems to exist in the area of drug abuse.) Let us therefore disc
two issues raised by this review: first, the demonstrated efficac
family therapy techniques for the treatment of alcoholism a
second, potential explanations for the disinterest of family therap
toward alcoholism.

Evaluation of existing research

The existing literature leaves us with a sense of guarded optim
about the application of family therapy techniques in the treatm
of alcoholism. Although every study we have mentioned conclud
with an enthusiastic statement encouraging greater use of fa
therapy, it is also apparent that very little hard evidence exist
this point demonstrating either the efficacy of family therapy
itself or the comparative value of family therapy versus
traditional forms of therapy in the treatment of alcoholism.

Even taking into account the inherent difficulties in psychot
apy research, most of the studies reported in this review shou

viewed as pilot or exploratory ventures rather than definitive attempts to validate a treatment method. The traditional difficulties: inadequate sample size, absence of an appropriate control group, lack of consistency in definition of subject population, variability in outcome measurements are all amply represented in these studies. However, it must also be recognized that these studies were often difficult to carry out, involving major manipulations of the environment, or the introduction of alien therapeutic techniques into a hostile treatment environment.

In addition to the traditional difficulties, however, there are at least two specific difficulties with the existing studies that deserve special mention. The first is the singular absence of trained family therapists carrying out these studies. The Steinglass study is the only report that specifically identifies its therapists as family rather than alcohol specialists. Meeks and Kelly followed family therapy principles outlined by Satir. All other studies were apparently carried out by alcohol specialists, firmly grounded in group therapy techniques, who were extending their approaches to include additional family members. As a consequence, very little technical information is available in the existing literature for the family therapist interested in specialized techniques applicable to alcoholic families.

The second aspect that deserves special mention is the issue of outcome measures. The criterion of abstinence has been the traditional outcome measure for alcoholism treatment programs (34). One of the contributions of the studies described in this review has been to expand notions of treatment outcome to include not only other aspects of individual functioning but also aspects of interactional and social functioning. On the one hand, we should view this as a positive contribution. On the other hand, it introduces a whole panoply of outcome measures that are highly subjective in nature, vary from study to study, and are not necessarily appropriate to the patient population being treated. The current trend in family therapy research has been toward more objective outcome measures, e.g., need for rehospitalization, number of flare-ups of a psychosomatic illness, adolescent arrest records in delinquent families, etc. Alcohol consumption would be such an objective measure but clearly a distasteful one. Until this dilemma in outcome measures is resolved, it will be impossible to ascertain the particular effectiveness of family therapy for alcoholic families.

Given these rather extensive reservations, what does the existi literature suggest about the efficacy of family therapy for alcoholis that gives us grounds for optimism. The most obvious finding is tha every study reported positive results. Although their reasons f doing so differed, all investigators wound up enthusiastically su porting the involvement of families in the treatment process. Thre studies in particular, Ewing's and Smith's studies of concurre group therapy and Cadogan's study of multiple-couples gro therapy, yielded impressive results when experimental groups we contrasted with controls.

Of particular importance is the apparent ability of family-orient techniques to increase longevity of involvement in treatment on t part of the identified alcoholic. Since attrition rates have characte istically been high in alcoholism programs, engagement of t patient becomes a critical feature separating success from failure. remains unclear whether these programs owed their success inherent technical superiority, therapeutic enthusiasm, or con quences of theoretical orientation (elimination of the alcoholic as identified patient, shift in emphasis from alcohol consumption marital interaction, etc). However, initial results are clearly pro ing and warrant considerably greater enthusiasm and interest th to date exists.

Disinterest in Alcoholism on the Part of Family Therapists

In exhibiting a singular lack of interest for the treatment alcoholism, family therapists have merely been following the pre lections of their colleagues in the mental health professions. Prof sional stereotypes toward alcoholism and the alcoholic have b well-documented. The alcoholic is viewed as a distasteful, self-ind gent, weak individual involved in a pernicious cycle of self-destr tive behavior. Motivation for change is thought to be extremely and therapeutic work therefore felt to be unrewarding. Althou originally viewed as scientifically objective, these stereotypes h more recently been characterized as culturally determined, and applicable at all, applying only to a very small percentage of alcoholic population. Therefore, in part the family therapis merely suffering from cultural prejudices.

However, family therapists have prided themselves on their ab to look afresh at traditional mental health problems. Are t

particular aspects to alcoholism that have produced relative blind spots for the family therapists? Apparently so. Alcoholism, in fact, is a chronic behavioral process that has been demonstrated to exist most typically in an extremely stable and often rigid interactional context. As exhibited by the typical alcohol addict living in an intact family, its behavioral characteristics seem paradigmatic of homeostatic behavior in a steady-state system. In this regard it seems to be typical of chronic disease processes as they are utilized by family systems to stabilize both internal interactions and relationships with the external environment.

Why then the difficulty in viewing alcoholism as a family systems problem and recommending family therapy as an appropriate course of treatment? Although the answer is hardly clear-cut, several possible suggesions can be offered. The first difficulty lies in the area of definition. Drinking behavior exists along a continuum starting with total abstinence, progressing through occasional drinking associated with social rituals, social drinking, heavy drinking, and ending with addictive drinking. Somewhere along this continuum one makes the judgment that drinking behavior has reached abusive levels. Where to draw the line separating pathological from customary behavior, however, has been a controversial and usually subjective issue. Since many therapists are unclear as to where their own drinking might fall, there is a natural reluctance to take a judgmental stance about a patient's drinking behavior.

A second issue is the existence of the symptom in the parental generation. Family therapy historically developed in response to clinical conditions manifesting symptomatology in the childhood generation (schizophrenia, delinquency, school phobia, psychosomatics). The concept of the identified patient, a key concept in the development of family therapy, has traditionally been applied to situations in which a child in a family becomes symptomatic in response to a dysfunctional family system. Alcoholism in a family context perhaps represents the reverse situation; a parent becoming periodically symptomatic as an *adaptive* or *stabilizing* mechanism for the family system. Family therapists, by dint of their traditional orientation, might wind up overlooking such a situation.

Another possible explanation lies in the nature of intoxicated behavior itself. Most therapists experience discomfort in the presence of intoxicated patients. The intoxicated person is viewed as excessive, sloppy, impulsive, and lacking in self-control. The natural

response is to assume that therapeutic work cannot proceed during the intoxicated state. Rules are therefore usually established prohibiting intoxicated behavior during therapy sessions. However, if some of the theoretical notions mentioned in this review are correct (3, 6, 12, 17, 40), then abusive drinking behavior in the family context can only be understood if one examines carefully the relationships between intoxicated and sober interactional behavior. Since family therapists prefer to work with directly observable behavior, retrospective reports of intoxicated behavior are often overlooked or not solicited, and abusive drinking rarely becomes a focus for therapeutic interest.

Although the above suggestions may help explain the lack of interest in alcoholic families expressed thus far by family therapists, it also seems clear that none of these explanations represents a valid reason for ignoring this condition. This lack of substantial contraindications, combined with the tentatively positive results reported in this review, would certainly support a growing interest on the part of family theorists and therapists in the area of alcoholism. In fact, if comprehensive studies are undertaken and substantiate the pilot results reported in this review, it is conceivable that future therapy programs for alcoholism might be split into two distinct approaches: a family-oriented, psychosocial approach applied primarily to middle- and upper-class alcoholics with intact families and a biomedically oriented approach combining pharmacotherapy, behaviorist techniques, and group therapy applied primarily to single alcoholics. The former approach would structure itself on a family systems theoretical framework, while the latter approach would remain within the more traditional framework of the medical model of alcoholism.

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To,

Dr. C.M. Francis,
Director,
St. Martha's Hospital,
Banglore 560 009

Dear Dr. C.M. Francis,

Greetings from Kerala.
I came back safe from Banglore on 3rd of this month. I had a plesant journey. It was fortunate that I could be in touch with you during my visit to Banglore. We could share a lot of things connected with the addiction field. May I thank you most sincerely for your great support and help while I was there.

As I had talked with different professionals like you in Banglore to start a treatment centre I could very well realise the need for it. For a follow up of further discussions and to make a blue print for a treatment programme I will be coming there on 27th of August. Hope to meet you in between 27th and 29th for further discussion and support.

Hope we can do something in this field together to help the addicts.

Thanking you once again for your warm welcome and the useful time you spent with me.

CM/S/7

Yours sincerely,

(Fr. George Kolath)

Director.

ALCOHOLISM: AN ILLNESS

FACTS TO HELP THE ALCOHOLIC HELP HIMSELF

TABLE OF CONTENTS

CHAPTER 1

Alcohol and Man

Alcohol has had its place in the lives of men from very early times. References to alcohol use have been made even in ancient Vedic literature.

As time passed, man began to understand and appreciate alcohol's effects such as intoxication, excitement, euphoria, and tranquilisation. Large industries producing alcohol grew up and consumption of alcohol increased and became widespread.

It has also been understood that long term use of alcohol proves harmful. It causes dependence, illnesses and destroys the very fabric of man's personality and character.

What is Alcohol ?

The alcohol present in all alcoholic beverages is ethyl alcohol. Ethyl alcohol or ethanol (C_2H_5OH) is a colourless, slightly volatile liquid with a harsh, burning taste. It is a product of fermentation.

When yeast acts on sugarcane, honey, fruits, berries, cereals or potatoes it releases an enzyme which converts the sugar in these materials into carbon-di-oxide and alcohol. This process is known as fermentation.

Alcohol is the only drug which can also be classified as a food because of its high calorie yield. It contains negligible amounts of vitamins and minerals and contributes nothing to the cells' nutritional requirments.

Alcohol is a mood changing drug like opium, cocaine, barbiturates and amphetamines.

The alcohol content and source of some alcoholic beverages is given below.

Name of the beverage	Source	Percentage of alcohol
Brandy	Distilled wine	40—55%
Whisky	Cereals	40—55%
Rum	Sugar Cane (molasses)	40—55%
Wines (Port, Sherry, Champagne etc)	Grapes	10—22%
Beer	Cereals (barley)	6—8%
Toddy	Palm juice	5—10%
Arrack	Molasses	50—60%

What happens when you drink Alcohol ?

Alcohol differs from other foods in that it requires no digestion and by the process of simple diffusion, enters the circulatory system from the stomach and intestines. About 20% of the alcohol consumed is absorbed into the blood stream from the stomach walls and 80% from the intestines. Its small and simple molecular structure allows it to pass right through the cell membranes of the stomach and intestines. Once alcohol enters the blood stream, it circulates throughout the body reaching every organ, including the brain, heart and liver. Small amounts of it are eliminated from the body through urine, sweat and the respiratory tract. Once it enters the body, most alcohol remains there, until it is broken down by the liver.

The liver plays a major role in the break-down or oxidation of alcohol. Alcohol is oxidised by the liver at a rate of 8-15 ml per hour. The oxidation process is brought about by enzymes produced by the liver. Alcohol is first oxidised to acetaldehyde by the enzyme alcohol dehydrogenase. The acetaldehyde in turn is converted to acetate by the enzyme aldehyde dehydrogenase. Oxidation of acetate yields carbon dioxide, water and energy. The energy yield of alcohol oxidation is about 7 kilo calories per gram of alcohol.

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CHAPTER 2

Effects of Alcohol

Short Term Effects : These effects appear rapidly after a single dose and disappear within a few hours or days. The most predominant short term effect of alcohol is that it temporarily removes normal inhibitions. It also acts as a psychic anaesthetiser, temporarily erasing painful feelings of anxiety, worry, tension, hopelessness and anger.

The effects of alcohol are directly related to the concentration of alcohol in the blood. With the first few drinks or at a blood alcohol level of 0.03% there is an illusion of clarity of mind. As alcohol consumption increases, and when blood alcohol level reaches 0.06% consciousness becomes blurred, thinking slows down and reasoning and judgement become blunted. At a blood alcohol level of 0.09% to 0.12% physical signs of intoxication like slurred speech, clumsy movements and impaired muscular co-ordination are observed. As drinking continues and the amount of alcohol in the blood rises to 0.15% behaviour becomes irrational and the drinker becomes stuporous. At blood alcohol level of 0.5% coma (unconsciousness) sets in and at level of 1%, the respiratory centres in the brain are paralysed causing death. The concentration of alcohol in the blood depends on factors like speed of consumption, food in the stomach, body weight and percentage of alcohol in the drink.

Long Term Effects : These effects follow frequent heavy drinking over a long period of time. These are manifested in the form of severe physiological, social and psychological damages. Some of the physiological damages caused by alcohol are gastritis, fatty liver and cirrhosis, cardiomyopathy and polyneuritis. These are explained in detail in the chapter on "Effects of Alcohol on the Body Systems" Psychological damages include depression, anxiety, feelings of fear and inability to maintain good relationship with others.

The individuals' social life is also affected. Often the heavy drinker has poorly developed social skills leading to frequent quarrels at home, change of jobs, social disapproval, and involvement in crimes and violence.

Effects of alcohol on body systems

Alcohol is rapidly absorbed into the blood stream from the stomach and intestines. Once absorbed, alcohol mixes with blood and circulates throughout the body, reaching every organ. Hence most of the body systems like the central nervous system, gastrointestinal system, cardiovascular system, reticulo-endothelium system and respiratory system are affected. The type of damage to each of the systems is discussed below.

Central Nervous System : Alcohol is a depressant of the central nervous system. In small doses, it depresses those higher inhibitory centres in the brain which modulate human activities. Initially alcohol removes normal inhibitions leading to talkativeness, and excitement, associated with a general sense of well being and euphoria.

As the amount of alcohol consumed increases, the depressant action spreads to more and more areas in the brain, impairing the faculties of judgement and discrimination. At still higher doses, the vital centres of the brain are depressed causing failure of essential functions of the brain with resultant unconsciousness and death.

Gastro Intestinal System : In low concentrations, alcohol increases gastric secretion and hence acts as an appetiser. Heavy alcohol ingestion leads to erosive

gastritis or inflammation of the mucosal lining of the stomach. Gastritis occurs due to the twofold action of alcohol, namely increasing gastric acid production and increasing the permeability of the mucosal cells to the back-diffusion of gastric acid.

Liver : Synthesis of fatty acids by the liver is stimultated by alcohol, with a resultant increase and accumulation of fat in the liver cells (fatty liver). A fat infilterated liver is susceptible to damages due to infections and toxins. Ultimately it ends in scarring of the liver tissue, an irreversible condition known as cirrhosis.

Cardiovascular System : Alcohol is a vaso-dilator-dilates the blood vessels. It increases the heart rate and output of blood in circulation. Excessive alcohol use weakens the heart muscle and leads to cardiomyopathy.

Respiratory System : Alcohol depresses the respiratory centre in the brain. In cases of high doses of alcohol intake, death could result from respiratory failure.

Alcohol and Nutrition

Prolonged use of alcohol leads to a deficiency of several nutrients. Malnutrition in alcoholics is caused by several factors, the chief being alcohol's interfererce with central mechanisms that regulate hunger and appetite. A significant reduction in the levels of proteins, vitamins and minerals in the body of heavy drinkers is observed.

Proteins : A reduced serum protein level in alcoholics is brought about by poor dietary intake and decreased synthesis of proteins by the liver.

Vitamins

Vitamin A : Metabolism of vitamin A is affected by alcohol. Alcohol causes abnormal dark adaptation resulting in impaired vision during nights.

Vitamin B_1 (Thiamin) : Alcohol causes greatly reduced levels of B_1 in the body, resulting in a condition known as alcoholic beri beri. The symptoms include

heaviness of legs, parasthesias (pins and needles sensation) alteration of reflexes, anorexia (loss of appetite) and constipation. When there is severe B_1 deficiency, the symptoms manifested include opthalmophlegia (paralysis of eye muscle), ataxia (uncoordinated gait), and nystagmus (involuntary rapid eye movement).

Vitamin B_3 (Niacin) : Prolonged intake of alcohol leads to B_3 deficiency, characterised by the four D's namely Dermatitis, Dementia, Diarrhoea and ultimately Death.

Vitamin B_6 (Pyridoxine) : Another vitamin which alcohol depletes with heavy use is B_6, the deficiency of which can cause many disorders including dermatitis, anaemia and seizures or convulsions. B_6 deficiency seizures known as delirium tremens, can be observed during withdrawal.

Vitamin C : The level of Vitamin C is also decreased by drinking, leading to reduced resistance to infections.

Minerals : The levels of magnesium, calcium and zinc are greatly reduced in alcoholics due to poor dietory intake and increased urinary loss The magnesium deficiency syndrome consists of tremors, athetoid (repetitive and involuntary) movements of the extremities, mental aberrations and convulsions. Clinical features of zinc deficiency include dermatitis, usually generalised, loss of taste and slow wound healing.

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CHAPTER 3

Myths and misconception

There are many myths and misconceptions in the minds of people regarding alcohol and its use. Some of these have been clarified and discussed below.

1. Is alcohol a nutrient ?

No. Modern brewing technology completely destroys every dietary property in alcohol except for providing calories. The calories in most alcoholic beverages are known as "empty" calories since they do not provide any nutritional benefits.

2. Is alcohol an aphrodisiac ?

No. it is a sedative and in sufficient quantities an anaesthetic. People mistake it for an aphrodisiac because of its inhibition lowering effect. A large quantity of alcohol acts as a sex-inhibitor and can render a man (who is addicted to it) temporarily impotent.

3. Is alcohol a stimulant ?

No, it is a depressant, especially of the central nervous system.

4. Does black coffee/cold shower/food, help in sobering up an individual ?

No. Only time can sober him up. The alcohol in the body should be completely oxidised by the liver to restore a sober state of mind.

5. Is drinking essential in certain occupations ?

No. Drinking is never essential. Any occupation can be carried on and very well too by a non-drinker if he has the occupational abilities needed for that job.

6. Is an ability to drink more than other people, a sign of virility ?

No, but it is likely to be an early symptom of alcoholism. Most alcoholics had the capacity to drink more than their friends when they started drinking.

7. Does drinking help creativity ?

No. Alcohol releases inhibitions, and small amounts may help some people overcome certain blocks caused by inhibition. However, since even a small amount affects judgement, the results are not as good as the drinker believes.

8. Can anybody become an alcoholic ?

Yes. Any type of person can and does develop alcoholism. There is no immunity conferred by background, position in life, money, profession or occupation. Many people think that alcoholics are only found in slums, and are jobless, without family and home. It is not true. Alcoholics may also be married, employed, living with their family, just like those around us.

9. Is it true that beer drinkers never become alcoholics ?

No. There have been alcoholics who were almost exclusively beer drinkers.

10. Is it a fact that alcoholism is a disease ?

Yes. The American Medical Society has classified alcoholism as a disease and through the years this has proved to be a helpful concept in the treatment of alcoholism Once an alcoholic starts drinking he may not be able to control his compulsion to continue drinking just as T. B. patients cannot voluntarily control their coughing.

11. Can alcoholics go back to social drinking ?

Never It is not safe for an alcoholic to imbibe alcohol in any form including wine, beer, certain cough medicines/ tonics, or anything else that contains alcohol even in small quantities. Even the smallest amount of alcohol can and sometimes does trigger the disease into becoming active again.

12. Will switching drinks keep one from getting drunk ?

No. It is the alcohol content that causes drunkenness irrespective of whether it is wine, beer, whisky, gin, brandy or rum

13. Is there something wrong with people who do not drink ?

No. Drinking or abstaining from alcohol is purely a personal choice. Apart from religious and health reasons, a total

abstainer may dislike the taste or the effect of alcohol on him. He may also be aware of the long-term effects of alcohol.

14. Is drinking essential in certain social situations ?

No. One can always be firm and refuse. Unfortunately there are some persistent, ill-mannered hosts who force drinks on guests and non-drinkers, even after a polite refusal.

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CHAPTER 4

Alcoholism

Who is an alcoholic ?

There may be many reasons, but most often, alcohol is consumed because it makes people feel better. People drink for pleasure, to relax or to escape from day to day stress. Some people like its taste, others feel a social pressure and drink to conform with the group. Those who are highly inhibited find it difficult to make small talk and take to drinking as it lowers their inhibition and helps them feel comfortable with others.

The question of why people who start off drinking for such seemingly innocent reasons, end up as problem drinkers remains unanswered But it has been found out that one in every ten people who drink for social reasons become dependent on and addicted to alcohol.

Alcohol dependence can be both physical and psychological. Psychological dependence exists when alcohol becomes so central to a person's thoughts, emotions and activities that it becomes practically impossible to stop using it. The ethos of this condition is a compelling need or craving for alcohol. Physical dependence is a state wherein the body has adapted to the presence of alcohol

and if its use is abruptly stopped, withdrawal symptoms set in. The symptoms range from nervousness to tremors, convulsions, hallucinations and possibly death.

Hence an alcoholic can be defined as 'someone whose drinking causes a continuing problem in any compartment of his life'—Marty Mann.

What is alcoholism ?

In the year 1956, Alcoholism was declared a disease by the American Medical Association.

According to Keller and Effron : "Alcoholism is a chronic illness, psychic, somatic or psychosomatic, which manifests itself as a disorder of behaviour. It is characterised by the repeated drinking of alcoholic beverages' to an extent that exceeds customary dietary use or compliance with the social customs of the community and that interferes with the drinkers' health or the social or economic functioning. The characteristics of alcoholism are as follows:

1. **It is a primary disease :** Initially alcoholism was considered to be a symptom of a psychological disorder. Now, it is understood that alcoholism as such is a disease, which can cause mental, emotional and physical problems. These associated problems cannot be treated effectively until alcoholism is treated first.

2. **It is a progressive disease :** The disease progresses from bad to worse. Sometimes there may be intermittent periods of improvement, but over a period of time, the course of the disease is inevitably towards serious deterioration.

3. **The disease is terminal :** A person drinking excessively may die due to any complication, but the factor which induces the complication itself is alcohol, and thus alcohol is the real agent behind the death.

4. **It is a permanent disease :** The disease cannot be cured but can be successfully arrested by totally abstaining from alcohol Ingestion of even small amounts of alcohol will lead the person to obsessive drinking within a few days. In other words an alcoholic can never go back to social drinking even if he has remained sober for many years. Hence alcoholism is considered to be a permanent disease.

Stages of the disease

As mentioned earlier, alcoholism is a progressive disease. Three stages of progress can be identified, namely : Early stage, Middle stage and Chronic stage.

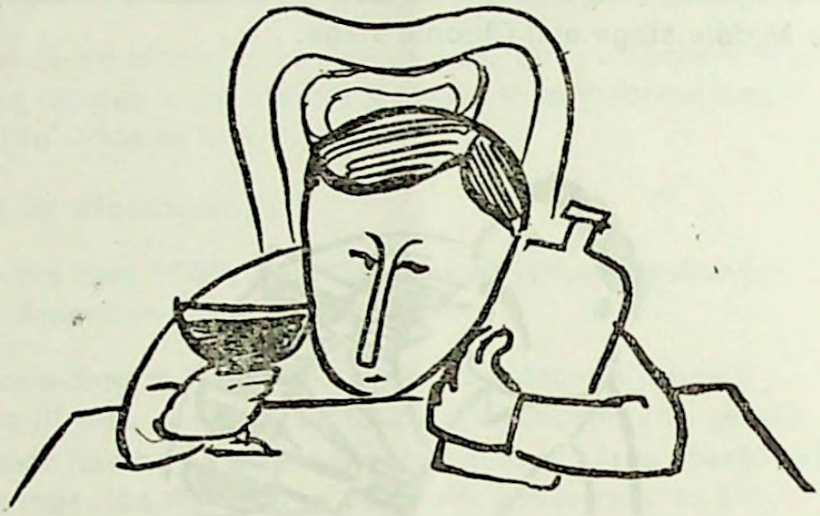
Early Stage Symptoms :

Increased Tolerance : The first warning sign for many who later develop alcoholism is a need for higher amounts of alcohol to produce the same degree of intoxication. This means, needing three or more drinks to effect mood changes where earlier only one or two was sufficient. As tolerance for alcohol increases, the individual also starts gulping his first few drinks, so that the intoxicating effect of alcohol is felt immediately.

Black-Out : Black out is alcohol-induced amnesia, wherein the person may go through many activities without being able to recall even a trace of these activities later on. During a black out, the drinker may go through his routine activities like eating, driving etc, but later on he may not recollect the details or may even deny doing them.

Pre-occupation With Drinking : Even when the person is not drinking he is always pre-occupied with thoughts of how, when and where to get his next drink.

Avoiding References to Alcohol : This symptom stems from feelings of guilt about his excessive drinking.



Middle Stage Symptoms :

Loss of Control : Initially there is loss of control over the amount of alcohol consumed. Later on with the progression of the disease, there is loss of control over the time and place of drinking. At this stage, drinking becomes compulsive.

Justifying His Drinking : The person develops an elaborate defensive system to rationalise his drinking and thus deals with his feelings of guilt and remorse.

Exhibiting Grandiose Behaviour : Another way by which the problem drinker avoids the truth about himself and his condition is by exhibiting grandiose behaviour which is usually inconsistent with both his financial and professional capabilities For example lending money to others when his own finances are low.

Aggression : It is an expression of self hatred which is directed towards others, whom the alcoholic thinks is responsible for his problems.

Abstaining From Alcohol : The alcoholic may abstain from alcohol for stated periods of time, due to social pressure, but these periods of abstinence do not last long. A person may stay away from alcohol for 30 or 40 days together at a stretch due to any religions or social reasons, but after this abstinent period he goes right back to compulsive drinking.

Changing the Drinking Pattern : The person changes his companions or places or type of drinking, with the intention of regaining control over alcohol. This is a characteristic symptom of this phase of the disease.
At this stage social relations begin to decay, old friends keep away and problems at work emerge.

Chronic Stage Symptoms

This stage is characterised by overt physical, mental and social deterioration. The symptoms include :

Binge Drinking : The alcoholic goes on a drinking spree and drinks continuously for several days at a time. At the end of each such "binge" he promises never to drink again only to return to compulsive drinking.

Decreased Tolerance For Alcohol : The alcoholic gets drunk even with small amounts of alcohol. This decrease in tolerance is due to severe physical deterioration.

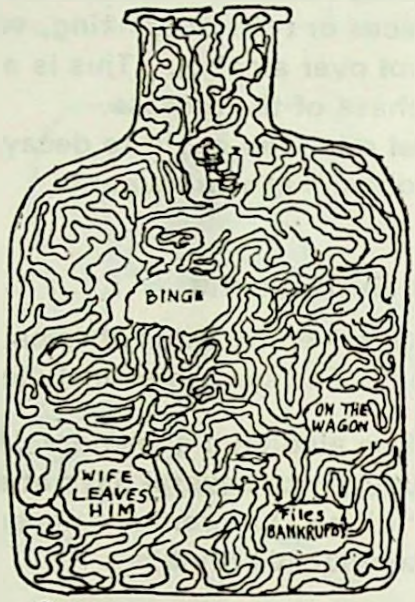
Ethical Breakdown : At this stage, there is complete ethical breakdown. The drinker is so dependent on alcohol that he will lie, borrow, or steal in order to maintain his supply of alcohol.

Feelings of Fear : The alcoholic experiences indefinable feelings of fear. For example, he might be frightened to do simple things like crossing the road.

Paranoia : The alcoholic becomes highly suspicious and believes that he would be harmed by everybody. He may refuse to eat for fear of being poisoned.

Hallucinosis : Auditory (imagining voices speaking to him), visual (seeing things) and tactile (feeling things on his skin) hallucinations are experienced by the alcoholic.

Psycho Motor Inhibitions : He loses most of his motor co-ordination. He is unable to do even simple things like tying his shoe lace until he 'steadies himself' with a few drinks. His legs and arms do not respond automatically to the minds desire. He also experiences tremors and shakes. At this juncture the alcoholic's family life, job, finances, health and every other compartment of his life gets affected. He now has three choices open to him.



GET THE ALCOHOLIC OUT OF THE BOTTLE

1. He can seek help to give up alcohol

2. He can continue to take alcohol and end up in a mental asylum

3. He can continue to take alcohol and die prematurely

★ ★ ★

CHAPTER 5

Treating Alcoholism

Alcoholism is a treatable disease. The objectives of treatment are to break the alcoholics' dependence (both psychological and physical) on alcohol and to effect positive changes In his life style. Chances of recovery are greater if treatment is started at the earlier stages of dependency. People with great deal of physical and psychological problems also have chances of recovery, though at a slower pace. Treatment of alcoholism is brought about through medical and psychological methods.

These are aimed at three levels.

1. Management of acute episodes of intoxication to save life and to overcome the immediate effects of excessive alcohol consumption.
2. Remedy for chronic health problems associated with alcoholism.
3. Changing the attitude and personality characteristics of the alcoholic individual so that he is able to improve the quality of his life by abstaining from alcohol.

Management of Acute Episodes of Intoxication :

When a person is acutely intoxicated, the concentration of alcohol in the blood rises to toxic levels. Detoxification is the process by which toxic levels of alcohol is eliminated from the body. This process is an integral part of the treatment procedure which requires hospitalisation and the attention of specialists. Detoxification allows people to recover from the effects of intoxication in a supportive and comfortable atmosphere. People who are addicted to the drug alcohol, depend on the continuous consumption of the drug to attain a feeling of well being. So, sudden withdrawal of alcohol from the body results in an abrupt cessation of the depressant effect of alcohol, leading to the hyperactivity of brain cells. This leads to several withdrawal symptoms which can be mild or severe. These symptoms include

tremors, anxiety, sleeplessness, visual and auditory hallucinosis (hearing and seeing things) paranoia, sweating, fever, tachycardia (rapid heart rate) and tachypnea (rapid respiration)

During detoxification, glucose, vitamins and other nutrients are given to improve the general health of the individual.

Management of Chronic Health Problems:

Some of the chronic health problems associated with alcoholism are gastritis, cardiomyopathy, neuritis, fatty liver and cirrhosis. These complications have to be medically treated by specialists from respective fields.

Changing the attitudes and personality characteristics of Alcoholics.

Management of acute intoxication and health problems associated with alcoholism does not complete the treatment picture. Treatment should also effect changes in the behaviour and thus the life style of the alcoholic individual. This is brought about by psychological treatment in the form of individual, group and family therapy. This helps the patient to bring to light, forgotten or repressed feelings of anger, guilt, shame, fear and low self-esteem, and also to deal with them in the present.

Psycho-social techniques

Psycho-social techniques help in bringing about positive personality changes which is a pre-requisite to abstaining from alcohol.
This technique comprises of individual psycho-therapy, group therapy, family therapy and relaxation methods.

Individual Psycho-Therapy: Here the therapist enables the patient to sort out his problems in interpersonal relations and to analyse his personality make up. This helps him to develop positive coping mechanisms.

Group Therapy: Here the patient interacts with others who are also dependent on alcohol. During group therapy, members share and discuss the problems in their lives. In these sessions they are free to confront each other and

clarify the issues that are being discussed This enables every individual to work through in actual interchanges with others, some of the underlying emotional conflicts that have been keeping him or her defensive, frightened, anxious or depressed.

Family Therapy : This is directed towards the family of the alcoholic with a view to overcome the family members' guilt or resentment against the alcoholic.

Relaxation Methods : It is often found that the alcoholics have problems in handling tension and anxiety. They are unable to relax or sleep well. Relaxation methods help to rectify these problems.

Quite often. the treatment procedure also includes the use of drugs, namely antabuses. These drugs are especially helpful to those alcoholic patients who want to stop drinking but have periodic motivation to return to alcohol. These drugs are usually administered during the initial stages of recovery. If alcohol is imbibed when already on the drug, adverse effects are brought about. These effects are due to the chemical "disulfiram" in the drug. This chemical interferes with the metabolism of alcohol, resulting in an accumulation of acetaldehyde in the body, which is responsible for the adverse effects including nausea, vomitting, sweating, palpitation and at times even death. Thus it can be seen that the treatment of alcoholism requires a multidisciplinary approach and the help of specialists from various fields. The co-ordinated work of such professionals helps the alcoholic to recover.

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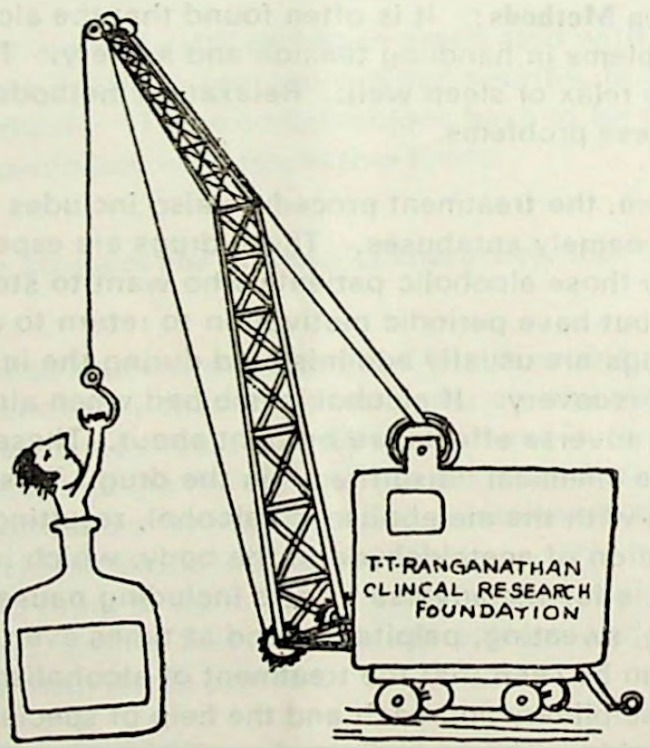
Treatment at T. T. Ranganathan Clinical Research Foundation

The treatment at T. T. Ranganathan Clinical Research Foundation is based on the concept that alcoholism is a treatable disease. By tackling each individual's problems with a multi-faceted programme, the Foundation helps him to take his place as a reliable and responsible member of society.



The first stage, of treatment involves detoxification, which requires hospitalisation, for a period of one week to ten days depending on the severity of the patient's condition. After discharge from hospital the patient undergoes an intensive three week therapeutic programme at the Foundation. Therapy at the Foundation comprises of individual counselling, lectures, group and family therapy, dietary counselling, relaxation and transcendental meditation and films on alcohol use and abuse.

After treatment at the Foundation there is a rigorous follow up programme wherein the patients are expected to call at the Foundation once in 10 days or 15 days for at least a year. It includes medical check up along with counselling sessions and after care group meetings.

A social support system has also been mobilised to help individuals who have already taken steps to give up alcohol. The social support programme aims at exploring the possible support the recovering people have, from the society in which they live.

The patients are also encouraged to attend Alcoholics Anonymous meetings after treatment at the Foundation.

CHAPTER 6

Guidelines for the Family of Alcoholics

Alcoholism is an illness which has tremendous emotional impact upon the immediate family. The disease not only affects the alcoholic, but also hurts the people close to him. The family members require assistance and counselling in order to help the alcoholic overcome the disease. A few guidelines for the family to deal adequately with alcoholism are given below.

— Learn all the facts about alcoholism

— Accept these facts. One cannot accept an alcoholic as a sick man in need of help if he is going to be blamed for all actions resulting from the illness. Changing one's attitude and approach to the problem can speed up recovery.

— Try to remain calm, unemotional and factually honest in speaking with the problem drinker about his behaviour and its day to day consequences.

— Be patient and live one day at a time. Alcoholism generally takes a long time to develop and recovery does not occur overnight.

— Try to accept set backs and relapses with calmness and understanding.

— Establish and maintain a healthy atmosphere in the home, and try to include the alcoholic member in family life.

— Explain the disease concept of alcoholism to the children.

— Encourage the alcoholic to participate in leisure time activities.

— Discuss the problem with someone you trust

Given below are a few things the family should avoid doing :

— Do not hide or serve liquor.

— Do not argue or quarrel with the person while he is drunk. Asking him why he drank does not help in anyway.

— Do not accept the lies of the alcoholic, especially regarding his drinking, expenses etc., as the truth.

— Do not allow the alcoholic to take advantage of the family members' vulnerability.

Such as asking the wife to get loans from friends or her parents; or expecting the wife to feed him when he is drunk.

— Do not accept promises of giving up alcohol from the alcoholic. The wives usually make the alcoholic promise never to drink again on God or the children. This does not help him to give up alcohol or control his drinking.

— Do not cover up the consequences of drinking. This reduces the crisis but perpetuates the illness. For instance when he is involved in an accident due to drinking, do not be supportive.

— Do not attempt to punish, threaten, bribe, preach or try emotional appeal on the alcoholic.

Punishment : "I will not cook, if you drink"

Bribes : "If you don't drink, I will clear debts with the help of my father"

Threats : "If you drink, I will leave home or I will commit suicide".

Emotional appeal : "If you love me or if you love the children, You will not drink again."

— Do not take up the responsibilities of the alcoholic, like Paying off his debts

— Do not feel guilty for his behaviour.

— Do not put off facing the fact that alcoholism is a progressive illness. To do 'nothing' is the worst choice one can make.

— Do not expect 100% recovery.

— Do not create an impression that he is giving up alcohol for the family. He has to give up alcohol for his own self.

— Do not justify his drinking by agreeing with the rationalisations of the alcoholic.

CHAPTER 7

What is Alcoholic Anonymous?

Alcoholics Anonymous is a world-wide fellowship of men and women who help each other to stay sober. They offer the same help to anyone who has a drinking problem and wants to do something about it. Since they are all alcoholics themselves, they have a special understanding of each other. They know what the illness feels like and they have learnt how to recover from it in AA

AA members say that they are alcoholics today-even when they have not had a drink for many years. They do not say that they are 'Cured'

Once people have lost their ability to control their drinking they can never become 'former alcoholics' or ex-alcoholics.

But in AA they can become sober alcoholics, and recovered alcoholics.

They share their experiences, strength, and hope with each other so that they may solve their common problem and help others recover from alcoholism.

The only requirement for membership is a desire to stop drinking.

There are no dues or fees for AA membership : They are self-supportive through their own contributions.

AA is not allied with any sect, denomination, political organisation or institution; it does not endorse or oppose any cause. The primary purpose is to stay sober and help other alcoholics to achieve sobriety.

How does AA help the alcoholics? : AA offers help to the alcoholic in the form of friendship and understanding. AA welcomes the alcoholic not "in spite" of his drinking but "because" of his drinking It provides opportunities for sympathetic mutual discussions with other members thereby helping the alcoholic in relieving his burden of complexes and self recriminations. It advises the alcoholic to seek medical help, if there are any physical problems, to turn to God for spiritual well being and to set right all the past mistakes, as far as possible, in order to relieve one's inner conflicts.

CHAPTER 8

About Us

The T.T. Ranganathan Clinical Research Foundation is the first of its kind in the country dedicated to the treatment and rehabilitation of people addicted to alcohol and other drugs.

It is also engaged in Research work on various aspects of alcohol and drug abuse. The Foundation carries on education work for prevention of alcohol and drug abuse.

The treatment programme is conducted by a team of dedicated professionals like psychiatrists, clinical psychologists and social workers. Individual needs of the patients are catered to by the multi-discilpinary approach of the professional team.

An expert committee consisting of eminent doctors and other professionals concerned with the disease of alcoholism advises the Foundation on the latest development in treatment methodology.

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For Further information Contact :
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Working Hours : Monday to Friday : 10.a.m. to 4.30.p.m.
Saturday 10.a.m. to 1.p.m.

Printed at :
M. W. N. PRESS
27, LLOYDS ROAD
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A Word about Ourselves

The T. T. Ranganathan Clinical Research Foundation is the first of its kind in the country, dedicated to the treatment and rehabilitation of alcohol dependants.

A team of dedicated professionals treat alcohol dependants, both in and out of hospital. The staff includes doctors, social workers, psychologists and psychiatrists.

An Expert Committee consisting of eminent doctors and other professionals concerned with the disease of alcoholism advises the Foundation on the latest developments in treatment methodology.

The clinic offers a course of intensive treatment for the patient and his family. To begin with the patient is withdrawn from alcohol through medication. The first step involves detoxification, a process that requires hospitalisation. The duration of stay depends on the severity of the patient's alcoholism.

The next stage is of utmost importance. The patient after being discharged will have to maintain his sobriety. The T. T. Ranganathan Clinical Research Foundation assists this process of recovery with individual counselling, group counselling, nutrition therapy and relaxation therapy. The patient also attends Alcoholic Anonymous meetings every week.

The T. T. Ranganathan Clinical Research Foundation regards alcoholism as a treatable disease. It underlines the fact that there is no need for the problem-drinker (at any phase) to feel that he has passed beyond the point of no return. His family need not develop pessimistic attitudes towards chances of his recovery. The Programme aims at changing attitude and behaviour and developing positive traits in the personality of the individual. By tackling each individual problem with a multi-faceted programme and with care and treatment, the Foundation helps to put these people back on their feet, and take their rightful place as responsible members of the society.

T. T. Ranganathan Clinical Research Foundation
91, Santhome High Road,
Madras - 600 028.
(Opp. Ayyappan Temple)

British Journal of Addiction to (1985), 755–763

(DR THOMAS MT)

International Review Series: Alcohol and Alcohol Problems Research
4. Canada

Reginald G. Smart
Addiction Research Foundation, 33 Russell Street, Toronto, Ontario, Canada.

Summary

Canada has been a leader in alcohol research for several decades. This research, both in Canada and elsewhere, is often associated with a Temperance Movement. In Canada, several of the most important alcohol research centres were established with the support of the Temperance Movement. The largest alcohol research agency is the Addiction Research Foundation in Ontario and it accounts for most of the alcohol research money spent in Canada. It has a research budget of about $8 million and about 50 scientists on staff. Currently there are 31 lines of research and a much larger number of projects. Provincial agencies outside Ontario typically do very little research, except to evaluate their own programmes. In addition, some grants for alcohol research are made by medical research agencies. Several important approaches in alcohol research can be identified as typically Canadian. At present, research funding is not being expanded and actual declines in support have occurred in some areas.

A Introduction

Countries with ambivalent attitudes about alcohol tend to have more alcohol research than those with consistent attitudes [1]. This is the case with Canada, a world leader in alcohol research for several decades. We have a long tradition of social support for the Temperance Movement, side by side with traditional drunkenness among the Indians, pioneers, miners, railway-workers, lumbermen and soldiers who originally built the country. Canada now has close government control of all aspects of the manufacture, distribution and sale of alcohol, a system which contrasts with that found in more liberal wine-growing countries. However, popular support for drunkenness still remains in Canada; national and local cultural events, for example, New Year's Eve, the Grey Cup football game, and indeed most spectator sports, often involve heavy drinking. As with Scandinavian countries, there is excellent support for alcohol research which flows out of a long preoccupation with drinking as a social problem.

It is interesting to examine some of the historical traditions around alcohol and the way Canadian society supports alcohol research. Such research is done to some extent in universities on a grant basis by the federal government via Health and Welfare Canada and by various provincial alcohol commissions and foundations. Undoubtedly the largest contribution to research has been made by the Addiction Research Foundation (A.R.F.) in Toronto. Its research and many of its researchers have become internationally well-known and hence its activities are a prominent part of this review. However, efforts are made to examine the role of various university research groups and provincial alcohol commissions outside Ontario. Because of my long association with A.R.F., biases toward that organization are impossible for me to avoid. Readers should take this paper as a personal view, coloured greatly by my own experience and they are advised not to look for too much objectivity.

B Historical perspectives

(i) *Pioneer times and alcohol*

Alcohol was not a problem for the first Canadians — the Indians and Eskimos who inhabited Canada before the Europeans. However, soon after the immigrations from Britain and France alcohol problems arose, became entrenched, and have remained largely unsolved ever since. Neither Indians nor Eskimos discovered the art of

The views expressed in this publication are those of the author and do not necessarily reflect those of the Addiction Research Foundation. The author wishes to thank R. E. Popham, M. Shain and A. C. Ogborne for their comments on this paper.

[1] For example, the Gospel Temperance Movement founded by D. J. K. Rine, a former alcoholic, concentrated only on drunkards and skid-row derelicts in the 1880s.

fermentation but when the British brought rum and the French brought brandy to North America they were among the heaviest users. There soon began a series of government alcohol control activities and anti-drinking movements which have continued to the present day.

Reports of early missionaries, such as the 'Jesuit Relations' [2], contain hundreds of references to drunkenness among Indians and early settlers. For example, 'Every night is filled with clamors, brawls, and fatal accidents, which the intoxicated cause in the cabins.' and 'It (drunkenness) is so common here and causes such disorders, that it sometimes seems as if all the people of the village had become insane so great is the licence they allow themselves when they are under the influence of liquor.' [2]. French authorities made many unsuccessful efforts to control drinking by Indians, as did the Hudson's Bay Company, which eventually prohibited trade in alcoholic beverages.

Drinking was also a problem among many pioneers and traders whose drinking was often as heavy as that of the Indians. Pioneer inns and taverns were very numerous and they were usually the first buildings put up in new settlements [4]. Logging, mowing and barn-raising bees were common and it was the custom to provide a gallon of whiskey per attending family [3]. Susanna Moodie [5] writing in the 1840s, found that bees were, 'noisy, riotous, drunken meetings often terminating in violent quarrels and sometimes even in bloodshed.'

(ii) *The temperance movement and alcohol research*

The 'Temperance Movement' began as a response to the excessive drinking in early Canada. The movement derived most of its support from Methodist, Presbyterian and Baptist churches and became part of the 'Social Gospel' movement of the early 1900s. Canadian temperance movements had many activities similar to those of modern alcohol commissions or foundations. For example, the Women's Christian Temperance Union (W.C.T.U.) tried to have alcohol education courses, called 'scientific temperance', introduced into schools and was successful in several provinces.

Temperance movements always took some interest in rescuing drunkards and some Canadian movements did this exclusively.[3] In addition, temperance movements frequently challenged municipal, provincial and federal governments to change the licencing laws to make alcohol less available and more expensive. Much of what they said was not very different from the so-called 'neo-temperance' approach now attributed to A.R.F., although their arguments lacked scientific sophistication.

Temperance organizations often gathered data and first-hand experiences about drinking. We might not call it 'research' but at least it was intelligence gathering. The Dominion Alliance for the Total Suppression of the Liquor Traffic and the W.C.T.U. constantly bombarded governments with statistics on alcohol sales, the extent of drunkenness, and the economic costs of alcohol, or 'the drink bill' as it was called. Although biased and unsophisticated, these reports do represent a rough early approximation to social research.

When various provincial foundations were established after the Second World War they were often supported by temperance leaders in the legislatures. Several of the foundations had temperance members on their boards or as chief executives, at least in the beginning. To some extent, modern alcohol agencies took over (and greatly improved) the work of the temperance organizations in treatment, education and research. In general, countries with a strong temperance tradition, such as North America, England and Scandinavia, support more alcohol research than those with no such traditions, as in Latin America and Western Europe.

C Current alcohol research organizations in Canada

(i) *Overview*

Health services in Canada are largely a provincial, rather than a federal government concern. There is, therefore, considerable Balkanization in health-related efforts and alcohol research is no exception. Provinces are responsible for the provision of most health and education services, except for some native groups and the armed forces. Also, they are responsible for the education of health professionals through the universities, and for the administration of health insurance schemes. However, the federal government does contribute financially to both health and education through a complex system of grants to provinces.

Several mechanisms exist for funding alcohol research. The Federal Department of Health and Welfare conducts some alcohol studies internally, chiefly epidemiological research and evaluations of its prevention programmes. Also, several federal agencies, such as the Medical Research Council and the National Health and Welfare Development Program, give grants to universities for alcohol-related projects. In addition, all provinces, except Quebec and British Columbia, have an 'Alcoholism' or 'Drug Dependency' Commission or Foundation, although only Ontario's has 'research' in the title. By far the largest, in terms of both total budget and research effort is A.R.F. in Ontario (Table 1).

Table 1. *Provincial alcohol agencies doing research in 1984*

Agency	Location	Total budget[1]	Research budget[2]	Research staff[3]	Research effort (1984)[3]	Per cent of budget spent on research
Addiction Research Foundation	Toronto Ontario	$28,462,460	$8,297,492	50[3]	37 lines 171 published books, book chapters, papers	29.0%
Alberta Alcoholism and Drug Abuse Commission	Edmonton Alberta	$24,253,326	$350,000	9	7 projects 6 reports	1.4%
Alcoholism Foundation of Manitoba	Winnipeg Manitoba	$7,839,491	None Specifically Allocated (about $40,000?)	1	14 reports	0%
Saskatchewan Alcoholism Commission	Regina Saskatchewan	$3,668,406	$186,000	4	7 projects	5.0%
Nova Scotia Commission on Drug Dependency	Halifax Nova Scotia	$5,860,400	$180,000	4	about 6 projects	3.1%
Alcoholism and Drug Dependency Commission of New Brunswick	St. John New Brunswick	$5,872,977	Not Available (about $160,000?)	4	10 projects	2.7%

[1] This information has been derived from the Annual Reports of the various Commissions and Foundations.
[2] This information has been obtained from personal correspondence.
[3] Scientist positions only.

(ii) *The creation and early development of research at the Addiction Research Foundation*

Prior to the Second World War, there was almost no research on alcohol problems in Canada. Various Royal Commissions had collected statistical data and the opinions of presumed experts, such as physicians, since at least 1895. Otherwise original research did not exist beyond a few projects on basic mechanisms at the biochemical level. Social research, epidemiology, and the total alcohol policy and alcohol treatment field were largely undeveloped. In fact, temperance organizations provided analyses of government figures on morbidity and mortality from alcohol, together with their own interpretations, but this research was often highly biased. The major actor in the alcohol field was Alcoholics Anonymous, an organization which had come to Canada in the early 1940s [6]. They had no interest in alcohol research but did help to interest H. D. Archibald, the first Executive Director of A.R.F., in the problems of alcoholism.

The Foundation, originally called the Alcoholism Research Foundation, and now officially the Alcoholism and Drug Addiction Research Foundation, was established for a number of reasons. 'Liquor by the drink' had been introduced into Ontario in 1947 in bars which had only been beer parlours before. This change led to much criticism of the government. At that time, there were no public treatment facilities for alcoholics in Ontario, except for mental hospitals which took mainly cases complicated by serious mental illness. A need for treatment facilities was recognized by many groups. A prime mover was H. D. Archibald, a lecturer in social work at the University of Toronto. It is most unlikely that A.R.F. would have been established as a research centre without his influence. A certain number of recovered alcoholics also argued for its creation. In addition, Temperance interests were in favour of a new treatment centre for alcoholics. A number of Temperance leaders actively supported the idea of treating alcoholics and one, William Temple, argued in the legislature that: 'it is high time this province did something to care for the victims of the alcoholic traffic, instead of giving all their energy to promoting the sale of liquor.' The wife of the Premier, Leslie Frost, was a prominent Temperance leader and is said to have been influential in creating A.R.F.

The first suggestion for a name was the *Alcoholic* Research Foundation and legislators agreed only to a treatment centre. The idea that research would become so prominent seems not to have occurred to those present at the creation of A.R.F. The provincial governments, then as now, were not heavy supporters of research in social or medical problems. However, when the Act incorporating A.R.F. was passed in 1949, it was termed the *Alcoholism* Research Foundation, a change suggesting a wider mandate than research on 'alcoholics'. Archibald has stated [6] that the stress on treatment was a strategy developed to suit the times as any stress on research in the debates might have provoked hostility from the legislators.

Research was not very important at A.R.F. for the first few years. The first annual report stated that 'in preference to setting up its own staff it will make grants to universities, colleges and hospitals' [7]. In the first year there were three external grants given for a total of $9,450; by 1953 there were 31. However, in 1954 an internal research department was established with R. E. Popham and R. J. Gibbins as the principal investigators. They had been part of the earliest grants programme. Although the research grants programme was maintained until 1974, payoff in terms of publishable research often seemed inadequate. However, grants and fellowships for graduate students did continue, and served to encourage some research workers who eventually became part of the intramural research effort.

At first, virtually all of the research was done in the Research Division, including a biological research unit established in 1959 in the Department of Pharmacology under Dr H. Kalant. By 1970, the intramural enterprise had a staff of about 50. In 1970, research was begun in the newly-formed Clinical Institute, a university-affiliated hospital, clinical research, and teaching centre, for professionals in the addictions field. In 1979 research was extended into the Regional Programs Division with the establishment of a special unit, mandated to concentrate on the evaluation of community development programmes.

The original concept of research at A.R.F. was that researchers should have independence in choosing their own projects [8]. Much emphasis was put on hiring the right person and then letting him decide on his own projects within the mandate of A.R.F. In the early days of Research Division there was little control on who could do what project. Scientific curiosity was given free reign and interdisciplinary projects were fostered. Control on what projects were done was exercised informally through collegial relationships, rather than executive fiat.

In my opinion this system had many dividends in exciting and productive research. A number of high-risk/high-profile projects were begun that many administrators and granting agencies would have refused to support. In one of these, research assistants spent years counting and deciphering the names and addresses on forms which purchasers of alcoholic beverages had to complete in order to buy alcohol from liquor stores. The slips for individuals were then aggregated to form a picture of the distribution of alcohol use in the population of Ontario. The report [9] stimulated an important line of research which led to the so-called Single Distribution Theory of alcohol consumption, with major implications for prevention [10]. Both the ethical and financial aspects of this work would have discouraged most granting agencies. Of course, the freedom for researchers generated some problems. Sometimes the wrong researchers were chosen or some work wandered off into unproductive areas for too long. These problems became more obvious with time and, in the view of some, began to outweigh the benefits.

In the 1970s a growing concern with accountability of researchers at A.R.F. and elsewhere developed. Freedom to 'do one's own thing' disappeared as clear goals and objectives were laid out for the organization and for the researchers. Budgets became tighter and research positions more difficult to find. Review of projects on an individual basis was instituted in 1976 for both scientific adequacy and ethical acceptability. Also, there is now an internal review of total research effort done every few years and a comprehensive external review utilizing outside experts every 5 years. Clearly this has meant more paperwork, committee meetings (and headaches) for A.R.F. scientists. However, there seems to be no major impact on productivity; probably the extra time needed is taken from collegial contacts and the large amount of informal interaction which was featured in the past.

Early influences on the research programme at A.R.F. included many people who sadly are now dead or have left the field. In the early 1950s J. K. W. Ferguson began work on anti-alcohol drugs, such as disulfiram and developed a new one, calcium carbimide (Temposil), which is still widely used. This continues to be a topic of research, although in a greatly modified form. E. M. Jellinek was at A.R.F. in 1958–59 and later went on to the Alberta Alcoholism and Drug Commission. While in Toronto he generated interest in his formula for estimating the numbers of alcoholics from liver cirrhosis mortality; this line continued for some years. His 'Disease Concept of Alcoholism' was influential in that Canadian researchers often debated the existence of such an unlikely disease, but the 'disease concept' probably did contribute to increased funding for alcohol problem research. In the 1950s we were influenced by Finnish researchers, such as Pekka Kuusi and Kettil Bruun, who were interested in alcohol policy and studying the effects of policy changes. Leonard Goldberg of Sweden had an important influence on the establishment of research on both tolerance and dependence and drinking-driving. A further influence was H. Ward Smith, whose work with R. E. Popham in the early 1950s on drinking-driving, generated a line of research which is still vigorous. Around 1959, John Seeley, the first Director of the Research Division, began to study the effect of real price on alcohol consumption. He awakened interest in this line by suggesting the possibility that the prevalence of alcoholism could be reduced through manipulation of alcohol taxes [11].

(iii) *The current research programme at the Addiction Research Foundation*

Currently, research at the Foundation is done in three main areas: the Social and Biological Studies Division; the Clinical Institute (a university-affiliated and teaching centre); and the Community Programs Evaluation Centre in London, Ontario. For the most part, epidemiological, programme development, and social policy relevant research is done in the Social and Biological Studies Division. Basic pharmacological and biochemical research is also done there. Most research on clinical problems and treatment assessments are done in the Clinical Institute. Evaluation of A.R.F. community development programmes is done in the Community Programs Evaluation Centre. Currently there are about 50 staff scientists engaged in research, with a research budget of $8,297,492.

The research programme at A.R.F. is large and involves many different lines. In 1984 there were more than 31 lines concerned mainly with alcohol problems and an additional eight lines concerned with non-alcoholic drugs. Each line involves several projects and results in a number of publications. For example, in fiscal year 1983–84 there were 171 publications by Foundation staff, including books, book chapters and journal articles, plus a number of unpublished internal reports.

The 31 lines involving alcohol are listed below, together with the names of the people currently responsible for them.

Social and Biological Studies Division

Acting Head of Division: W. Schmidt. Social Policy Research; Acting Head: E. Single.

1. Compilation of alcohol and drug statistics.
2. Studies of measures to control alcohol problems.
3. Adverse effects of alcohol use.

Programme Development Research: R. G. Smart.

4. Research on drinking/driving practices and new programmes.
5. The extent of youthful drinking and drug use and the effects of legal controls.
6. Development and testing of educational programmes for students and other high-risk groups.
7. Development of methods of preventing and treating alcohol and drug problems in employed populations.

Biobehavioural Research: H. Kalant.

8. Studies of factors governing self-administration of alcohol and other drugs.
9. Studies on mechanisms of tolerance to alcohol and other drugs.

Clinical Institute

Biomedical Research; Research Co-ordinator: R. C. Frecker.

Clinical Pharmacology: C. A. Naranjo.

10. The pharmacotherapy of alcoholism.

Gastroenterology: H. Orrego

11. Clinical evaluation and treatment of alcoholic liver disease.
12. Clinical research relating to portal hypertension and portal systemic encephalopathy.
13. Investigations into the pathogenesis of alcoholic liver disease and interaction between alcohol and anaesthetic agents.

Neurology: P. L. Carlen.

14. Study of the organic brain syndrome and its reversibility (neurological aspects).
15. Portal-systemic encephalopathy.
16. Cellular neurophysiological studies into the mechanisms of acute intoxication and alcoholic brain damage.

Psychiatry: F. B. Glaser.

17. The development of alcohol and drug problems in a professional population.
18. The epidemiology of psychiatric disorders in patients with alcohol and drug problems.
19. The treatment of alcohol problems through an integrated pharmacological-psychological approach.

Biochemical Research: Y. Israel.

20. Studies on the effects of alcohol in the liver.
21. Development of markers of alcohol consumption.

Behavioural Research: H. Cappell.

22. Behavioural treatments.
23. Experimental behavioural research.
24. Neuropsychological research.

Health Care Systems Research: H. Annis.

25. Identification and assessment of alcohol problems.
26. Low-cost interventions and treatment matching.
27. Relapse prevention training.
28. Follow-up systems studies.

Community Programs Evaluation Centre

Community Programmes Evaluation: M. Faveri.

29. Evaluation of programmes designed to improve treatment systems in Ontario.
30. Evaluation of employee assistance programmes.
31. Evaluation of public education and health promotion programmes.

Many of these research lines have been in existence for 20 years or more, for example, those concerned with drinking-driving (No. 4), measures to control alcohol problems (No. 2), mechanisms of tolerance (No. 9),

pharmacotherapy of alcoholism (No. 10), etc. The projects and staff involved may have changed frequently but a consistent thread could be seen. On the other hand, several lines are relatively new, for example, epidemiology of psychiatric disorders (No. 18), identification and assessment of alcohol problems (No. 23), and the evaluation of programmes to improve treatment systems (No. 29). In general, research lines change slowly and even the 'new' lines are several years old. A tendency at A.R.F. is for scientists to have long careers and to study their area of interest for many years. Research facilities are excellent and consequently A.R.F. provides one of the few places in Canada where scientists can develop a career in alcohol studies.

(iv) *Provincial Alcoholism Agencies outside Ontario*

Research in provincial alcoholism agencies is on a much smaller scale than at A.R.F. These agencies serve provinces with a smaller population than Ontario. Most have as their main purpose the provision of treatment services, with a smaller educational component and few have any mandate or funds to do research, except on their own programmes. Table 1 summarizes some of the basic data on alcohol research in the provincial commissions. Clearly, most employ few researchers and have small research budgets. Nova Scotia, New Brunswick and Saskatchewan each have four research positions; Manitoba has one; and Alberta has nine. The total budget for alcohol research outside of A.R.F. is only about $936,000 (assuming $40,000 per position for those provinces not specifying a budget). The total involves 22 people nationwide, or less than half the number of researchers at A.R.F. The commissions spend between 0 and 5.0 per cent of their budget on research, while A.R.F. spends 29 per cent.

Research programmes in agencies outside Ontario are mostly created in order to serve programme needs. The major purpose of most research in the commissions is to provide an in-house data collection service for administrators and policy staff. For example, the Alberta and Nova Scotia Commissions have both studied their extensive primary prevention programmes for adolescents and evaluated most of their inpatient and outpatient programmes, as well as compiling survey data on adolescent alcohol and drug use. Recent research in New Brunswick has been devoted to determining outcomes of the Commission's treatment programmes and to the analysis of various statistics on local alcohol use and resulting problems. Research in the Alcoholism Foundation of Manitoba is just beginning and only a few externally funded projects have been undertaken in recent years.

Very little of the research done in the smaller commissions is widely available in the international audience. Several agencies seem to confine publications to internally or locally available reports. However, the Alberta, Nova Scotia and Saskatchewan Commissions do make their reports available to the general public of the province, legislators, and those interested in addictions. Almost no research from the provincial commissions is published in the open scientific literature such as journal articles, books or book chapters. This contrasts greatly with research at A.R.F. where virtually all research is eventually published in journals or books and internal reports are discouraged.

(v) *Research grants to university researchers*

Currently, few grants seem to be made in Canada for alcohol-related projects. The National Health Research Development Program funded four projects in Canadian universities in 1984–85 for a total of $204,494. Two of these deal with the effects of maternal alcohol use on offspring. One is concerned with alcohol consumption and blood pressure, using data from the Canada Health Survey. The last examines the aetiology of fatal traffic accidents involving alcohol and cannabis. This project is being carried out by the Traffic Injury Research Foundation (T.I.R.F.), a small research group in Ottawa with a continuing interest in research on alcohol-related accidents.

The Alcoholic Beverage Medical Research Foundation gave four grants to Canadian researchers in 1983–84. All were relatively small and the total amounted to $93,544 U.S. Two were concerned with the effects of alcohol on membrane function. One was a grant to T.I.R.F. for a study of alcohol-related accidents among youth. Finally, a small grant ($5,000 U.S.) was given for a time-budget study of drinking patterns.

The largest supporter of health research in Canada, the Medical Research Council, gave six grants in 1984–85 for alcohol projects for a total of $218,000. Four of these grants dealt with the fetal or neo-natal consequences of heavy alcohol intake, obviously a prime area for funding agencies. One concerned basic mechanisms of tolerance, and the other the effects of high alcohol intake on liver disease.

Lastly, the Ontario Mental Health Foundation gave two grants for alcohol research in 1984–85 for a total of $50,137. One dealt with the effects of alcohol on memory and the other with methods of screening alcoholics in a psychiatric population.

In general, alcohol research is not heavily supported by granting agencies. In 1984 a total of 16 grants can be identified for a total of $566,175. Probably some grants are missed in this accounting as smaller agencies supply little information on the topics of research grants they support. Also, fellowships and scholarships are difficult to count as there is no information on the research area in which they are held. It certainly seems that the majority of the funding for alcohol research in Canada comes from the provincial commissions, chiefly A.R.F. in Ontario.

D Some Canadian themes in alcohol research

Some general themes have appeared in Canadian research on alcohol and a few have been adopted in other countries. Many of them appeared early and have continued into the present. One important theme is to emphasize epidemiology, especially general epidemiology. Much research is concerned with drinking and alcohol-related problems in the whole population. Scientists at A.R.F. were among the first to point out that the drinking problems in a society was closely associated with the overall level of drinking. This research began more than 20 years ago and continues to the present in an effort to establish a biomedical definition of safe level of consumption. Preoccupation with general epidemiology has led to a neglect of clinical epidemiology, a general untapped area in Canada. It is also associated with a relative lack of interest in the 'disease concept of alcoholism', studies of the 'alcoholic personality' and of acute social problems associated with alcohol use, such as child neglect and marital conflict. There has been a growing tendency among Canadian researchers to view alcoholics as not very different (except in degree of drinking) from other kinds of drinkers. Probably this is why some treatment studies of alcoholics have involved training in social drinking. The idea that some alcoholics could return to social drinking (or try it for the first time) after appropriate treatment does not strike most Canadian researchers as radical.

Another theme in Canadian research has been to emphasize studies relevant to government alcohol control policies. Clearly, we have this in common with our Scandinavian colleagues. In any case, Canadians like a high level of government involvement in their lives; they expect government to be involved in the control of social problems and to serve their interests. Interest in alcohol control policy began very early and by the 1850s Prohibition was being sought throughout the settled areas of Canada. All provinces had Prohibition at least for a short period and the present control system developed as an alternative to Prohibition. Alcohol researchers in Canada have often oriented their work to studies of overall availability, alcohol price controls, and to a myriad of studies of small changes in the control systems, such as in opening hours, drinking age, drinking-driving laws, modes of retailing liquor, and the like. A general aim has been to search for the best government control solutions to the alcohol problem. In many countries the main problem is seen to be the drinker and he is the object of prevention research. In Canada, the objective is much more likely to be changes in government policies and procedures for making alcohol available.

Another theme in Canadian research is to de-emphasize treatment studies. With the exception of research on pharmacotherapies and a little on controlled drinking in alcoholics, searching for the best treatments for alcoholics had been of little long-term interest. To some extent this is changing as sociobehavioural treatment and treatment-systems research is being developed at A.R.F. However, much of this has been concerned with treatment system efficiency or with discovering how patient characteristics and treatment interact to produce the maximum benefit. Treatment research in many of the smaller provincial agencies consists essentially in monitoring the progress of patients through the available programmes, rather than in discovering new treatments and evaluating their effectiveness.

E The practical impact of alcohol research in Canada

It is difficult to assess how alcohol research done in Canada has determined policies for the management of alcohol problems. Certainly there are some major successes in the treatment area and space allows that only a few can be pointed out here. Early work on disulfiram and other antialcohol drugs at A.R.F. led to the development of new treatments for alcoholism. Research following that in other countries which showed that inpatient and outpatient care of equal likelihood of success has been important in controlling the growth of expensive inpatient care. The development of propylthiouracil (P.T.U.) by Dr Y. Israel and his colleagues [12] as a treatment for alcoholic liver disease has shown how the length of hospital stay might be reduced. Research on the Ontario and Nova Scotia treatment systems has also clearly contributed to local planning and community development initiatives. However, some treatment evaluation research, such as that on Detoxification Centres [13] seems to be ignored by treatment agencies and policy makers [14].

Observers from other countries sometimes see Canada as the place where alcohol research clearly showed how to prevent alcohol problems and are surprised when told that it has not had much apparent impact on government policy. No province, including Ontario, has adopted the

basic premise that the prevalence of alcohol problems varies with *per capita* alcohol consumption and that problems can be controlled by reducing overall availability and increasing real price. Matters of economics, such as the need to maintain employment levels and the need for government revenue, still largely determine alcohol policies in Canada. However, it may be that research on availability of alcohol slowed the liberalization of alcohol laws in Canada and resulted in lower *per capita* alcohol consumption.

There have been several instances where research has affected policy in limited areas. Research on adolescent drinking problems was clearly an important factor in having the drinking age increased in Saskatchewan and Ontario. The current concern with increasing penalties and many prevention programmes for drinking drivers can also be traced to research findings. Research on good alcohol education programmes has also increased their acceptability to schools in Ontario. In general, however, researchers in Canada have been disappointed with the impact of their research on social policies relating to alcohol. There are many reasons for this. Research findings are but one part of the arguments for and against particular alcohol controls. Of course, governments worry about reduced revenues if *per capita* consumption decreases. Also, many jobs in farming, manufacturing, and the hospitality industries depend upon alcohol sales. When unemployment rates are high, policy makers are reluctant to reduce the consumption of alcohol and possibly take jobs away from even more workers.

F The future of alcohol research in Canada

Galsworthy reminds us that 'If you do not think about the future, you cannot have one'. Both thinking about the future of alcohol research in Canada and actually planning it are quite difficult. Certainly, there is no lack of work to be done. There is still no reliable method of preventing alcohol problems and no generally accepted effective treatment. Government alcohol control policies can be improved in all areas of Canada and treatment services should be extended to many isolated areas. The major agencies, such as A.R.F., Health and Welfare Canada, and the Medical Research Council, will likely continue their support of alcohol research in Canada, but major increases in funding or programmes are unlikely in the near future.

Alcohol research centres in many areas of Canada are currently small and underfunded; only Ontario is well served. In general, research budgets are not expanding. The alcoholism treatment and education centres in Quebec were disbanded many years ago. Although the Ministry of Health now organizes services no successor was established to do research. Prince Edward Island and the Northwest Territories have no alcohol research effort. The British Columbia Alcohol and Drug Commission was disbanded in 1984 and, like Quebec, many of the functions were moved into the Ministry of Health. However, this shift did not include the research and no alcohol research exists at present. The A.R.F. had its budget frozen in 1984 after several years of increases below the level of inflation.

On the federal level too, funds for research have been curtailed. The Medical Research Council had its budget reduced by $30 million in 1985, although a campaign is underway to have it restored. Also, the federal government has reduced research funds for environmental projects and has withdrawn funds for several large-scale National Research Council projects in petroleum and industrial research. It appears that money for research on alcohol problems, and many other problems as well, may be more difficult to find in future.

There is still considerable spending on alcohol research in Canada, but researchers might have to do with less in the near future. Alcohol consumption has been essentially stable in Canada for several years and there are recent signs of a decline. The same is true of some associated problems, such as alcohol-related fatalities and liver cirrhosis mortality. Little argument can be made that alcohol research will expand in the near future as the population ages and the proportion of young people, that is those with serious alcohol problems, declines. However, there are several topics requiring research input in Canada, such as, the effects of aging, unemployment, and family breakdown on drinking problems. No doubt there will be more pressures for alcohol research to be well-justified, cost-effective, and clearly of benefit to the population as these new areas are studied.

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ALCOHOLISM

THE GROWING

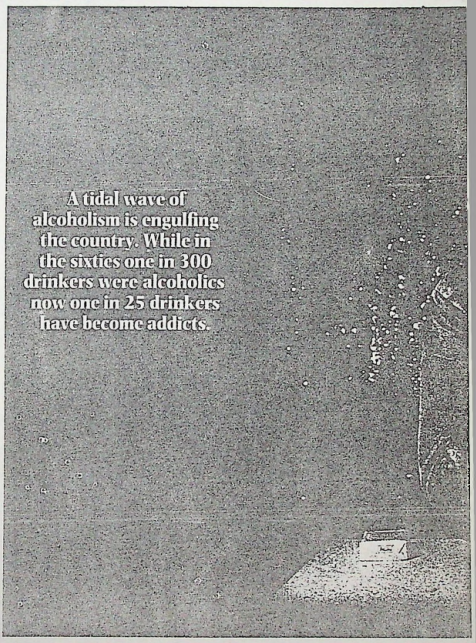
POUR yourself your favourite drink. Sit down, loosen your shoe laces and relax. Take a large swig. Feel the warmth flow into your system. Let the surge of confidence envelope you. Then answer these questions:

Do you prefer to drink alone?
Do you drink daily?
Does drinking spoil your sleep?
Do you drink to bolster your confidence?
Is drinking affecting your work?
Is your family affected by it?
Have you ever had a black-out?

If you have answered yes to any three of these questions then it's bad news. You are either an alcoholic or well on your way to becoming one. Worried? Then push your glass firmly away and listen. You are not the only one.

At exactly 7 p.m. every Saturday in the annexe building of a church on the busy Parliament Street in New Delhi, 20-odd people sit around a hexagonal table lit by candles. Among them are salesmen, businessmen, armed forces officers, government officials, factory workers and even a doctor. As the meeting progresses each one narrates how alcohol has destroyed his life. Choking with emotion, Mathew, a copy-writer in an advertising agency, says: "After a drunken bout I was like an animal. I messed my pants, vomited on the floor and crawled on the floor, unable to pick myself up." A tremendous feeling of empathy builds as the session ends, because each understands the others' sorrows, and many of those present weep openly. The meeting is typical of those held regularly across the country by 100 such Alcoholics Anonymous groups, a rapidly growing organisation of ex-drinkers trying to reform addicts.

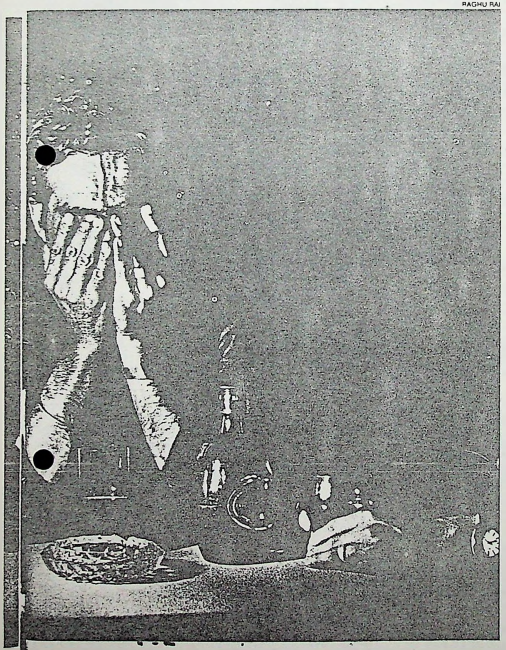
A different kind of scene is being enacted in a white-tiled room at the National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore. Krishnappa, a 38-year-old coffee planter, has his left arm strapped to a 40-volt transformer and is given a glass of whisky. As soon as he takes a sip he is given a mild shock that makes him grimace in pain. This aversion therapy is designed to make Krishnappa's psyche reject alcohol. Krishnappa started drinking when he was 16 and today he does little other than drink. His disgusted wife brought him for treatment last month.



In more homely surroundings inside a sprawling house in Madras, Swaminathan, a 55-year-old former bank manager, struggles with a picture puzzle given to him by psychologists at the T.T. Ranganathan Clinical Research Centre (TTRC) in Madras. The puzzle is designed to test how badly Swaminathan's brain has been damaged by alcohol. Swaminathan never drank when he was young, and was introduced to liquor after he was promoted as regional manager in the bank and forced to attend 'wet' parties. Soon he was hooked. At home he became irritable

MALAISE

RAGHU RAI



and constantly abused his wife, throwing the food she served and breaking crockery. His drinking affected his son so badly that he dropped out of college.

Mathew, Krishnappa and Swaminathan are only the froth on a tidal wave of alcoholism engulfing the country. And there is a cask-full of statistics to prove the point. In the '60s only one in 300 drinkers was considered an alcoholic. Then the World Health Organisation (WHO) reported that by 1980 an estimated three million—or one in 25—of the 80 million Indians who consume alcohol had become severely addicted.

Last year the Indian Council of Medical Research (ICMR) surveyed the problem in four regions—Bangalore, Delhi, Dibrugarh and Ranchi. It found that 20 per cent of urban consumers had become totally dependent on it, while in villages the figure went as high as 30 to 40 per cent. Davinder Mohan, who coordinated the ICMR survey and heads the Department of Psychiatry at the All-India Institute of Medical Sciences (AIIMS) in New Delhi, says: "Alcohol is already taking a heavy toll in the country. It's soon going to be evident even to a blind person."

The toll, in fact, has already become starkly evident:

► In major hospitals alcoholics now form 20 to 30 per cent of the patients in psychiatric wards when five years ago they constituted only 0.2 per cent. In Bombay's KEM Hospital, which treated 309 patients in the past two years, sociologist Hema Shah says: "Initially we got people only from the slum areas. But soon bank officers, managers, factory workers and even government officers started coming for treatment. The middle class too seems to be affected in a big way."

► Last year's ICMR study indicated that half the industrial workers surveyed drink regularly, and companies report that alcoholism has become one of the major causes of absenteeism and falling productivity. At Ashok Leyland in Madras, for instance, managers estimate that atleast 500 of the company's 7,500 workers are problem drinkers. Says Executive Director J. Joseph: "We used to have 10 per cent absenteeism, but now we find it going up to 22 per cent and we think alcoholism has a great deal to do with it."

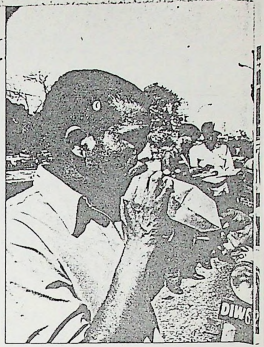
► Alcohol is a major cause of broken marriages. Relationships with wives were seriously disrupted in 64 per cent of the alcoholics. In Bombay, Bagashri Parikh, a marriage counsellor at the city court, says that one out of 10 divorces is because of alcoholic husbands and adds: "Most of the alcoholics beat their wives regularly."

► The country's road research institutes estimate that 25 per cent of road accidents are alcohol-related and that a third of the drivers on the highway are under the influence of alcohol. In Delhi, when the AIIMS did a study of accident victims suffering from head injuries, it found that 20 per cent had consumed alcohol.

Surveying the damage Hira Singh,

director, National Institute of Social Defence (NISD), the Union Welfare Ministry's striking arm against drug abuse, says: "There is no doubt that alcohol is the most widely abused drug in the country." That's certainly true. Alcoholism has traditionally been associated with the tribal areas in the country, where people brew their own liquor. Then the problem spread to the hilly areas and the north-east, and to industrial townships and the coalfields. Now other areas too have got ensnared, hitting people from all walks of life: professionals, businessmen, industrial workers, government servants, armed forces officers and farmers. The statistics reveal that most of the patients (98 per cent) are predominantly male, and usually in the prime of their lives at 25 years and above. A majority are married and have children. In most cases they are the only bread-winners in the family. "It's hitting the most productive section of our society and is creating havoc," says psychiatrist J.V. Devar in Madras.

Researchers are hard put to explain the dramatic rise in alcoholism in a country that has prohibition enshrined as a Directive Principle of the Constitution. But prohibition as a policy has been steadily given up by one state after another, and liquor is now as easily available as tea leaves in most parts of the country. Alcohol consumption, inevitably, has gone up quite dramatically. In 1976, liquor manufacturers used up 169.4 million litres of pure alcohol, enough to manufacture 350 million bottles of rum.



Police test driver for drinking:

CASE STUDIES

Shattered Lives

BHASKER Maben, 49, a machine operator in a Bangalore factory, began drinking heavily 15 years ago after he was superseded at work and his youngest son, Dinakar, died the same year. Maben recalls: "At work I had no peace and at home I was unhappy. Booze was the only way I could forget my sorrows." Instead it brought him more.

Maben became a chronic alcoholic and began sleeping with a bottle under his pillow. He started absenting himself from work regularly. His increments and promotions were stopped.

With liquor burning up all his money, Maben was invariably broke. His favourite clarinet, guitar and camera found their way to the local pawn shop, as did his wife Evelyn's jewellery, including her *mangalsutra*. The worst came when he was evicted from his rented two-bedroom house. Arun, his son, remembers that incident with a shudder and says: "I can never forget how ashamed I was. All our furniture and clothes were lying on the footpath. I just ran to my friend's house and hid."

Someone suggested that he send Rs 50 to St Francis Xavier's tomb in Goa, but that did not help. Nor did money sent to Tirupati Temple, or consulting a *Mantravadi*, who made him wear a charm around his waist and charged him Rs 1,000 for his pains. He was admitted to NIMHANS as many as 18 times for treatment, and the doctors refused to admit him any more.

Frustration and desperation drove him to drink a bottle of varnish last year, which nearly killed him. After that he vowed to give up drinking and over a period of one year, Maben's transformation has been dramatic. He has started doing yoga, which helps him sleep well. He goes to work regularly.

BHAWAN SINGH

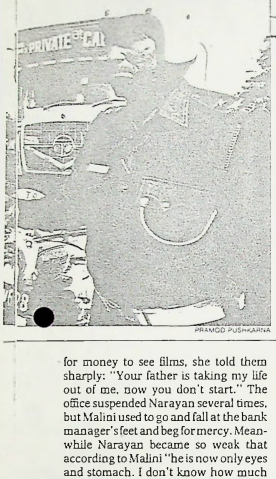
Maben: back from the brink

At home things have improved considerably, and Evelyn now goes shopping with him—something they had not done for years. Every Sunday all of them sing *bhajans* at a nearby spiritual centre. Daughter Chandrika says: "There is so much peace at home now."

■ Narayan, 44, a peon in a bank in Madurai is not so happy. An alcoholic for the past 10 years, Narayan was brought in a delirious state to the TTRC for treatment last month. His office had suspended him for coming drunk to work and warned him that unless he gave up drinking they would dismiss him.

While Narayan is still unable to speak coherently about his addiction, his wife Malini, who faced the brunt of his drunken bouts, has a sorry tale to narrate. She remembers that initially when Narayan drank she thought he was just trying to forget his worries. But soon he was coming home drunk every day, beating her regularly, and always in a foul mood. He threw away the food she made for him, he was nasty with the children and prevented them from studying at night. He did not give her money and pawned all her jewellery.

She was forced to take up work as domestic help to feed the children. And when her children worried her



PRAMOD PUSHKARNA

But despite tough measures by the puritanical Janata government and the Congress(I)'s resolve to hold production at the 1981 level of 207.9 million litres, the consumption of pure alcohol climbed to 331 million litres last year—sufficient to produce 1,134 million bottles of rum!

Indians now have more than 200 brands of whisky, 50 brands of rum, 30 kinds of brandy, 10 brands of gin, 15 of wine, 50 beers and a couple of hundred varieties of country liquor to choose from. "People have become more brand conscious and are willing to pay more for better quality liquor these days," says Ravi Jain, general manager of McDowells, a subsidiary of United Breweries, which is among the big five in the liquor business. McDowells has registered a 10 per cent growth every year.

That Indians are drinking more than ever before is evident by their revolutionary change in attitudes towards drinking. The traditional taboo against liquor is rapidly vanishing. More and more homes serve liquor to guests, 'dry' parties are considered boring, and traditional festivals like Ganesh Chaturthi, Diwali and Holi are used as excuses to go on drinking bouts. Salesmen and businessmen prefer to do business over lunch lined with liquor. Among youngsters, to drink is to be manly. "We are becoming more of an alcoholised society," says psychiatrist Rajat Ray of NIMHANS. And in Lucknow, psychiatrist B.B. Sethi says: "A house not serving liquor is now an exception."

Drinking in bars, once the haunt of only the hardened alcoholic, has gained increasing acceptance. In the outskirts of

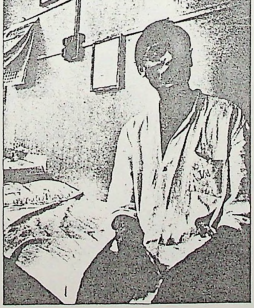
for money to see films, she told them sharply: "Your father is taking my life out of me, now you don't start." The office suspended Narayan several times, but Malini used to go and fall at the bank manager's feet and beg for mercy. Meanwhile Narayan became so weak that according to Malini "he is now only eyes and stomach. I don't know how much longer he would live."

▣ Anand, 33, unlike Narayan who has not yet lost his job, finds his life has been completely shattered and is only now beginning to pick up the pieces. Anand has been an alcoholic for the past 13 years. Last year his wife walked out on him, taking their two children with her. Immediately after that he lost his job as a sales executive in a Delhi travel agency.

Anand, the son of an air force officer, started drinking when in his second year at college in Nagpur. Since his father was in the armed forces, drinking was considered normal. Anand found himself increasingly attending parties where liquor was served and he says: "I used to be the first to start drinking and the last to leave. I had a tremendous capacity, and even if I had five large pegs I would never get drunk." After graduation, his job as a sales executive offered even more opportunities to drink and, as he puts it: "It was an ideal job for an alcoholic, became a 24-hour drinker."

For a while he thought he could handle his liquor and he made sure that his work was not affected. When his wife, a doctor, told him to give up drinking, he said: "I thought she was a bloody nag and I used to get the feeling that the whole world was after my blood." Soon he became irregular at work and extremely irritable at home, hitting his wife once in anger. That's when she left him. He was forced to give up his job because, as he says: "I couldn't handle two careers at one time: my drinking and my job." Last year he touched rock bottom: "I had become like a caged tiger. I didn't know how to escape and all I did was hide myself in the bottle." He finally joined an Alcoholics Anonymous group in Delhi, and now he says: "My obsession is gone. But I'm not going to take up a job till I'm fully recovered."

NAMAS BHOJANI



David: groping for help

▣ David, 32, a packer in a Bombay firm, has had equally traumatic experiences with alcohol. Living in a dirty slum in Ghatkopar, David, who looks older than his age, is slowly recovering from the damaging effects of alcohol. His speech is still slurred and he gropes for words. His hands tremble uncontrollably. Doctors attending on him say his brain has suffered some damage and it may be a year before he is able to recover.

David, who is married and has two children, remembers that he started drinking when he was around 15 years old: "At that time we drank because after a hard day's work we had nothing to do and we wanted to forget the strain of living." Initially he drank only on week-ends, but: "After some time all I did was to drink, drink and sleep."

He refused to go to work, and last year was absent for 140 days. His office sent him a charge-sheet, which he ignored. His entire pay packet of Rs 1,000 went towards liquor: "I used to be so desperate that I even sold my shoes and shirt." His wife left him twice to go and live with her father.

David was admitted last month to the KEM Hospital for severe withdrawal symptoms of alcohol like hallucinations and shakes. Says David: "Alcohol has ruined my life."

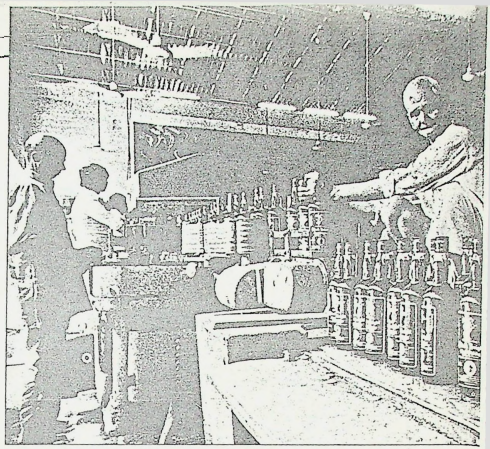
—RAJ CHENGAPPA

Patna, for instance, roadside *bhattis*, the poor man's bars, have become popular drinking places. One of them on the by-pass road to Patna is a huge, sprawling dimly-lit shack reeking with the stench of liquor, rotten fish and frying eggs. Around 250 people, among them teenagers, sit around in groups chatting and shouting. They come from all social groups—rickshaw-pullers, factory workers, professionals and even police constables in civilian dress. Babu Lalrai, 35, a tailor, chirps up: "Yes, I drink daily and go to the Ganga for a holy dip daily."

THE WAVE of alcoholism is by no means confined to the cities. While the sale of country liquor in most states has risen slowly, this masks the fact that people have increasingly moved to cheaper, illicit liquor because even country liquor costs as much as Rs 8 for a bottle. In Bangalore, arrack king H.R. Basavaraj estimates that "illicit liquor sales equal that of country liquor". The repetitive liquor tragedies testify to this. In Bangalore more than 300 people died of liquor poisoning in 1981. The next year in the island of Vypeen in Kerala, 72 people were killed. On an average more than 200 people are killed in the country from liquor poisoning every year.

What worries many psychiatrists is the widespread acceptance of beer and the current boom in beer sales. In Maharashtra, beer sales have jumped from 14 million bottles to 24 million bottles in the last five years. And in West Bengal, more beer is consumed than whisky, rum and brandy put together; last year's beer sales totalled 12 million bottles. But if people regard beer as some kind of milk product, the harsh truth is that one bottle of beer has about the same alcohol content as a peg of whisky, and psychiatrists say that beer drinking not only opens the floodgates for more potent liquor but is addictive and in the long run can do as much damage as whisky or rum.

The spiurge in drinking is reflected in the high per capita consumption of liquor. While the annual per capita consumption of alcohol by adults in India hovers around one litre of pure alcohol (equivalent to two-and-a-half bottles of rum), a little lower than Australia's 1.5 and a lot less than the US's 4.1, this figure is misleading. Unlike Australia and the US, women in India hardly drink. So if only the male population's consumption is calculated, the figure jumps to around two litres of pure alcohol. And if only the drinking male population is taken into account, the figure is as high as 4.1, or 10 bottles of rum per head annually: high enough for the alarm bells to be ringing. Even this excludes the millions of litres of illicit liquor that goes down parched gullets. And in states like Punjab, the per capita consumption is 6.5 litres of pure alcohol, or 16 bottles of rum.



On the production line: 350 brands to choose from

All drinkers are not drunkards. So when does a normal drinker turn alcoholic? The popular concept of the drunk is one who falls into gutters or staggers around on streets mouthing obscenities. But that, say psychiatrists, is only the last stages of alcoholism. A social drinker becomes a problem one when, according to Shanthi

INTOXICATION

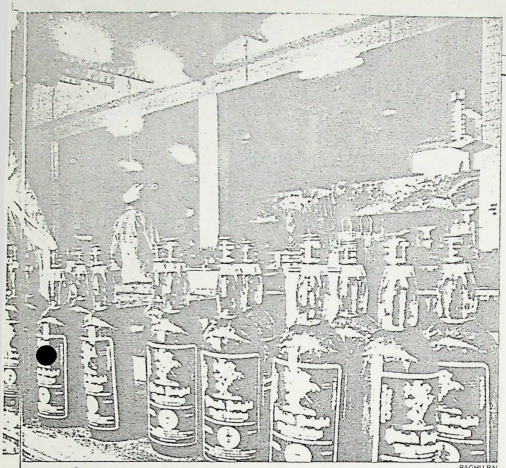
The Effects

ALCOHOL is formed by fermenting sugar with yeast spores. Ethyl alcohol, or pure alcohol, is the basic raw material for the various liquors available. It has no nutritional value but produces feelings of sedation, euphoria, intoxication and finally unconsciousness. Whisky, rum and gin have around 40 per cent pure alcohol, wines 8 to 15 per cent and beer 4 to 8 per cent alcohol content.

Depending on what the person has eaten, 20 per cent of the alcohol that is drunk is absorbed immediately by the blood stream through the walls of the stomach and the rest from the small intestine. Eating peanuts or cheese helps line the stomach walls and slows down the absorption of alcohol.

Until the liver oxidises all the alcohol it keeps coursing through the body, including the brain. The effects of alcohol depend on the blood alcohol level (*see illustration*). It is in the brain that the effects of intoxication are produced. Research has shown that alcohol primarily depresses the central nervous system resulting in thought becoming jumbled and disorganised and the faculties of discrimination, memory, concentration being dulled and then lost.

The early warning signs of alcoholism include an increasing tolerance for alcohol, the quick gulping of pegs, blackouts or temporary amnesia and an intense preoccupation with drinking. The crucial stage comes when the drinker loses complete control over the amount of alcohol he consumes and when to have a drink. He has ready excuses for his drinking. He exhibits grandiose behaviour, spends money lavishly and starts showing signs of aggres-



RAGHU RAI

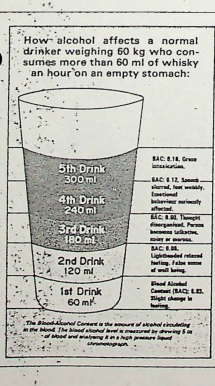
Ranganathan of TTRC: "His drinking starts affecting on a continuous basis his health, his job efficiency and his relations with his family and friends." The WHO estimates that one out of 10 drinkers turns alcoholic.

Most drinkers do not realise when they have crossed the thin red line between social drinking and alcoholism. In Madras, Udayakumar, 37, a former packing manufacturer, thought he had his drinking well under control till one day he realised that "a quarter of a bottle of rum was a mosquito bite". In Bangalore, a salesman remembers that while initially he could hardly hold a peg of whisky he suddenly found himself going on long drinking bouts. Says he: "Every cell in my body craved for alcohol and when I took a drink it was like setting off a nuclear reaction. I didn't stop drinking till I became unconscious." And in Bombay, a factory supervisor initially started drinking during week-ends with friends. Then, as he says, "after a while I drank once in three days, then every alternate day, then every day and then every hour".

Researchers now agree that alcoholism is a disease, because alcoholics exhibit predictable symptoms and suffer the same physical and mental trauma: severe shakes, damage to vital organs like the heart and liver, and psychological breakdown. Society, which always dismissed alcoholics as weak-willed or immoral people, is only now reluctantly beginning to accept this home truth. In Bangalore, Malavika, a 30-year-old management consultant married to a lawyer, confesses: "I thought all drunkards were bums till my husband became an alcoholic. Initially I was bewildered and thought he drank because he was not happy with me. But I soon realised that although he desperately wanted to give up alcohol he couldn't. It was as though he had caught a disease."

ONCE the disease catches on it moves with terrifying rapidity, reducing its victims to physical, mental and moral wrecks. Try as they will, they find it impossible to kick the habit even though they know that it is steadily destroying their lives. A Bombay naval officer remembers with tears how he tied his favourite Labrador and whipped it mercilessly in a drunken fit. The next morning the dog seemed to forget the beating and wagged its tail, but he was so insane that he whipped it again. Mathew, the Delhi copy-writer, remembers how he threw up a Rs 5,000 job in a leading advertising agency when he quarrelled with his employer over a trivial matter.

Probably the most damaging effect of this wave of alcoholism, according to a doctor, is that "not only the drinker but everyone in the family suffers. Alcoholism is a family disease." As the alcoholic becomes more and more obnoxious, the wife and children get seriously affected. Malavika, the lawyer's wife, recalls the traumatic experience when guests dropped in one morning and found her husband lying on the floor dead drunk. The naval commander's wife once tried to jump out of a fourth floor window after he slapped her in a drunken rage. Children too are badly affected. In a typical case in Bangalore, Arun, 17, whose father has been a chronic alcoholic, dropped out of school and took to drugs and alcohol. In



sion. He begins to stock up alcohol and if threatened hides his stock. He drinks to prevent withdrawal symptoms.

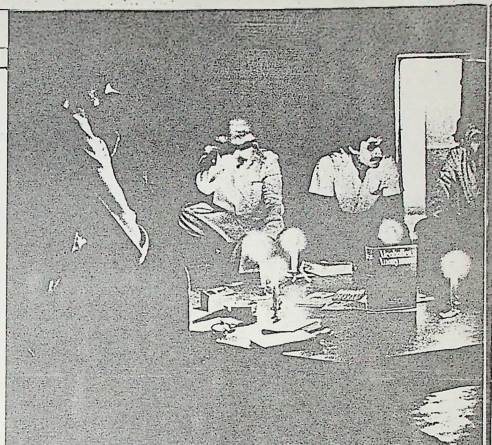
The chronic stage is reached when the drinker goes on binges for days together, followed by abstinence for a short period. He develops suspicious ideas, fears and doubts about both himself and his family members. He loses his tolerance for alcohol and gets highly intoxicated on small quantities. If he does not take alcohol his body develops severe withdrawal symptoms like hallucinations, tremors and shakes, profuse sweating and irritableness.

In the chronic stage, apart from the severe breakdown of relationships with his family, friends and colleagues at work, the alcoholic also suffers from acute physical damage. Prolonged drinking can lead to cirrhosis of the liver, jaundice, stomach ulcers, heart and brain damage. Little wonder then that alcohol is called the demon drink.

fact studies abroad indicate that alcoholism could also be a familial disease, with children of alcoholics being more prone to becoming alcoholics themselves.

If more families have not broken up, it is because most Indian wives fear the stigma attached to divorce. Last fortnight Malathi, 38, the wife of a former supervisor in a printing press and mother of five children, came to social workers at the Maharashtra State Women's Council for help. Her husband had beaten her so badly that her cheek and shoulder were blue-black. She said her husband had lost his job because of his drinking, and when she wanted to take up a job her husband refused to allow her and suspected her fidelity. Her eldest son had become a vagrant and a petty thief. But when the council suggested she divorce her husband, she flatly refused. Says the council's social worker Anila Merchant: "For most sufferers marriage provides some form of security."

A just-published study of workers at the Madras Port Trust, done by sociologist Saraswathi Sankaran, shows how damaging alcohol can be. Of the 162 workers surveyed, more than half of them drank heavily, and a third were addicts.



DE-ADDICTION

Creditable Successes

WHEN Shanthi Ranganathan, a Madras housewife, decided to set up an exclusive centre for the treatment and rehabilitation of alcoholics five years ago, many people dismissed her efforts as the desperate actions of a grief-stricken wife. Her husband, an industrialist, had just died from alcoholism and Shanthi recalls: "People thought I was crazy. They could not accept that alcoholism could be cured, and doctors warned me that it was a disease of only relapses and remissions." But Shanthi was determined. She did a course in the US on the treatment of alcoholics and started a centre in her sprawling home on the quiet Santhome High Road in Madras.

Today the T.T. Ranganathan Clinical Research Centre (TTRC) has grown into an institute of national repute and is the only one of its kind for treating alcoholics. Given the excellent cure rate of almost 60 per cent, the annual inflow of patients has gone up from 62 in 1981 to 320 last year.

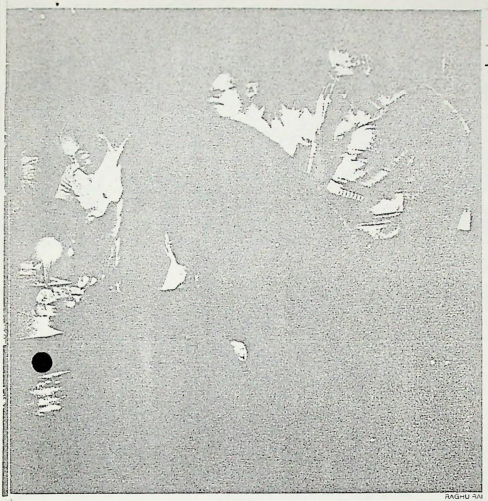
Before the centre was established, there were hardly any treatment facilities for the country's three million alcoholics, most of whom were admitted to the general wards of hospitals and treated for symptoms like jaundice or hallucinations. The centre's success lies in the fact that it offers treatment for the basic problem of alcoholism. It first admits patients to a nursing home for withdrawal symptoms like hallucinations, vomiting and severe shakes.

Once the "detoxification programme" is over, patients undergo intensive individual and group therapy sessions. Surprisingly, most patients blame their problems on others. A bank manager undergoing treatment blamed his son who dropped out of college as the reason for his heavy drinking.

The group therapy sessions help patients to confront their problems head on. In these sessions, recovering alcoholics narrate their problems, providing hope and making it easy for new patients to identify with them. Says psychologist Christina Chakravarthy: "Group therapy helps in assuaging their guilt and releases their bottled-up feelings." The patient is able to admit both to himself and others that he is an alcoholic.

But to ensure that he does not yield to fresh temptation, the patient is put on a drug called Antabuse which causes a violent reaction if he consumes alcohol while on the course. The reactions could even result in death unless an antidote is administered within 24 hours.

However, getting an alcoholic to admit that he is one and keeping him off liquor for a month is, as psychiatrist J.V.Devar puts it, "only half the battle won". The more difficult part is rehabilitating him so that he can start living a normal life. Most alcoholics have snapped ties with their family and friends and have probably lost their jobs too. One of the major hurdles psychiatrists face is in getting the family to be more understanding towards recovering alcoholics. This is easier said than done as the wife of a salesman-turned-alcoholic narrates. Her husband found that he hardly had any role to play at home because she did all the work and the children held back



RAGHU RAI

Alcoholics Anonymous meeting

Sankaran compared the work performance of the drinkers and non-drinkers and was stunned to find that the heavy drinkers were absent every sixth working day. As a result, the non-drinkers took home 50 per cent more money than the drinkers, spent 8 per cent more on food, 30 per cent more on clothing, 168 per cent more on health care and a whopping 300 per cent more on children's education. Says Sankaran: "Whether it is poverty, malnutrition, marital disharmony or delinquent children, alcohol seems to have been the major cause of all their miseries."

While alcoholism and its related problems increase alarmingly, progress on the treatment of sufferers has been tardy. Most alcoholics find that doctors treat the problem as a moral issue. Said one alcoholic: "My whole world had crumbled, and here was this doctor telling me I must develop the will power to stop drinking." Only now are hospitals recognising alcoholism as a major problem and setting up specialised clinics for treatment *(see box)*. As a consequence, there are facilities for treating hardly 3,000 of the estimated three million alcoholics in the country.

from him. To overcome these problems, the centre holds regular family counselling sessions where the families are taught how to cope with recovering alcoholics.

While centres like TTRC may be difficult to replicate in the country, elsewhere, hospitals which have set up de-addiction clinics (and there are very few) have also been reporting encouraging results. One of them is the de-addiction centre run by the KEM Hospital in Bombay. Using a judicious combination of group therapy, individual counselling and an Antabuse course, the clinic reports that in the past two years it has been able to achieve a cure rate of 46 per cent. And in Bangalore, NIMHANS psychologists using aversion have reported an encouraging 45 per cent cure rate.Says psychologist V. Kumaraiah: "We found that married people responded to treatment better than the rest, and the recovery rate was the highest among the older groups.

But probably one of the most successful ways of treating addicts is the Alcoholics Anonymous (AA) programme which is fast gaining popularity in the country. AA is a non-profit world-wide organisation of recovered alcoholics trying to help others in the same plight. There are already 100 such groups in the country, with Bombay alone boasting 55. Other cities with AA groups are Bangalore, Goa, Madras, Secunderabad, New Delhi, Cochin, Mangalore, Nagpur, Pune and Mysore.

Aversion therapy: effective

DEEPAK PAWAR

AA members find that the group sessions help alcoholics to come to terms with their problems. In fact the first step for all AA members is to admit that "we were powerless over alcohol, that our lives had become unmanageable". The sessions also help other recovering alcoholics by constantly reminding them of their earlier plight. Said an AA member: "By trying to help others we are actually motivating ourselves to keep away from liquor." For the families of alcoholics, the AA has a separate group called Al-Anon where members hold similar group therapy sessions. Rather than impose a set period of abstinence, the AA's golden rule for its members is: Get up every morning and tell yourself that you will remain sober for the next 24 hours. It seems to work.

—RAJ CHENGAPPA

But even hospitals that have clinics are faced with problems like the lack of professionally trained staff.

The problem is compounded by the fact that most alcoholics do not get themselves treated till at a very late stage, because most people do not like to admit they have an alcoholic in their family and try to conceal the fact as long as possible. A factory supervisor in Bombay, who had been an alcoholic for five years, first tried to give up drinking himself. When that failed his wife sent him to the local *mantravadi* to exorcise the spirit that seemed to have possessed him. It was only when he was admitted to the KEM Hospital with an attack of jaundice that he learnt the hospital had a de-addiction programme. In fact, most of the patients usually go to hospital when they have serious health problems such as cirrhosis of the liver or fits of delirium.

If the psychiatrists find it difficult to cope with the problem, the Government seems equally helpless. The state governments, which control alcohol supply and distribution, indirectly encourage people to drink more because revenue from excise forms a major part of the income of most states. In Karnataka liquor is the second biggest money-spinner for the state, netting Rs 50 crore last year, while in Tamil Nadu it accounts for a good 8 per cent of the revenue. Total liquor sales in the country would amount to something like Rs 3,000 crore, and liquor companies form a powerful lobby group.

Ironically, although the Constitution has made prohibition a Directive Principle, the states have made only feeble attempts to make prohibition effective policy. Most governments in fact have given up prohibition altogether. Tamil Nadu and Maharashtra, which had imposed prohibition at different times, ran into trouble and were forced to relax their rules. Tamil Nadu needed the excise revenue to run its midday meal scheme. Maharashtra, which strangely enough has placed prohibition and excise under the same department, is quite happy counting the take from liquor sales while running a prohibition programme to educate people on the evils of drinking. Bihar was forced to lift prohibition after a large number of people died in of illicit liquor in the Dhanbad coalfields. Right now Gujarat is the only state where prohibition is enforced, but liquor is still easily available and the policy is commonly seen as a farce.

WISER from these experiences, many states have set up temperance boards in the fond hope that if they cannot prevent people from drinking they can convince them to drink moderately. But most of these boards do little more than release advertisements in newspapers showing a bottle marked with the familiar skull and crossbones.

With governments unable to curb the menace, people have taken matters into their own hands. In distant Imphal in Manipur, women were so disgusted with their menfolk's excessive drinking that they formed themselves into an association against drunkards and kept vigil at nights in front of liquor shops and bars. Any drunkard they catch is publicly humiliated by making him walk with a string of bottles tied around his neck, and then handed over to the police. In the hills of Uttar Pradesh and tribal areas of Bihar similar people's movements have met with notable success in an area where drunkenness has become a major social problem.

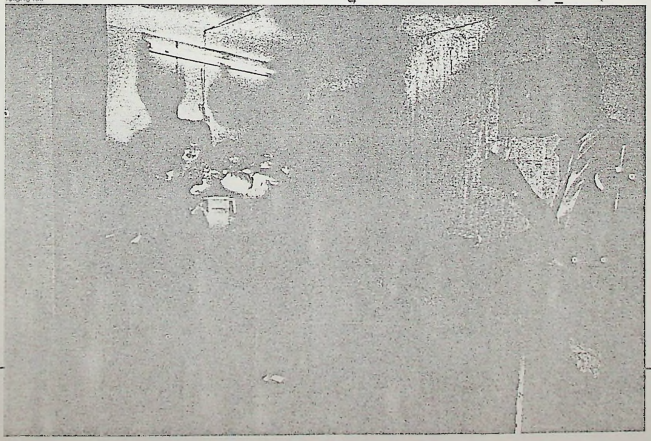
Meanwhile, to formulate fresh strategies against drinking and drug abuse, the Welfare Ministry two years ago set up a working group chaired by its joint secretary. This is likely to stress education programmes on the evils of drinking. As Rajendra Kumari Bajpai, minister of state for welfare says: "A stroke of the pen, like imposing a total ban, is not going to help. If we have to bring alcoholism under control we need to use persuasion, create awareness and show a strong determination to implement our programmes."

But with both the Central and state governments failing to make a serious dent in the two vital factors that the WHO says leads to an increase in alcoholism—easy availability and increasing demand for alcohol—the battle of the bottle may be well and truly lost.

—RAJ CHENGAPPA with bureau reports

RAGHU RAI

Youngsters drinking: vulnerable age



..and a word rom a brewer who cares

$ *'boutique breweries' go for quality, not quantity*

By Nicolene Hengen
in Boston, USA

"AMERICANS ARE beginning to take beer seriously," says a content James Coch, brewer of the new, upscale Samuel Adams Boston Lager. He should know, his Boston Beer Company is one of a wing number of small, regional brewes that are springing up across the ntry to meet the demands of what perts describe as an increasingly re-ed consumer palate. Americans be-ne educated consumers of wine from all vineyards about 10 years ago; now ericans are refining their ability to dis-minate among fine beers.

The typical Samuel Adams drinker, ording to Coch, is someone who "has little more sophisticated taste and derstands enough about beer to know t freshness matters." As he sees it, ch is never going to be fighting big ewers for a piece of the market. "I'm t going to reform any Coors drinkers," jokes [illegible]erring to a giant brewery. "A ors dr[illegible]er has to be hospitalized after ey drink my beer."

American beers, he says, have become adily less flavorful, a trend he attributes the drastic decline in the number of erican breweries during the last 40 ars (from 800 in 1948 to 80 in 1986). ralling production costs have forced st of the smaller, specialized breweries t of business. "Twenty years ago," he ags, "people wouldn't have touched ors."

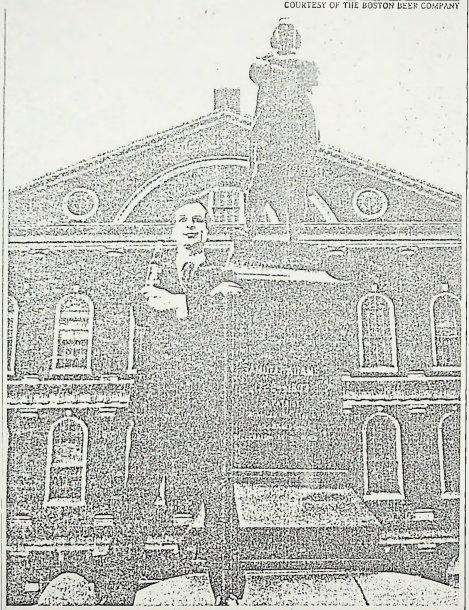
Under such pressure, what is a beer noisseur to do? There are always the ports but, for Coch, "some of those tle like they were shipped over in a sketball." He argues that imported beers tend to be stale and their recipes often include undesirable stabilizers and preservatives. While the Reinheitsgebot, the West German beer purity law, for example, requires that no German beer is lated for domestic consumption contain more than water, yeast, malt and hops, exports are under no comparable restrictions.

For Coch, the American market is divided between consumers who drink more flavorful but stale imports and those who opt for fresher, domestic brews. "That's why I started Samuel Adams, to give beer drinkers flavorful beer that's fresh," he says.

His efforts have not gone unnoticed. Samuel Adams was the first American beer to be selected for two consecutive years as the best American beer by the 5,000 brewers, distributors and beer lovers who gather at the Great American Beer Festival held annually in Denver, Colorado. Everyone is given a single vote and two evenings to sample as many as 100 different American beers.

"Samuel Adams proved that there was a market for high quality American beer," Coch says proudly, and a lot of entrepreneurs agree—as evidenced by the blossoming of small breweries nationwide. Around two dozen small breweries have been launched in just the last year. The "boom phase" should last for another two or three years before competition narrows the field.

Currently, the Boston Beer Company, with only 12 employees, brews 25,000



COURTESY OF THE BOSTON BEER COMPANY

A brewer and a patriot who is trying to throw the foreigners out.

ilene Hengen is a Boston-based writer and
or. This is her first piece for WorldPaper.

cases of Samuel Adams each month in Pittsburgh, Pennsylvania. "I make in a week what Anheuser-Busch makes in 20 seconds," he adds, referring to another market giant. But Coch and his investors are in the process of renovating the former Heffenreffer brewery in Boston—a project that will eventually cost US$5 million. "I wanted the brewery in Boston," he says. It has not been decided yet whether the Pittsburgh brewery will remain open but, he says, "it would be nice to have a backup in case something blew up."

Samuel Adams is snapped up by about 250 restaurants, clubs and liquor stores in the Boston area, where a bottle sells for $2 to $4. Besides the American Northeast, Samuel Adams beer is also available in Australia, through yacht skipper, hotelier and brewer Alan Bond. James Coch reflects on the inroads into a new market by commenting that "Basically we can decide how much, and how fast, we grow."

Right now, he is excited about the renovation of the old brewery, scheduled to be completed in October. "There's some romance in being in an old brewery. There are good vibes." And good vibes are not something to be ignored when felt by a man who represents the fifth generation of his German immigrant family to work as a brewer.

Coch was the first in his family to go to college. After three years as a mountaineering instructor and seven as a consultant he decided to make beer. His father was incredulous, but Coch argues that he "grew up on beer and making beer is what I wanted to do." The recipe he uses is his great-grandfather's.

As a brewer, Coch now spends one or two days each week in Pittsburgh. The actual brewing process takes a single day but the fermentation and aging process takes six weeks. "It's like baking, once it's in the oven you don't have to think about it." Because it has no perservatives, once the process is complete, it has to be consumed before it has time to get stale. "I want the stuff in people's stomachs within two or three months," Coch says.

According to him, "Americans have an inferiority complex about beer"—something he is out to change. Coch's beer is aptly named for one of America's favorite firebrands, a man who, as Coch explains, "was a brewer and a patriot who threw the foreigners out. What could be more fitting?"

Small type and flint glass

India beats the ban on liquor ads

A WHITE ROLLS ROYCE with a chauffeur stands waiting in front of a stately white mansion. The advertisement's copy reads "When the immaculate taste of aristocracy spills beyond stately mansions and white Rolls Royces, it finds colour in Aristocrat—the spirit of excellence." Miniscule type at the bottom states "For your requirement of coloured and flint glass, write to Jagatjit Industries Limited."

This odd ad is a liquor company's attempt to sell liquor despite a national prohibition against the advertising, but not the sale, of distilled alcohol. Aristocrat is a popular Indian whiskey produced by Jagatjit Industries. The company's sales of colored and flint glasses could never justify the cost of the ad.

Liquor is a booming business in Rajiv Gandhi's India—the prohibition of it is a hangover from Mahatma Gandhi's India. While most of what the Mahatma stood for has fallen by the wayside, he remains the father of the nation, and lip service is still paid to his values.

Some states still observe the prohibition, but even there you can find your way around the ban by getting an alcohol permit (for medical reasons), by being a non-resident Indian or by being approved because you entertain foreigners for business reasons.

Even though one can sell liquor, one cannot openly solicit people to use it; thus there are advertisements like the one showing three elegant young people holding liquor glasses, photographed through the amber liquor of a whiskey bottle. One of the three is pouring McDowell's soda into his glass. The legend reads, "Unmistakably number one—McDowell's." Needless to say India's premier liquor manufacturer is not advertising its like-named soda water—a product made explicitly to beat the advertising ban.

"Liquor manufacturers," says Kusum Dudlani, an advertising consultant with considerable experience in liquor campaigns, "invariably go in for thinly disguised, indirect advertising. There is always a saving device to beat the government or excise the ban on advertising. But the ads always create an awareness of the product's name, giving it a certain image."

In their constant push against the lawful limits, "when the manufacturers see no government action forthcoming, they get more and more blatant," explains Dudlani.

By Arun Chacko, WorldPaper's associate editor in South Asia.

coholism has begun ravage African nations

mption has increased by as much as 900 percent

By Macharia Gaitho
in Nairobi, Kenya

OL IS BIG business in Kenya. ican Breweries Ltd., the biggest in the country, produced 22.2 ns of beer, more than one ton for in, woman and child in the coun- recorded profits of 394 million hillings (US$24 million) in 1986. an half of the company's profits the government as taxes. East Breweries, which holds Kenya's nopoly, is now making efforts to ets in neighboring Uganda and , as well as in the United States United Kingdom.

has a major alcohol problem and beer presents just the tip of the No nationwide survey has been ut but in one study done in the rict it was found that up to 27 per- e men, 24 percent of the women rcent of secondary school stu-

Gaitho is a reporter with the *Weekly* Nairobi, Kenya.

dents were alcoholics. The study found that alcohol is widely available in the community and that most households brew or distill their own alcohol and sell their surplus.

Drinking is a primary social activity for all classes. Kenyan President Daniel Arap Moi early this decade decried the drinking habits of Kenyans and ordered the closure of all beer halls in the country—to no avail. In his 1987 New Year's message, the president returned to the theme once again when he ordered the closing of all unlicensed drinking establishments in the country, but observers were skeptical that it would work. The only beneficiaries would be the proprietors of licensed bars who would have more patrons.

Studies show that alcoholism is not a problem unique to Kenya; it is a feature of the developing world. In a 1985 World Health Organization (WHO) study it was found that beer consumption in a number of African countries had risen by staggering amounts between 1961 and 1981. The Republic of the Congo recorded an increase of 893 percent, followed by South Africa with 723 percent and the Ivory Coast with 506 percent. Kenya recorded an increase of 247 percent. The same study found that during the same period, world production had risen by 124 percent, with the rise in Asia and Africa recorded at 500 percent and 400 percent respectively.

In 1982, Kenya hosted the fourth world congress of the International Commission for the Prevention of Alcoholism and Drug Dependency (ICPA), yet it took three years for the Kenya National Committee to be founded in 1985.

The committee organized seminars and workshops to highlight alcohol-related problems and noted the need to introduce alternative social pastimes. Other measures such as curbing the availablity of alcohol, providing adequate facilities for the treatment of alcoholism and imposing stiff penalties for alcohol-related crimes were also suggested. WHO recommends national regulations of alcohol production, control of alcohol imports and the reduction of alcohol sales by limiting sales outlets and banning alcohol advertising.

While many feel that such strong measures are necessary, they have yet to reckon with the corporate might of the alcohol industry. East African Breweries' sales contribute substantially to government coffers and employs over 4,000 Kenyans directly and another 40,000 indirectly.♦

PHOTO BY ALEX WEBB/MAGNUM PHOTOS, INC.

A major alcohol problem, where the legal beverages represent just the tip of the iceberg.

ALCOHOL AND TOBACCO

L. America is going up in smoke

Cigarettes are the region's main public health problem

By Myriam Bautista
in Bogotá, Colombia

LATIN AMERICA HAS PROBLEMS. As if the well-known financial and social difficulties in the region were not enough, it now seems that the health of Latin Americans is going up in smoke.

This is the unavoidable conclusion to be drawn from the statistics on tobacco consumption in the region. In contrast to what is happening in developed nations, where campaigns and legislation against smoking have managed to reduce the numbers of smokers, what happens here is precisely the opposite.

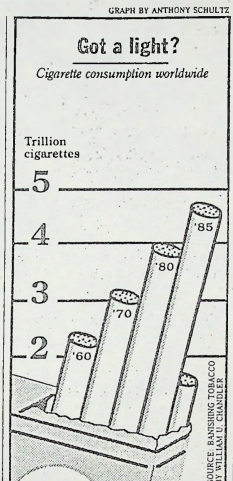
According to the Latin American Committee for the Control of Tobacco Use, an organization of twelve countries, smoking-related diseases are the number one public health problem in the region. In Colombia, for example, 30 percent of all cancer victims are smokers.

But the pernicious effects of tobacco are not limited to just one area: South American governments spend twenty times more in treating patients affected by cigarette-related diseases than they collect in taxes on tobacco products. Absenteeism from work, illnesses and premature deaths, as well as subsidies and pensions for disabilities, are also related to tobacco use.

The figures are alarming. In Colombia, according to a survey conducted by the Ministry of Health, there are 7.650 million smokers in a population of 27 million, and of these over 1.2 million smoke more than one pack a day. In other countries the situation is very similar.

In 1984, the annual consumption of cigarettes in Argentina was 1,220 cigarettes per inhabitant. In Brazil, it is estimated that there are 27 million smokers, consuming annually 1,191 cigarettes. In Chile, the figure is 1,014 cigarettes, and in Uruguay 1,146. The latter is the only country where the situation looks a little less grim. From 1971 to 1985, the per capita consumption went up *only* 33 percent, while in Brazil, in the 1970s, it went up 80 percent.

GRAPH BY ANTHONY SCHULTZ



Experts say that the main reason why Latin Americans smoke so much is the marketing strategy used by the tobacco multinationals.

Juan Manuel Zea, director of the Cancerology Institute of Colombia, points out that multinational corporations such as Phillip Morris have brought their hard-sell advertising to these new markets after legal restrictions and anti-smoking campaigns started to take place in developed nations. Advertising in Latin America is aimed at 12-18 year olds, the age when it is determined if an individual is or is not going to become a smoker.

Even though there are no official statistics, it is believed that the large, multinational tobacco corporations doing business in Latin America have made enormous profits. It is estimated that in 1986, the proceeds of cigarette smuggling in Colombia reached $151 million. The Colombian Tobacco Company, which sells domestically produced cigarettes, topped its 1985 profits by 166 percent in the first half of 1986.

In an attempt to address the smoking problem, some countries in the region have launched timid prevention campaigns. In 1979, a "quit smoking" course was taught in Argentina. The follow-up found that 20 percent of those who took it did not quit smoking, 40 percent quit for good, and 40 percent quit for six months.

In Colombia, the National Council for Cigarettes and Health recently published a pamphlet called *The Pleasure of Not Smoking*. It was addressed to elementary and high school teachers nationwide.

"Most tobacco advertising," says the pamphlet, "sends messages that have no relation whatsoever with the product they want to sell, and often expresses ideas opposite to the intrinsic qualities of the product. For example, many cigarette ads talk about fresh air, nature and freedom. This directly contradicts reality because the cigarettes we burn produce dirt and pollute the air. Furthermore, instead of freeing the individual, they enslave him to this addiction."

Undoubtedly, advertising is one of the most influential factors affecting the increase of cigarette smokers. In this regard, Latin American countries are starting to pass very clear laws, even though they are not strictly enforced.

In Colombia, the government of President Belisario Betancur ruled that no cigarette commercials could be aired on television until after 9:30 p.m. and for up to 30 seconds in length, 20 seconds of which would have to be used to inform viewers about the ill effects of smoking. In Chile, 69 percent of all tobacco advertising is on television, 20 percent in magazines, and 11 percent in newspapers.

Restrictive measures are not limited to advertising. Even though it is not fully complied with, in most Latin American countries smoking is forbidden in public places such as buses, theaters, schools, hospitals and public offices. But the magnitude of the challenge is such that experts insist that corrective steps must be taken before it is too late.

Thus, to the economic recession, the unpayable foreign debt, the wild growth of the informal economy and the deterioration of the standard of living on the continent, we must add the enormous public health problem created by widespread tobacco use.

Myriam Bautista is an investigative reporter with the Bogota-based weekly *Semana*.

CHAPTER

15

ALCOHOLISM IN INDIA

BY

DR. GURMEET SINGH

HISTORY

India has always been described as an abstinent culture, i.e. a society where the majority do not drink and have clearly negative attitudes towards the use of alcohol. In such a situation one would expect the overall consumption level to be generally low. There is little information on the habits and culture of the earliest inhabitants of this subcontinent, who were probably negroid in type. They were followed by the Dravidians, whose staple diet was meat and fish with rice, and who consumed two intoxicating drinks 'Ira' and 'Masura'. In the Rig Veda, two types of beverages are described:
(a) 'Soma' juice was most important, but there is controversy as to whether this juice was used as such, or after fermentation, since the word 'Soma' has been used occasionally by subsequent authors as a general term for all intoxicating drinks. Evidence from Vedic hymns indicates that Soma was identified as a mushroom and a cannabis like substance (Sethi, 1978). However, it is generally agreed that 'Soma' was a juice extracted from a plant brought from the mountains where it grew wild, particularly 'Maujavanta' in the Himalayas. It was often drunk as such, or with clarified butter, milk, or curds to improve its taste. It was believed to inspire confidence, courage, faith and bestow powers of eloquence and immortality.
(b) The other beverage was 'Sura' an intoxicating drink prepared from fermented barley after distillation. Its popularity was evident from a verse in the Atharvaveda, where it was mentioned as a reward for performance of sacrifices. The praise of Sura in the Aitareya Brahmana, and the placing of a Sura vessel in the hands of a king suggested that the Kshatriyas were in the habit of drinking Sura.

CHAPTER XV

During the next five centuries i.e. the Sutra period (800 to 300 B.C.), we have reference to alcohol in (a) the Brahmanical literature viz. the Sutras, and (b) the Buddhist and Jain works, which represented a non-Brahmanical, if not exactly a Kshatriya tradition. Drinking was apparently common in the days of Panini as he mentioned words meaning liquor, distillery, must, and sediment. A number of socially accepted occasions were mentioned in the Sutras, when liquor was served to guests e.g. when entering a new house, on the arrival of the bride at the groom's place, and to the women dancers at the time of marriage. Besides Sura, a number of other drinks were introduced during this period, e.g. 'Kilala'—a spiced drink prepared from gud (brown sugar) which became very popular. Wines were also imported, chiefly from Kapisi (in Afghanistan) and commonly known as 'Kapisayani'. The Jatakas which represented the Buddhist and Jain traditions also affirmed that during this period liquor was manufactured and consumed on a large scale. Taverns were present in most cities, and were distinguished from other shops by flags. At the time of festivals and feasts drinking was permitted and friends invited.

Buddha and Lord Mahavira did not allow their followers, especially monks, to indulge in wine. In fact the Jain Canon does not permit a monk to even reside at a place where jars of wine are stored, but other believers were allowed to drink on certain special occasions, or in the case of illness. The evil effects of drinking were mentioned at several places, and the example is given of the princes of Baravi, who were ruined because of their addiction to 'Kadambari' wine. For the Maurya and Sanga period we have information from the Arthshastra of Kautilya, the edicts of Ashoka, the writings of Patanjali, and accounts of great historians, about alcohol during those periods. From the writings of Kautilya it is apparent that drinking was fairly common and well organised. There was an official superintendent of liquors, and the manufacture and sale of wine was a state monopoly although, on festive occasions, the right to privately manufacture beer for four days was recognised on payment of a licence fee. There were well appointed liquor shops providing rooms, beds, and seats, with other comforts like scents and flowers. These shops were located at specified intervals, and liquor was sold only to persons of good character and in small quantities. During this period a number of new wines, particularly from grapes, were introduced, although the two most popular varieties 'Kapisayani' and 'Harahuraka' were still imported from Afghanistan.

In the Epics, there are numerous references to drinking. Lord Krishna is said to have enjoyed drinking freely with Arjuna, and

we are told that the Yadavs were killed in a drunken brawl. Even virtuous ladies, like Sudesna, drank wine and some of them drank so hard that they could not walk straight. Inspite of its widespread use even the Kshatriyas, who drank the most, considered it sinful. It is during this period that social class differences appeared concerning alcohol use, e.g. drinking liquor prepared from molasses was considered inferior to the use of other types of liquor and wines. 'Maireya' seems to have been the most popular drink and is said to have been the wine served by the sage Bhardwaj to the party of Bharat.

The principal sources of information for the Kusana and Saka Satarahana period (75 A.D. to 300 A.D.) are the medical treatises of Charak and Susruta, as also the Samhitas of Bhela and Kasyapa. All the medical works prescribe a limited use of wines and consider the habit good for health especially in the winter season. Charak for the first time made a distinction between drinking in moderation and excessive drinking. Whereas the former was regarded as 'pleasing, digestive, nourishing and providing intelligence', the later was said to cause 'various ailments'. Charak commented thus, "Food, which is the life of living creatures, if taken in improper manner destroys life, and even poison, which by nature is destructive of life, if taken in proper manner acts as an elixir". Then in reference to alcohol he went on to state, "if a person takes it in right manner, in right dose, in right time and along with wholesome food, in keeping with his vitality and with a cheerful mind, to him, wine is like ambrosia". On the other hand, "to a person who drinks whatever kind comes in hand to him, and whenever he gets an opportunity and the whole body is dry on account of constant exertion, this very wine acts as a poison". Charak described in detail the different modes of consumptions, the types of wines, and the accompanying foods to be taken by persons of different morbid humours (Kapha, Pita, and Vata) and of the different psychic types (Sattvic, Rajasic, and Tamasic). He also gave a detailed clinical description of the three stages of intoxication and then proceeded to give numerous recipes for the treatment of both the acute intoxication as well as for those addicted to alcohol. The very fact that he had devoted a full chapter to the problem of alcoholism suggested that there had been a considerable number of people who were in the habit of drinking alcohol in excess.

During the Gupta period, there were many references in the Anga Vijja and the works of Kalidas suggesting that the use of alcohol during this period was common. It was even believed that

a state of intoxication gave a special charm to women and that many ladies of royal families (e.g. Indumati—the queen of Aja,) enjoyed their drinks. Police officers, soldiers and their friends were singled out as enjoying themselves by drinking at the liquor shops. The Matsya Puran described Krishna as drinking with sixteen thousand ladies and did not consider him a sinner. Yuan Chwang mentioned that the Kshatriyas preferred wines made from the juice of grapes or sugar-cane, whereas the lower cases, Vaish and Sudras, drank strong fermented drinks. Shastri reported that in South India too drinking was common except among the Brahmins., whereas the rich drank liquor imported from the West, the poor enjoyed country wine.

During the post-Gupta period, it appeared that the habit of drinking had spread to a considerable section of Indian society., even Brahmin youths were described as wasting their time in the company of dancing girls who were addicted to drinking. Somadeva gave an interesting account of such a drinking place in his Kathasaritsagara. At marriages and other festive occasions, drinking was common among the Kshatriyas. Medhatithi also confirmed that while on such occasions Brahmin women did not drink, Kshatriya and other caste women often indulged in excessive drinking.

Drinking is expressly forbidden by the 'Quran', but was recommended by Persian tradition. All the Mughal emperors drank heavily and it was but natural that their subjects should follow their example. The state looked upon the evil of drinking with indifference. Ala-ud-din Khilji was the only monarch who tried to completely suppress drinking by instituting rigorous control on the manufacture and sale of alcohol. In response to these prohibitive measures people resorted to the familiar devices of bottlegging—they began to smuggle spirits concealed in waterskins under loads of hay and firewood. Finally he was compelled to modify and relax his measures, and a new regulation was introduced which did not prohibit the manufacture and use of drink in private but made its public distribution and organisation of big drinking parties illegal. His successor, Mukbarak Shah, continued with these modified rules. The Mughal emperor, Akbar, more out of concern for the health of his subjects than any ideology, tried to control or limit the use of alcoholic beverages. He opened a number of public drinking places which were under official supervision and where a register giving particulars of sale to every individual was maintained. He personally felt that moderate drinking was good provided the person consulted a

physician and took due care of his health and also provided that such drinking did not lead to the commission of a public nuisance. Separate bars were opened for the 'common drunkards' where apparently fewer restrictions were enforced. His son, Jahangir, though himself a moderate drinker, attempted to go a step further, and issued an edict completely prohibiting the sale of wine and bhang, and those who tried to break the law were punished severely. However, it was apparent that this attempt, like the previous, one was not very successful, and by the time of Shah Jahan to quote Mohammed Ashraf, "people took wine like water".

EXTENT OF THE PROBLEM

Alcoholism is characterised by the chronic intake of large amounts of alcohol. Accordingly, one would expect the drinking behaviour of alcoholics 'en masse' to be reflected in the overall volume of alcohol consumption in the population. This expectation is borne out by the observation that the apparent per capita consumption is directly related to the proportion of heavy drinkers in the population. On the strength of this association, it has become customary to use per capita consumption as an index of rates of excessive use of alcohol as well as the Lederman Equation to estimate the numbers of mild, moderate and heavy drinkers in any population. According to the Lederman hypothesis it is believed (a) that the distribution of alcohol consumption is log normal in all populations and (b) that there is a constant relation between per capita or mean consumption and the prevalence of heavy drinking in that population. Apart from criticism of the Lederman hypothesis made by some workers on theoretical grounds, it is important to remember that the Lederman Equation was based on samples of drinkers from western societies in which drinking is socially approved and indulged in by the majority (approximately 70% of the population)—with the result that only a minority are abstainers, while the majority are moderate drinkers and a few are heavy drinkers. It is possible, therefore, that this state of affairs may not hold true for a society or culture where drinking is neither socially approved nor widespread e.g. India where less than 30 per cent of population is estimated to be drinkers, and where we will have a large number of abstrainers, and some moderate drinkers though among the drinkers, this author found in field studies a proportionately higher number of heavy drinkers — much more than was predicted by the Lederman equation based on mean alcohol consumption of the total population, which, in fact, tended to underestimate the number of

moderate and heavy drinkers in the Indian setting. Whereas it was generally believed that only 4-5% of all drinkers became alcoholics, our studies (1978, 1979) in India had shown that the number of heavy drinkers was much higher than predicted (it being nearer 10%) on the basis of mean consumption. Another factor which might contribute to this distortion from the expected log normal distribution was the marked sex difference in drinking habits in our society. Whereas in Western countries the ratio of male to female drinkers is roughly 3:1, in India it is estimated that roughly 50 per cent of adult males drink but the number of female drinkers is negligible (i.e. less than 1 per cent of all females.

There has recently been considerable speculation about the level of alcohol consumption in India. Very few field studies have been carried out to date — almost all of these studies have been conducted in the Punjab although passing reference to alcohol 'addiction' is made in several general mental morbidity surveys. Surya et al. (1964) in a study of mental morbidity in Pondicherry reported that 'alcohol addiction' was 3.6 per thousand. Similarly, Elnamar, Moitra, and Rao (1971) in a study of mental health in the rural population in West Bengal reported that 'alcohol addiction' was 10.8 per thousand. Verghese et al. (1972) in an epidemiological study of psychoneuroses in Vellore town reported that 'alcohol addiction' was 2.1 per thousand. Nandi, Ajmany, Ganguli, Banerjee, Boral, Ghosh and Sarkar (1975) in a study of the incidence of mental disorders in one year in a rural community in West Bengal reported that there were nineteen alcohol addicts per thousand. Dube and Handa (1969) in a study of the drug habit and mental disorders in a population of Agra reported that 0.77 per cent out of 29,468 in general population habitually used alcohol. Dev and Jindal (1974) in a study of the pattern of alcohol use in villages in Ludhiana district of the Punjab found that 74.1 per cent of adult males used alcohol. Mohan et al. (1980) in a study of prevalence of drug use in young rural males in the Punjab reported that alcohol use was very high (58.3 per cent). Sethi and Trivedi (1979) in a survey of a rural area adjoining Lucknow found that 32.1 per cent of males above ten years of age and none of the females took alcohol at least once a month. Venkoba Rao (1978) on studying 178 cases of drug addiction in Madurai reported that 27 per cent of the sample studied was addicted to alcohol and 29.2 per cent of the sample was addicted to both Cannabis and Alcohol. Lal and Singh (1978) in a detailed study of a large village in Sangrur district of Punjab reported that approximately half (49.6 per cent) of the males aged fifteen years and above were taking alcohol, there being only one

female alcohol user. In this study it was found that an overall rate of drug abuse for current users was as 20.4 per 1000 population. There were only two drugs that were commonly used (a) alcohol by approximately 50 per cent of adult males (rates being 174.4/1000) and (b) opium by approximately 35 per cent of adult males (rate being 125.5/1000). It was found that a large majority (89 per cent) of alcohol users were occasional drinkers and only 11 per cent were regular or dependent users. The alcohol users took it for recreation or pleasure. An attitude survey was also carried out. Out of a total of 497 persons interviewed, an attitude of strong approval was expressed by 13, qualified approval by 139, indifferent or non-response in 51, qualified disapproval by 223 and strong disapproval by 71. Thus, approximately 31 per cent could be said to have a generally positive attitude of approval to alcohol consumption, and 55 per cent an attitude of indifference or qualified disapproval, leaving a hard core group of only 14 per cent who were clearly and unequivocally against the use of alcohol under any circumstances. Varma, Singh, Malhotra, Das and Singh (1981) reported that out of the 1031 subjects they interviewed, only 23.7% of them were current users, and 45% thought people could drink 'none at al' without it having a bad effect on their health and 26.2% felt that they could have a few drinks once or twice a month. 59.2% of the sample perceived alcoholism to be a very serious problem in this part of the country (North India) and another 33.1% considered it to be serious enough.

It is evident that most of these studies were carried out in North India. Since there is bound to be a considerable difference in alcohol consumption in different parts of the country it would not be realistic to generalize the finding to the whole country but they do serve as an indicator of the extent of alcohol consumption in the existing Indian socio-cultural setting. In this context it has been documented recently that the world patterns of alcohol use are changing and the production and consumption of alcohol are increasing. A recent statistical analysis of data from 97 countries show that between 1960-1972 production of alcohol beverages rose by more than 60 per cent. This has been attributed to a rise in the prevalence of alcohol drinking in western countries, and secondly to a marked increase in drinking in third world countries. With increased alcohol consumption there evidence of widespread upsurge of alcohol related problems, particularly in many parts of Africa and Asia, where alcohol was previously forbidden by religious rules or social customs. I shall now briefly list some of the alcohol related problems that are likely to result in a community where alcohol consumption is widespread. Alcohol consumption produces certain physical and mental changes, some of these are

acute and reversible, others are chronic and irreversible and st
others permanent. Most of the ethanol is detoxified in the liver a
a constant rate—irrespective of the blood level (equal to a little
under 1 oz. ethanol per hour). Excessive alcohol use has been
reported to lead to a number of psychological, physical and social
complications, which are listed below although exact statistics for
each of them are not available for India.

In 1972 an Expert Committee under the Chairmanship of Prof. V. Ramalingaswami, Director, All India Institute of Medical Sciences, was appointed to report on the effect of human consumption of alcohol in different forms on health and nutrition. The Expert Committee reported that alcohol has the following deleterious effects on human health :—

i) It damaged the liver, it made the liver fatty in the beginning; thereafter it caused alcoholic hepatitis which developed into cirrhosis, finally resulted in liver cancer.
ii) It caused gastro-intestinal dysfunction, and impaired the intestinal transport of nutrient substances.
iii) It directly affected the nervous system, impaired task performance (like typing, driving, mountain climbing, etc.) which is dependent on well functioning conditioned reflexes. Functional disorders of other organs, like the liver, would also affect adversely the functioning of the nervous system.
iv) It affected the heart, causing, 'Beri-Beri' heart and alcoholic cardioinyopathy.
v) It caused diseases of the musole.
vi) It inhibited secretion, of the anti-diuretic hormones and oxytocin.
vii) It suppressed the formation of the red blood corpulson.
viii) It led to malnutrition, by importing the normal processon of food digestion and absorption. It also reduced intake through loss of appetite.

There have been a large number of studies on the relationship between the problem drinker and his family. In general, they confirm that family relationship and social roles are affected by and in turn affect the problem drink. In addition to the other factors, such as the composition of the family, their personality and nature of interpersonal relationships, I would like to stress the over-riding importance of the socio-cultural environment. Not only the nature of various coping mechanisms employed but also the degree of disintegration or breakdown of the family will vary from one society

to another depending upon the extended family, social and institutional and religious supports available, which would explain the absence of the typical skid-row alcoholic in India. Much has been written about the emotional repercussions of alcoholism on the family members, especially the spouse and children. A point worth making is the difference in the types of alcohol related problems likely to occur in a family depending upon their socio-economic and educational status. There is evidence that among the socially and economically weaker sections the health and social consequences are more severe since alcoholism is one of the most expensive diseases that can be acquired. On the other hand, among this group, the local community in general, and wives in particular, are more tolerant of excessive drinking and antisocial or violent behaviour by the males. They are inclined to accept this as inevitable and a part of life, and attribute it to their frustrations and hard manual work. On the other hand, an educated suburban housewife is more likely to protest and create a tense atmosphere in the family with its adverse effects on the spouse and children.

Traffic accidents are one of the leading causes of death in most western countries, and high on the list in most developing countries. India reportedly has the highest death rate per unit vehicle in the world — the death toll being 10 to 15 times that in the U.S.A. or U.K. According to a recent W.H.O. report, India records the highest road fatalities at 61.1 deaths per 10,000 motor vehicles, compared to just 4.7 deaths per 10,000 vehicles in the U.S.A., Spain 20.8, Australia - 22.8, Denmark - 10.6, Netherlands - 11.7, and Norway - 6.1. Errant drivers were primarily responsible for 70 per cent of these accidents, the main reason being that there was hardly any traffic control on our highways. In western countries, a majority of accidents involve private cars, and mostly involve the young or adolescent drivers. Bus and transport drivers are generally having the best safety records. According to figures available for India, however, the trend is just the opposite. Goods vehicles contributed the maximum towards the total road accidents (24 per cent) followed by buses (19.9 per cent). Motor cars and two wheelers came next with 19.77 and 9.5 per cent respectively. A number of factors are responsible for these accidents including (a) the drivers - their driving skill and road behaviour. This in turn depends upon their training, age, sex, marital state, education, socio-economic status and religious beliefs etc, alcohol being only one of the factors affecting their driving ability. The other factors being: (b) the mechanical state or road worthiness of the vehicle and (c) the conditions prevailing on the road, (d) an inadequate and ineffective system for enforcement of road rules on highways.

CHAPTER XV

ALCOHOL CONTROL POLICIES

In general terms, alcohol control policies aim at either: (1) total abstinence, or (2) minimizing heavy or harmful use. Total prohibition at first sight appears to be the logical answer and the money so saved will hopefully be spent on other useful purchases in the market. However, this simplistic view does not take into consideration the use value that alcohol has for the individual. If he is drinking mainly to quench his thirst then his money will be diverted to purchase of soft drinks, but if it is to get high or drunk then it will most probably be diverted to purchase of illicit, and if this is not available, to drugs of similar use value. It is also known that the consumption is closely related to the price of pure ethanol contained in a particular alcoholic beverage variety. Since country liquor is the main type of beverage consumed and the fact that 84 per cent of our population live in villages (i.e. rural areas), sales are not going to be dependent on the relative costs of different types of beverages available, but on the price of the cheapest brand in that type. Hence if good country liquor is available at Rs. 20/- per bottle, the overall consumption is more likely to be related to the availability of this than the price controls on so called 'foreign' liquor sold by official vendors.

Prohibition is now in force only in the States of Gujarat and Tamil Nadu, and in selected areas in Rajasthan, Uttar Pradesh, Karnataka and Maharashtra. In 1960, the Ministry of Home Affairs, in consultation with the State Governments, set up a Central Prohibition Committee to advise the Government on a phased introduction of Prohibition. A study team of Prohibition was appointed in 1983, headed by Justice Tek Chand, to study the working of Prohibition in the different States. Various aspects of alcohol problems were scrutinised and proposals made for offsetting financial losses where state revenue was diminished through prohibition, and recommendations were given for strengthening the legal framework. A plea was made for involving voluntary agencies in the implementation of prohibition, and a plan was developed for extending relevant educational work. Parts of only three states availed themselves of this offer, and one became wet again after a short time. Ii 1973, 1974, and 1975, the Government of India approved of a series of measures aimed at reducing alcohol consumption and preparing for total prohibition. They did not have the desired impact and the Government resolved that from October 1975 a minimum programme for Prohibition should be pursued by the states. A set of prohibition guidelines was formulated and distributed in 1978 based on the Government's

acceptance of the recommendation made by the Central Prohibition Committee in July 1977, for enforcement of total prohibition in four years. However, by 1980 it was becoming evident that these steps were not attaining the desired results. In fact according to a report in the "Indian Express" of 30th March, 1980, under the caption "Punjab's spirited reply to Prohibition" it was noted that instead of a decrease in consumption as a result of implementation of the Prohibition Programme, in the first year Punjab showed an increase of 30 per cent consumption of country liquor (touching a record of 4 crores bottles) in 1979-80 over the previous year. Further, the smuggling of liquor from neighbouring states and illicit distribution had also increased. In view of such reports and the great revenue loss entailed to the state governments on account of reduced official production and sale of alcohol the Prohibition Programme was revoked in most states by 1981.

TREATMENT OF ALCOHOLISM

There are two approaches to the treatment of alcoholism, (1) conventional methods, (2) learning theory techniques.

1. Conventional methods

Conventional treatment approaches imply an eclectic approach using several treatment modalities including, Drugs, Psychotherapy - (Individual or Group) and Eclectic treatment. Firstly the patient is examined for medical - biological problems; secondly his social history may be taken and he may be given psychological tests. Finally, he can be placed in a therapy group with other alcoholics, or have individual interviews with a psychotherapist or counsellor. The patient may be prescribed vitamins (particularly vitamin B, which is often deficient in alcoholics) as well as tranquillizers and antidepressants. He may also receive Antabuse (disulfiram) or Temposil (citrated calcium carbimide) - drugs that produce nausea if the patient drinks after taking them. Individual Psychotherapy involves trying to help the patient to overcome his various personal problems. Group Therapy is thought to be particularly appropriate for these patients who have limited verbal abilities. It can also provide an opportunity for patients to receive social support from individuals suffering from similar problems. The usual goals of conventional treatment programmes are first sobriety, and then total abstinence as well as alleviating other social and interpersonal problems.

2. Learning theory techniques

Learning theory has contributed the aversive conditioning technique. This procedure involves giving the patient alcohol and electric shock, in an attempt to change the alcohol from a positive to a negative stimulus. In addition to conditioning procedures based on electric shock, the same principle has been utilized with chemicals. In this procedure, a chemical (e.g. emetine or apomorphine) is administered and when the patient begins to drink shortly afterward, he experiences nausea and vomiting. This procedure is an attempt to associate alcohol with nausea. The reader is cautioned against assuming that one treatment is known to be "best" for all alcoholics. Pattison makes the case that alcoholic patients, treatment facilities, and treatment outcome vary. He argues that by taking such variations into account, and thereby making better use of existing facilities, considerable improvement could be achieved without an increase in available resources.

Gopalan Committee Report

Subsequently in view of the increased consumption of the alcohol and other drugs, the Government of India constituted another expert committee under the chairmanship of Dr. G. Gopalan, Director-General, I.C.M.R., New Delhi. The committee submitted its final report along with their recommendations in October, 1977. The recommendations fall into four main categories.

1. Legal and Penal measures
2. Educational measures
3. Social action
4. Setting up of a specialised centre in each state.

In essence, the functions of this unit would be:

(a) Treatment of alcohol and drug addicts.
(a) Training of paramedical and other social and voluntary agencies in handling alcohol and drug problems.
(c) To conduct ongoing research into the causes and treatment of alcohol and drug addicition.
(d) It will also function as a central registry and reference centre.

Unfortunately, no concrete action seems to have been taken so far on these recommendations, either at the central, or state level.

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CHIEF ADMINISTRATIVE MANAGER

Phone: Office 361886, Res. 608307
BHARAT ELECTRONICS LTD.
JALAHALLI P.O., BANGALORE-560 013

Date 7th Dec. 1988

Dear Dr. Francis,

Absenteeism in our industry has been posing a threat to our growth. An analysis revealed that one of the major reasons for this was employees addiction to alcoholism and Management was very much concerned over this aspect. It was most revealing when it was noticed that the rate of death in the factory was on the increase, one of the major reasons for this was alcoholism. Since then our Management has been working out various Schemes to reduce the number of alcoholics and thus saving the precious lives of our employees who are still needed to their families. Three years ago, we chalked out our own programme for alcoholics correction. We have corrected over 100 employees during this period. With a view to share our experience with other industries, we propose to organise a Workshop on Alcoholic Correction and Rehabilitation in Industry on Saturday the 17th Dec. 1988 at Hotel Rama, Lavelle Road, Bangalore-560001. We plan to invite senior officers connected with Employees' Welfare in both Private and Public Sector Organisations in Bangalore, besides professionals from medical field connected with this work, social workers of repute etc. The Workshop is divided into four sessions where papers will be presented by eminent personalities. We enclose a copy of the programme for your kind information.

CM 9/12

I shall be grateful if you kindly chair the session on "Alcoholism and its effects".

A line in confirmation will be highly appreciated.

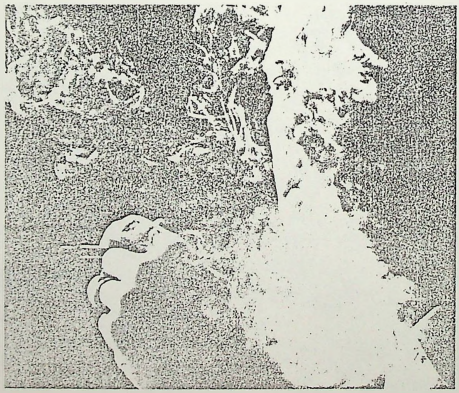
Yours sincerely,

Dr. S.M. Francis,
Director, St.Martha's Hospital,
Bangalore.

March
1987

ALCOHOL AND TOBACCO

Forget cocaine. Here are the killers.



COURTESY AMERICAN CANCER SOCIETY

The poisonous charm of drinks and cigarettes hasn't lost its appeal.

The media-promoted social acceptance of alcohol and tobacco makes it difficult to call these drugs, drugs— mood altering, addictive substances that affect our lives.

But, medically and practically, drugs are what they are and how they must be viewed. The necessary change in mindset which this presupposes, unfortunately, is slow in coming.

All over the world, alcohol production is growing faster than the population; consumption is increasing in total—and in per capita terms.

And tobacco. A recent study released by the Worldwatch Institute in Washington notes that "The leading cause of premature death among adults in 1985 was not Africa's famine, warfare, or the attacks of international terrorists: It was cigarette smoking."

Alcohol and tobacco affect rich and poor, young and old, and men and women differently. But, with all of these groups, alcohol and tobacco are equally seductive and morbid.

nks and cigarettes
strong opposition

· fights a 'brutal process of deterioration'

BY PEDRO ONTOSO
in Bilbao, Spain

L AND TOBACCO are the
ıging drugs in the world today.
use problems are particularly
Europe. In countries such as
ıany, England and the Nordic
ıhol, the preferred drug of soc-
ion, is part of the daily routine
:ctors of the population, par-
ie young
is so u[illegible]rsally accepted it is
part of the great popular festi-
·berfest in Munich, for example
he cult of alcohol is the link
ıarticipants, and where wine
are regarded as the elixir of

ɔiness fades, leaving behind ad-
l pathology. In East Germany,
0 youths are drug addicts; and

ɔ is a staff writer with *El Correo*
ily published in Bilbao.

one of the most commonly used drugs is alcohol. Social analysts use these figures to explain the fact that one-fourth of all convicted criminals are below 21 years of age.

In Spain, where there are over 100,000 bars, there are over 2,300,000 alcoholics. According to a recent study published by the Spanish Ministry of Labor, in the Basque region alone over 400,000 people consume excessive amounts of alcohol, with those between 21 and 24 consuming the most. For the experts who have repeatedly voiced their concern, this is something without precedent in Europe.

"For years," points out Dr. Javier Aizpiri, a neuropsychiatrist and social anthropologist in Bilbao, Spain, "the deterioration in this area (excessive alcohol consumption) has been taking place at an alarming speed. The young, those between 14 and 30, decided to destroy themselves. These groups get together to consume massive amounts of alcohol. There is no communication: they are happy being nothing, just drinking savagely. It reminds me of the mass suicide of the whales. It is a brutal process of deterioration."

Doctors and sociologists have urged the implementation of preventive measures to slow this juvenile annihilation. They have warned of the high social cost to be paid by a society whose maturing process has been disturbed, severely limiting its possibilities of development and adaptation. These measures should include the promotion of healthier alternatives for the use of free time.

According to a study of Madrid's youth, 30 percent of this population spends its money in bars, pubs and cafés, the favored places for social interaction. In East Germany, to take another European example, a study conducted by the Youth Research Institute in Hanover, revealed that the most favored meeting places were fast food restaurants and cafés.

Once used to dull the harsh conditions of the industrial revolution, alcohol is now used to flee post-revolution malaise.

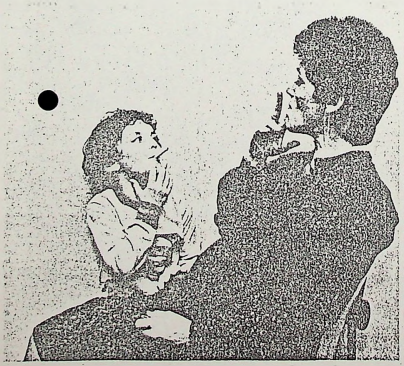
Smoking is the other widely accepted, and dangerous, addiction. Even though smokers are the only drug addicts without a negative social image, tobacco is a main cause of disease. Recently, however, awareness campaigns conducted by several European governments, as well as legislation limiting advertisement of tobacco, are starting to change the image of the smoker. In some countries, such as France, the level of addiction was kept stable.

In Spain, 50 percent of the population smokes, and over 40 percent do it daily. Last year alone, almost 4 billion packets of cigarettes, 823 million cigars and six million bags of pipe tobacco were sold. These figures show just a small increase over those of previous years, maybe a consequence of higher prices.

In less than 20 years, Spain has quadrupled its tobacco consumption. The tobacco industry—a government monopoly—has over 235,000 outlets generating over 123 billion pesetas (almost one billion US dollars) for the state treasury. At the same time, the Ministry of Public Health has revealed that 300,000 Spaniards died last year as a result of tobacco-related illnesses.

The solution for the medical problems of our era—as pointed out by the World Health Conference—is not to be found in research laboratories, but in legislatures. The Spanish House of Representatives has already presented a bill promoting the

PHOTO BY JIM ANDERSON/STOCK BOSTON



The phenomenon of tobacco becomes the problem of tobacco.

ALCOHOL AND TOBACCO

filtering of tobacco products and limiting advertisements directed at those below sixteen years of age. The EEC, as part of its fight against cancer, is proposing, among other things, that European tobacco plantations, which produce about 50 percent of Europe's total consumption, should be replanted with other crops. It has also requested that higher taxes be levied on tobacco products.

In Norway, the National Health Council Against Tobacco developed an ambitious program to eradicate tobacco consumption by 2000. It includes the prohibition of smoking in all public places, including restaurants, hotels and nightclubs by 1990; as well as the proscription of tobacco sales in kiosks and supermarkets, the creation of a government monopoly for importing and retailing, anti-tobacco campaigns financed by a 20-peseta surcharge on cigarettes, and an increase in scientific and epidemiologic studies.

France became aware of the tobacco problem ten years ago when it passed the Veil Law that regulated tobacco advertising and smoking in public places, and demanded that information about levels of tar and nicotine be printed on cigarette packages. Since then cigarette sales have stabilized and the positive image of non-smokers is making great strides.

Today, in Europe, even though three out of four smokers agree that smoking is a pleasure, a still higher percentage feels that smoking is bad. And 72 percent of all smokers believe that smoking over a pack a day is harmful. More important yet, 40 percent have attempted to quit.

Europe, like the rest of the world, is becoming aware of the inherent danger posed by both alcohol and tobacco, habits that end up by costing us dearly.♦

Our New Look

Cleaner, more contemporary

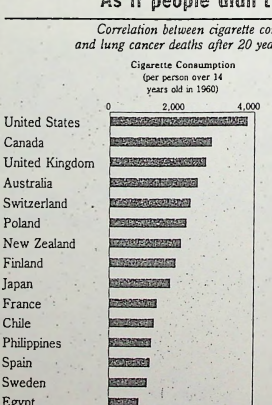
With this issue of WorldPaper we introduce a new layout conceived by Boston-based designer Ronn Campisi.

In the words of every publication with a new design, we are doing this to make WorldPaper cleaner, more contemporary and easier to read. We also have chosen this design because we like it.

We hope you do too.

As if people didn't know

Correlation between cigarette consumption and lung cancer deaths after 20 years of smoking



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Volume IX, Number 3
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BHARAT EARTH MOVERS LIMITED
K.G.F. COMPLEX

COUNSELLING, TREATMENT AND REHABILITATION OF ALCOHOLIC ADDICTED EMPLOYEES.

by

WELFARE DEPARTMENT

KVV/[illegible]
W

BHARAT EARTH MOVERS LIMITED
KOLAR GOLD FIELDS - 563 115

TREATMENT AND REHABILITATION OF EMPLOYEES WHO ARE ALCOHOLIC ADDICTS

In recent years there has been great concern about the excessive use of Alcohol by some of our employees. Alcoholism, is now considered to be a disease and not merely a malady. Excessive use of Alcohol is known to be very harmful not only to the individual, but also to the family, industry and society at large.

We find many workers are the victims of circumstances and environment and thus fall prey to Alcoholism. It also happens due to personality make-up, heredity traits, family problems, work and social environment.

The unhappy individuals are unable to carryout their work in the industry and family roles effectively because of their prolonged abuse of Alcohol.

With a view to rehabilitate employees who are Alcoholic addicts in order to make them more productive workers and responsible family members, a two day seminar was arranged by the Welfare Department with the help of Psychiatrists of NIMHANS, Bangalore during September 1984. The main objective of the seminar was to make the employees addicted to Alcohol, to realise the evil effects of Alcoholism on their personal lives and their poor performance in industry.

Since then, we have successfully counselled 39 hard-core Alcoholics, who having realised their mistakes, during the counselling, volunteered to undergo treatment to overcome Alcoholism. Accordingly, their treatment was arranged at NIMHANS through ESI. Out of the 39 addicts, we have successfully weaned away 24 employees and now, they are productive workers and face their family and social life with more responsibility. A regular follow-up is maintained through the concerned Department Heads, to know their mental/physical health and their work performance as a further step in their rehabilitation.

Employees who have unfortunately reverted back to their former habits are not abandoned. They are further counselled regularly and if necessary, are sent for another course of treatment. Employees who have successfully come out of the habit are also counselled so that, they do not slide back. They are invited to meet the concerned Officers at Welfare Department in groups once a week in order to exchange their views and share, experience and give suggestions if any, that may help other unfortunate victims.

Our efforts are continued to identify other employees who are victims of Alcoholism so that they may receive similar benefits of counselling and treatment to make them productive workers again.

BHARAT EARTH MOVERS LIMITED
KOLAR GOLD FIELDS - 563 115

REHABILITATION OF ALCOHOLIC ADDICTS.

CASE STUDY NO: 1 :

Mr. 'A' aged 48 years has been working in our Company as Watch & Ward Guard since 22.05.1980. He was very irregular in his duties and subsequently he became a chronic absentee. The employee had availed of all the available loan facilities from the company and outside and was in the clutches of indebtedness. He has a family with wife and four children. Since the employee absented himself from duties, he was without salary and his family was neglected and was on the verge of starvation.

His wife and brother came and reported that the employee indulges in heavy drinking and refuses to come for work and his health is in a very poor condition. When the Welfare Officer visited his house, the employee was found to be in a very bad state of health and his family members were in poverty sticken condition. The Welfare Officer counselled the employees on the evil effects of Alcohol and the damages it has caused on his health and family life and the poor performance at his job.

The employee has responded to the counselling in a positive manner and volunteered himself for treatment and expressed his desire to give-up Alcohol. The Welfare Department has arranged for his treatment at NIMHANS, Bangalore through ESI. He underwent inpatient treatment at NIMHANS for a period of 12 weeks (from 13.9.85 to 15.12.85). During the period of treatment, he has co-operated with the Doctors and shown good improvement in his health condition. After his discharge from the Hospital, the employee was in good health and regular in his duties.

The Welfare Department monitors his case by meeting him once in a fortnight and also by obtaining the monthly report from his Department Head. The monthly report clearly shows that the employee is now very good in his performance, attendance and conduct.

During the post-treatment period, the employee was extended all moral support and guidance to solve his problem of indebetedness. During our latest counselling the employee has expressed his happiness and informs us that he and his family are well settled in a spacious house with all comforts like TV Set, furniture etc., and the children are attending to schools regularly. He looks after his wife and children very well.

BHARAT EARTH MOVERS LIMITED
KOLAR GOLD FIELDS - 563 115

REHABILITATION OF ALCOHOLIC ADDICTS

CASE STUDY NO: 3 :

Mr. 'C' aged 43 years has been working our organisation as Turner since 2.11.1974. He is having a wife and four children.

His Department Head has informed Welfare Department that the employee is very irregular in his duties and he is a hard-core Alcoholic. Subsequently, the Welfare Department called the employee along with his family for counselling. During the counselling, it was revealed that the employee is residing in a Mining area where there is a social menace of manufacturing and supplying of illicit liquor and there he had ample supply of Alcohol and he was attracted to Alcohol and became an addict through regular drinking. Sincethen, he has been very irregular to his duties and absent himself for days together under the influence of Alcohol.

The employee was counselled during which he has realised the futility of Alcoholism and volunteered himself for treatment to give-up Alcohol. Meanwhile, the Welfare Department has arranged for his treatment at NIMHANS where he underwent inpatient treatment for 5 weeks (from 18.1.85 to 26.2.85). After discharge from the Hospital, the employee has resumed duty during February 1985. Sincethen, he is very good in his attendance, conduct and performance as reported by the Department Head. Welfare Department is simultaneously monitoring his case and meeting him once in a week. In order to give him a change of environment, the employee has been allotted a Company quarter on out-of-turn basis in the BEML Township. Now, the employee is very hale and healthy and leads a happy family life and also a productive worker in the factory.

B.E.M.L. KGF COMPLEX

STATEMENT SHOWING THE TREATMENT AND REHABILITATION OF EMPLOYEES ADDICTED TO ALCOHOL

Sl. No.	Year	No. of employees treated for Alcoholism	No. of employees given-up Alcoholism	No. of employees gone back to Alcoholism
1.	1984	1	-	1
2.	1985	20	12	8
3.	1986	4	1	3
4.	1987	3	2	1
5.	1988	11	9	2

STATISTICS

Total No. of employees treated for Alcoholism	No. of employees completely given-up Alcoholism with %	No. of employees gone back to Alcoholism with %
39	24 - 62%	15 - 38%

-

B.E.M.L. KGF COMPLEX

COUNSELLING, TREATMENT & REHABILITATION OF ALCOHOLIC ADDICTED EMPLOYEES

FAMILY STRENGTH-WISE DETAILS

Family Size	Total No. of employees treated for Alcoholism	Total No. of employees who have completely giveaup Alcoholism and continuing so	Total No. of employees who slided back to Alcoholism.
Employees having 3 children	24	16	8
Employees having 4 children and more	15	8	7

QUALIFICATION-WISE DETAILS

Qualification	Total No. of employees treated for Alcoholism	No. of employees completely given-up Alcoholism and continuing so	No. of employees slided back to Alcoholism
1. PUC & Above	2	1	1
2. SSLC/NAC/NTC	23	16	7
3. Below SSLC	14	7	

AGE-WISE DETAILS

Age Group	Total No. of employees treated for Alcoholism	No. of employees completely given-up Alcoholism and continuing so	No. of employees slided back to Alcoholism
1. 30 to 40 Years	14	5	9
2. 41 to 50 Years	22	17	5
3. 51 years and above	3	2	1

B.E.M.L. KGF COMPLEX

COUNSELLING, TREATMENT & REHABILITATION OF ALCOHOLIC ADDICTED EMPLOYEES

TRADE-WISE DETAILS

Trade	No. of employees treated for Alcoholism	No. of employees given-up Alcoholism	No. of employees slided back to Alcoholism
1. Turner/Fitter/ Grinder/DBM Operator/Miller	14	10	4
2. Welder	6	4	2
3. Watch & Ward	3	2	1
4. Crane Operator	2	2	-
5. Driver-cum-Mechanic	2	1	1
6. Sanitary Helper	3	-	3
7. Clerk	4	2	2
8. Messenger-cum-copying machine operator/Helper	5	3	2

SHOP-WISE DETAILS

Department/Shop	No. of employees treated for Alcoholism	No. of employees given-up Alcoholism	No. of employees slided back to Alcoholism
1. Machine Shop	7	5	2
2. Fabrication Shop	1	1	-
3. Security Dept	3	2	1
4. Plate Shop	6	4	2
5. Electrical Maintenance	1	-	1
6. Tool Room	2	2	-
7. Transportation	3	2	1
8. House Keeping	3	-	3
9. LW Assembly	2	1	1
10. Mechanical Maintenance	1	-	1
11. Stores	3	2	1
12. CT Assembly	1	1	-
13. Progress Dept	1	1	-
14. Gear Shop	1	-	1
15. Accounts Dept	1	1	-
16. R & D	1	-	1
17. Hydraulic Shop	1	1	-
18. Heat Treatment	1	1	-
	39	24	15

CAN ALCOHOLISM BE ERADICATED ?

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Pathological was of alcohol resulting in social and occupational impairment with development of tolerence and withdrawal symptoms is designated as Alcohol dependence.

When a clinician is confronted with the question " Can alcoholism be cured?" by a relative of an alcoholic or alcoholic himself, varied responses ranging from optimistic to pessimistic is obtained which surprises the kith and kin of an alcoholic, leaves an enquiring mind bewildered. What is the truth then?

For the cure or management of an alcoholic it is very necessary to have a brief idea about the cause of alcoholism which enables us to plan the treatment well, upon which success depends.

Alcoholism is considered as an genetic disease, learnt habit, a disorder with lack of control. Alcoholism could be secondary to many other mental illness (Depression, mania etc.) or no obvious cause could be detected which can be termed as primary alcoholism. In both the type faulty personality is the common factor.

Ideal approach to manage any illness are to remove the cause, treat the effects of the illness and environmental manipulation. Till recently alcoholics have been managed in different ways depending upon the school of thought to which one belongs to, hence the various reactions. It is not uncommon to come across, an alcoholic undergoing various kinds of treatment with temporary/permanent improvement, or alcoholics discarding this havit for ever on their own, or alcoholics showing marginal or no improvement

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with the best treatment available to them. The real cause for this is the patients faulty personality aggrevated by environmental factors. Various methods of treatment are not to be blamed completely, as different methods are not suitable to all alcoholics, including the Alcoholic Anonymus approach which is claimed to be the only answer to this problem.

Ideal treatment approach should be one of the humaine approach with merits of behavious therapy, individual and group psychotherapy, marital councilling, regular follow up for 2-3 years along with drugs. This will enable us to achieve better results than any single approach. The aim of this approach is to make an unhealthy mind into a healthy one as much as possible, so that faulty habit dies. Everyone knows how difficult it is to get rid of a bad habit and how much time it takes for one to develop a good habit. Similarly it is not easy to convert an alcoholic into a teetotaler. As follow up studies have clearly shown 80-90% relapses occur during the first 6 months - 1 year after discharge.

For our convenience we can devide alcoholics into motivated and poorly or un-motivated group. The former group, shows good results with or without any kind of treatment. Latter group form the major bulk of alcoholics in whom high failure rate is met with. In this group motivation can be increased with measures which increase the fear in their mind about their health, job etc., which has to be done in a scientific way. This needs good co-operation of the employer, with the clinician. Added to this government authorities (police, Temperence Board etc) should have a tough altitude regarding availability of liquor, driving under alcoholic intoxication etc. To maintain what we have achieved it is necessary to have a continuous support from family members, society, employer, patients friend circle, and regular follow up with the Clinician.

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It is quite clear now that it is not a single citizen's battle with an alcoholic but of the whole societies against alcoholism. Last but not the least, let us not forget that all alcoholics are not criminals/animals but human beings with a bit of love, affection, in their heart, like any one of us, which needs to be kindled with love and warmth so that the lamp of knowledge, discipline, responsibility and fear glows brightly in his mind making him a responsible citizen of our country and not a burden.

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INDUSTRIAL ALCOHOLISM AND LOSS OF PRODUCTIVITY: A STRATEGY FOR INTERVENTION

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PAPER PRESENTED AT THE WORKSHOP ON 'ALCOHOLISM AND ITS EFFECTS' ORGANISED BY BHARAT ELECTRONICS LTD. ON 17th DECEMBER 1988.

INDUSTRIAL ALCOHOLISM AND LOSS OF PRODUCTIVITY: A STRATEGY FOR INTERVENTION.

Problem drinking in industries varies between five to 15 per cent, using different criteria for identification of cases according to various surveys conducted in India. In recent years, professional attention has increasingly been directed to the workspot as a potential locus for identifying the problem drinker. Most problem drinkers have jobs. They can be identified relatively early by the evidence of impaired work performance. Occupationally oriented programmes, offering help rather than dismissal, yield the highest reported rates of successful recovery from problemmes related to alcohol. Such intervention prevents the advance of the problem drinking to more serious stages.

In developed countries, the Employee Assistance Program for Alcoholism (EAPA) has been introduced, based on recognition of the adverse effects of problem drinking on productivity, social consciousness on the part of the management and specific awareness of the drinking problem within the industry. It has been very successful in organisations of large or medium size, employing more than 1000 workers. The workplace is viewed not merely as an agency of referral but also as an active force in the rehabilitation process.

PROJECT DESIGN:

Most programs directed towards helping problem drinkers make use of the available community resources. The more successful ones have a wider approach, including counselling for the employee and his family and identifying the possible environmental or intra-familial stressors that may maintain the problem.

Establishment of a central consulting service:

Merging of the EAPA with health and safety programs may present a logical starting point in developing an intervention strategy. In organisations where medical facilities exist, it would be desirable to establish a consulting service as an additional component. The central consulting service can preferably be situated at the industrial medical centre. A consultant psychiatrist from the department of psychiatry of a general hospital may be appointed as the programme director and coordinator. The physician in charge of the industrial medical centre, the personnel manager of the organisation, the industrial social worker and a clinical psychologist would form the rest of the team.

The central consulting service would specify the procedures for identifying the problem drinking individuals and referring them to the program. It would ensure the co-operation of the management and labour unions, educate the entire work force along with their families and carry out the training of the various supervisory personnel in the identification and follow-up of the problem. Apart from these, it would maintain the administrative liaison between other community facilities (e.g., the Halfway Home) and the general hospital, and conduct a periodic evaluation of the cost effectiveness of the program.

Identification of the problem drinker:

Many of the earlier industrial programs relied on the signs and symptoms of alcoholism for the identification of the problem drinker. However, training the work supervisors to detect such signs and symptoms have been found to be impractical. Moreover, the supervisors are usually reluctant to label and employee as an alcoholic, and often do not report their findings.

In most instances, problem drinking by an employee will manifest itself in impaired work performance and absenteeism, two parameters which are easily identifiable and more objective than signs and symptoms. The supervisors would not require extensive trainin with regard to these parameters and would find them easier to use. In addition, self referral, peer referral and referral through increased awareness of the program would be encouraged.

Offering assistance to the problem drinker:

In the developed industrial nations, two methods of referral are adopted - Firstly, there is the method of "constructive coercion", using a series of corrective interviews with the problem drinker where confrontation is the main stance. The second method is to provide assistance to motivated problem drinkers who seek help on their own. Such a program, though more acceptable, would involve major attitudinal changes in the family of the problem drinker, the labour union, and the drinker himself. Bringing about such changes on a large scale will require well planned strategies.

Stage of referral and documentation:

Once the offer is accepted by the problem drinker, he may be evaluated by the clinical psychologist at the central consulting service, to assess his motivation and to identify possible precipitating and maintaining factors for his drinking problem. He and his family would be informed about the method of treatment and their co-operation sought. Their personnel manager would be approached for necessary assistance regarding leave from the job for detoxification. After the initial documentation, the employee would be referred to the general hospital for detoxification and introduction of disulfiram if necessary.

Management in the general hospital:

Detoxification, treatment of complications if any and introduction of disulfiram would be the goals of management in the hospital, which will be the most expensive phase of the program. Three weeks of admission on an average would be necessary for this stage. This may be carried out as an ongoing program, hiring a fixed number of beds in the hospital, with additional staff for the same to be arranged by the industry. The additional staff required would include a medical officer, nursing staff exclusively for the project, and the clinical psychologist from the central consulting service who would also spend fifty percent of the time in the hospital. The consultant psychiatrist, in addition to being the program director, would also have the responsibility for the treatment of those admitted in the hospital. Initiation of counselling for the individuals and their families, identification and treatment of major psychiatric disorders and medical complications will be the other responsibilities of this team. The presence of the clinicial psychologist at both the central consulting service as well as the hospital to provide counselling and support for the individuals and their families would assure the continuity of the treatment and bolster the confidence of those being treated. At the end of the hospital stay, the individual is either returned to the factory for resuming active duty immediately, or sent to the half-way home if prolonged care is required.

Follow up and maintenance of abstinence:

It is known that frequent follow ups results in a higher probability of success in abstinence of alcohol. Since the most efficient follow up may be conducted at the workplace and since objective assessment parameters are available in this location, it could be carried out to advantage by the clinical psychologist at the central consulting service. Those who drop out from follow up may quickly be identified and their families contacted by the social worker. Regular follow up, assessment of performance parameters and contact with the families would be beneficial, because the cases with relapse would be spotted early and dealt with as necessary. It would also ensure accurate documentation of the data available for follow up. The problem drinkers are to be regularly followed up for a period of at least one year, since the maximum relapses are seen to occur in this time.

Training of personnel involved in the program:

The various personnel who would require training are the section supervisors, in identifying and reporting impaired work performance; and the medical team of the industry, in managing alcohol induced disulfiram reactions. In the course of time, the medical team may be given the technology and training to maintain the project on their own.

The half - way home:

A proportion of the problem drinkers would have deteriorated sufficiently to warrant additional, more technical and graded care for rehabilitation. Such individuals are to be referred to the half-way home after discharge from the hospital, where they may have to stay for three to six months.

ROLES OF THE DIFFERENT MEMBERS OF THE TEAM:

The clinical psychologist provides the important therapeutic management and gives the continuity to the program at its various stages, and at the different locations of treatment. His time is to be divided between the hospital and the industrial medical centre. Once the project is started and the problem drinkers are identified, the emphasis of the educational and motivational programs shift to the families of the drinkers. This is the primary responsibility of the psychologist. In addition, development of documentation systems, counselling the individuals and their families, helping them to cope with the intrafamilial and adaptive stessors, and carrying out subsequent followups of cases would be the responsibilities of the psychologist. Once an individual reaches the central consulting service, he is taken over by the clinical psychologist until he eventually leaves the program after successful follow up. Apart from these, evaluation of the current problems faced by the project and information to the rest of the team regarding the same are to be dealt with by the psychologist.

The social worker's primary role is to bring about attidudinal changes at the beginning of the program, through meetings of labour unions and the management, and meetings of family groups. This is essentially a program of health education. Apart from this the social worker can facilitate the liaison with the section supervisors in the identification of cases. He would maintain the liaison with other treatment agencies like the half-way home. He would also take part in the follow up by conducting home visits in order to detect and motivate dropouts. He would also keep track of public opinion and acceptance of the program among the different categories of workers in the industry and their families.

The medical officer of the industrial medical centre would primarily assist in the integration of the program in the existing medical service of the industry, and provide supportive medical management that may be required during follow up. He would ensure the availability of different personnel for the training and health education programs which are integral parts of the project.

The additional staff are recruited as and when required according to the necessity of the moment and facilities available in the general hospital and industry.

CONCLUSION:

Alcoholism manifests itself in the work place in the form of deteriorating job performance, usually far in advance of any physical symptom of the disease. Employee Assistance Programs for Alcoholism have been able to successfully control alcohol related loss of productivity among the industrial workers. This method also has an inherent advantage in that the population catered for is immobile and thus available for easy identification and follow up. Finally the cost effectiveness is considerable in terms of productivity to the industry, relief of problems to the family; reduced expense related to consumption of alcohol and management of alcohol related medical problems. However, in most programs the referral rates tend to be high in the early phase of the EAPA operation when it is easy to identify the most troublesome and chronic problem drinkers, but decline once these employees have been treated. It is expected that the industrial medical centre will be able to adopt the project technology in time and continue the program at a lower cost, at the stage when referral rates are low.

TTK HOSPITAL

TTK HOSPITAL

Treatment Centre for Alcoholism and Drug Addiction

TTK HOSPITAL

TTK Hospital/T.T. Ranganathan Clinical Research Foundation is a secular, non-profit, voluntary welfare organisation dedicated to the treatment and rehabilitation of persons addicted to alcohol and drugs.

Established in the year 1980, this institution is the first of its kind in India.

The 55 bed hospital offers the services of a team of competent professionals, skilled in their areas of specialisation and deeply committed to the mission. A comprehensive treatment facility covering both medical and psychological help is provided by the hospital in the treatment of alcoholism and drug addiction.

The facilities offered at TTK Hospital include:

- detoxification centre
- emergency ward
- general wards
- special rooms
- family ward
- therapy centre
- counselling units
- family therapy centre
- recreation centre
- dining hall

ADDICTION:

A DISEASE REQUIRING TREATMENT

Alcoholism or drug addiction is not a moral weakness, sin or crime.

It is a DISEASE.

A chronic and progressive disease that leads to severe physical, emotional and social problems.

As a serious health problem that cannot be neglected, the disease of addiction requires intervention and treatment.

Our authentic experience in having treated over 3000 patients during the last 8 years, has strengthened our belief that "addicts when provided with timely treatment and support, can lead better lives free of alcohol and drugs". This deep-rooted conviction supported by experience, forms the underlying philosophy of treatment provided at the hospital.

OBJECTIVES

Treatment at TTK Hospital aims at:

* total abstinence from alcohol and drugs for life and
* effecting positive changes in the behaviour and attitudes of the individual to enhance the quality of his life.

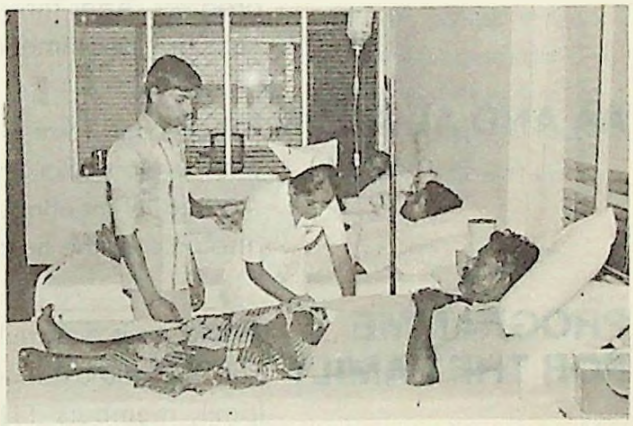
The treatment programme has been drawn up to offer the patient medical help and psychological support that will enable him to recover from the disease of addiction.

Family members are also educated about the disease and are provided with guidelines to improve their quality of life.

IN-PATIENT TREATMENT

The in-patient treatment programme at TTK hospital is a residential, multi-disciplinary therapeutic programme, conducted by a professional team of psychiatrists, physicians, psychologists, social workers, counsellors and nursing staff. The duration of the treatment programme is 4 to 6 weeks.

Incoming patients are directly admitted to the detoxification centre where the required medical treatment is given. Withdrawal symptoms due to sudden stoppage of drug usage, instances of acute intoxication and chronic health problems associated with addiction are dealt with during detoxification.



When the physical condition of the patient stabilises, he is transferred to the psychological therapy wing.

The psychological therapy comprises individual counselling, lectures, group therapy, relaxation techniques, recreational activities and educative films. Individual care and attention are given to each patient during therapy.

FOLLOW-UP

Follow-up forms an important part of the treatment at TTK Hospital and is maintained for a period of five years. Patients are asked to participate in an after-care programme held every week at the hospital. They are encouraged to meet the doctor and their counsellors every fifteen days in the initial stages to seek medical advice and report on their progress. After three months, monthly follow-up visits are recommended.

AA AND AL-ANON

Patients and family members are encouraged to attend Alcoholics Anonymous (AA) and AL-ANON meetings regularly. Meetings are also held at the hospital premises.

PROGRAMME FOR THE FAMILY

Addiction is a 'Family illness' that affects not only the addicted individual, but also his family members. TTK hospital offers a family programme providing information about the disease of addiction and its impact on each member of the family.

The family is given emotional help to cope with the stress caused by the behaviour of the addict.

The duration of the programme is two weeks. The programme includes lecture sessions, group discussions, assignments, relaxation techniques and AL-Anon.

PROGRAMME FOR THE CHILDREN OF ALCOHOLICS

Children form an integral part of the family. Addiction destroys feelings of love, security and warmth which are necessary for the normal development of children. These children need a lot of help and understanding.

The programme for the children of alcoholics consists of story-telling, exercises, drawings, etc. These are aimed at educating them about alcoholism, relapse and recovery.

SOCIAL SUPPORT PROGRAMME

The Social Support Programme aims at exploring the possible support the recovering patients can receive from the society in which they live and utilising it towards their recovery.

The support persons are usually family members — other than the spouse or co-workers or friends. Contact with the support person helps in stabilising recovery and ensuring regular follow-up.

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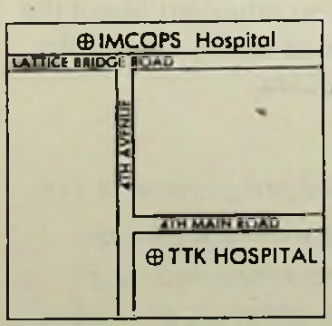

For additional information, contact:

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MH-2A-45

ALCOHOLISM AND ITS EFFECTS - WHAT CAN YOU DO?

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INTRODUCTION :-

Alcoholism has been a growing problem in our country during the past one to two decades. Similarly, this has been a difficult problem to deal with in many other countries of the world too. Several surveys carried out in different parts of our country have pointed to the growing prevalence of abuse of alcohol and various types of drugs. There have been other indicators of the increased prevalence of alcohol abuse too, for eg. it has been shown that the number of alcoholics being treated in our psychiatric wards have been steeply increasing. Persons admitted with various physical complications of alcohol like cirrhosis of liver has been on the increase too. A fairly high percentage of road accidents and fatalities related to such accidents has been alcohol related. One estimate has shown that a third of the drivers on our highways are under the influence of alcohol. Large number of crimes committed by individuals, like burglaries, assaults, rapes etc. are also committed after consumption of alcohol. Many of the growing number of suicides have been reported to be alcohol related. It is roughly estimated that there are more than 3 million alcoholics in our country. Yet another pointer to the problem of alcohol in our country is the amount of alcohol produced. In 1985, 331 million litres of pure alcohol was used, for production of various brands of alcoholic beverages. Today, we have more than 200 brands of whisky, 50 brands of rum, 30 brands of brandy, 50 brands of beer, 10 to 15 brands gin and wine, and over 100 varieties of country liquor being marketed all over the country. In addition, illicit distilling is also a growing industry.

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WHAT IS ADDICTION/ABUSE/DEPENDENCE ?

There are several substances like alcohol and different varieties of other drugs, which when consumed in certain quantities, influence people's thoughts, emotions, sleep, apetite, sexual functioning, social interation and various other aspects of behaviour. These substances also give'a sense of well-being' (euphoria) when people are under their influence. Because of these effects, people who have used these substances several times, have a tendency to continuously use them. When such constant abuse occurs people can get addicted to or dependent on such substances. For a person to be called an addict or drug dependent, he should have an uncontrollable and strong desire (craving) to procure the drug, constantly and consume it. He would not be able to stop the drug taking without help and when stopped, would develop various kinds of physical and psychological withdrawal symptoms. A tendency to increase the dose of the drug abused (tolerance) is another indication of the person becoming dependent on the drug. The common psychological withdrawal symptoms are an irresistable craving for the drug, irritability, restlessnes, feeling bored and disinterested, difficulty in concentration and work. The common physical withdrawal symptoms to various drugs are muscular aches and pains abdominal cramps, vomiting, diarrhoea, sweating, sleeplessness, running nose, tears, chills and fever and at times unfounded fears, confusion, violent behaviour and fits.

WHAT ARE THE CONSEQUENCES OF ALCOHOL ABUSE ?

The consequences would be primarily dependent on 3 factors, namely, the type, amount and period of alcohol abuse, the personality of the user and the social situation

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of the user. However, the various consequences can be considered under the following heads :

- health
- behaviour
- family
- work or occupation
- financial
- police and law enforcing authorities

The abuser initially shows varying types of altered behaviour as a consequence of regular alcohol taking. Many of these may ultimately result in psychiatric complications like Hallucinosis (perceiving things without any stimuli, continuously), Paranoia (abnormal and unfounded suspicious), Psychosis (complete loss of touch with reality) Dementia (various forms of memory losses). A person who chronically abuses alcohol can also develop various physical problems. The common physical problems are frequent infections, under-nourishment, degeneration of the liver etc. In addition such people are also prone for different types of accidents leading to injuries of head and other parts of body. The consequence of drug abuse on the family are constant tensions, frequent quarrels, violence between spouses, child abuse etc. and it may also lead to divorce and seperation. Alcoholics and drug addicts also have problems in retaining regular jobs and satisfactorily performing in their work situation. Constant decline in the efficiency may ultimately result in losing of employment. Yet another problem which addicts have to constantly face is financial problem. They slowly lose their ability to support their families and ultimately have also difficulty in finding sufficient money to procure their alcohol. This leads to their getting involved in stealing, burglary etc. Stealing initially starts in their own household, but later extends to other households too. Many addicts get into problems with the police and the law enforcing authorities.

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WHAT CAUSES ADDICTION ?

The various factors which interact to ultimately produce addiction in a person is not clearly understood. Many factors have been implicated which include genetic (hereditory) other biological, psychological and socio-cultural factors. It is currently believed that addiction occurs due to a complex interaction of the several factors.

WHO IS AT RISK OF MIS-USING ALCOHOL ?

It is widely known that men out-number women in problem of addiction all over the world. It is, young and middle-aged men who are at the highest risk of becoming addicts. Amongst them, it is the unemployed, bored and disinterested people as well as people going through various life-stresses who are at higher risk. As far as socio-economic factors are concerned there are no specific factors which consistently correlate with addition. Rich, poor and belonging to all economic classes as well as the educated, illiterate and with various types of educational background are equally prone to addiction. There are certain occupations which might aggravate the proneness for developing addiction. (eg. doctors, nurses, pharmacists, medical students, druggists, chemists etc. are much more prone for different types of drug addiction than the general population). Similarly, bartenders, waiters, excise personnel and dealers have a higher proness of becoming alcoholics.

TREATMENT FOR ADDICTION :

Most treatment strategies were developed 30/40 years back, and these strategies have been used with varying success. The first step in the management of an addict is to motivate him and his family for accepting treatment.

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Once this is achieved, a period of one to two week is taken for 'detoxification' which is generally done under an institutional setting with medical supervision. This is to take care of the withdrawal symptoms which the person may develop during the initial period of abstinence.

Following this, the person undergoes a treatment programme consisting primarily of counselling, and individual and group therapy in a residential setting. Special techniques like aversive conditioning may also be incorporated into the treatment programme during this stage. Ultimately, the person is put on a drug-deterrent agent like antabuse in alcoholism and discharged from the institutional setting. Subsequently, regular follow-up and entry into an after-care programme is encouraged. Persons are also encouraged to join self-help groups like alcoholics anonymous. During the therapy for the addicts, some kind of therapy is also taken up for the family members, like wives of alcoholic, or children from alcoholic families. Relapses are more the norm than exception in the management of alcoholism and drug addiction. The best of the treatment programme have only about 25 to 30 percent of success rates if success is considered as 3 to 5 years of abstinence continuously since discharge from the treatment programme.

WHAT CAN YOU DO FOR SOLVING THE PROBLEM OF ALCOHOLISM IN THE COMMUNITY ?

The following twelve steps are mentioned for any community leader who is interested in the problem of alcoholism and drug abuse in this community. Parish priests, village leaders, school and college teachers, community health workers, panchayat and zilla parisad members are all included under the term community leaders :

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- learn about basic aspects of alcoholism and drug abuse.

  This can be achieved by being aware of the problem through various kinds of simple booklets and other publication available on this matter, regular scanning of the media etc. Learn also about the existing resources in your own area/district/state for the management of alcoholism and drug addiction.

- detection and appropriate assessment of alcohol and drug-related problem in the community.

  Detection is not likely to pose much problem once, one is sensitised to the evils of alcoholism and drug addiction. However, a way of detecting person with this problem is by asking people routinely about drug use. Of course, the questions will have to be asked non-judgementaly and non-critically and people should be re-assured of confidentiality and your concern for their problems. Once detection has occurred, the next step is a detailed assessment of the problem. Enquire into the nature, severity, type, amount, frequency, duration, pattern of the drug taking habit. Enquiry should also be made into heald problems, family problems and other related social problems. This sort of an assessment will facilitate better understanding by the addict as well as his family of the problems. Assessment should lead to search for solutions and appropriate action accordingly.

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- learn and develop simple counselling skills:

  To work continously with addicts and their families one has to develop skills of establishing a relationship of trust and confidence with the addict and his family (rapport).

- refer to a resource centre :

  If the addict who is detected and assessed requires institutional treatment through professionals, refer them to such a centre. Referral does not mean only naming the facility but it should also include clear information about the centre as well as what the addict and his family can expect from such a treatment centre.

- Provide continuous re-assurance about your own interest and involvement in the Welfare of the addict and his family.

- help the addict and his family with the associated medical, social and legal problems which may have arisen as a result of the drug abuse.

- do not get discouraged with relapses. Relapse is the norm rather than the exception in the management of drug abuse. Some people may recover completely only after 15/20th attempts.

-Continue your supportive visit to the families of the addicts. Even if there are failures of the Management of the addicts, the other family members of addict will benefit from your continued visits.

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- accept limitations. There are few causes which nobody can help. Repeated treatment failures should not discourage you. Discuss with more experienced persons.

- develop new and innovative resources for the help of addicts and their families. You may think of starting voluntary associations, self-help groups for former patients or an alcoholic annonymous branch in your parish.

- enlist/mobilise community support. Take up measures to increase awareness of the community about drug and alcoholic related problems. This can be done by organising talks, exhibition, essay competitions etc. Once people understood the various problems related to alcohol and drugs, they will be more willing to help prevent various problems as well as detect cases early and help addicts to recover.

- Remember - CONFIDENCE WILL COME WITH KNOWLEDGE AND PRACTICE.

COMMITTEE OF CONCERN IN BHARAT ELECTRONICS

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A REPORT ON
CORRECTION AND REHABILITATION OF ALCOHOLIC EMPLOYEES
IN
BHARAT ELECTRONICS

Bangalore-13.

17th December 1988.

A Report on Correction and Rehabilitation of Alcoholic Employees in Bharat Electronics.

INTRODUCTION :

Absenteeism is one of the illness of Industrial Organisations. Research studies have identified its nature and causes. The causes for absenteeism may be grouped into two categories (a) sickness, indebtedness, laziness, lack of awareness, lack of interest towards widening the scope of life, mal-adjustment to a given situation, inclination to other business, job dissatisfaction, habit of easy earning without labour, and like other causes may be grouped as the first category. These may, no doubt affect the organisation in terms of production and productivity. But an organisation itself can tackle them appropriatly by adopting systematic counselling, motivation, guidance, and other corrective measures. (b) The second group of character of absenteeism are Maladoptive behaviour like addiction to alcohol and drugs. Besides loss of production and productivity, the latter generates other multifarious human relations problems to the Management, as the addicted employees are controlled not by themselves but by the chemical they consume. So it is beyond the limits of the Management to bring them back within the socially accepted norms and rightly fit them into the organisational setting. Yet many organisations are groping for solution to the second category of absenteeism, wherein majority of the chronic absentees fall under this group.

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Problem of Absenteeism in Bharat Electronics :

Bharat Electronics, Bangalore Complex, is engaged in manufacturing Electronic equipments and components. We have engaged about 13,000 employees, about 10% are habitual absentees. Indeed the observation and experience of the Management shows that 50% of the absenteeism is caused only by those who have history of Maladoptive behaviour of Alcoholism.

Alcoholism, therefore, is the major contributory factor for absenteeism in Bharat Electronics. Eventually the Management was facing not only loss of production and productivity but also other problems of human relations.

It is in this background, that an organisation running a Half-way Home, CAIM got in touch with us. CAIM defended the theory of alcoholism putforth by the Alcoholic Anonymous and belived that it alone could take the alcoholics to sobriety. They further pointed out that -

1. Alcoholics are not delinquents. They are suffering from the disease of alcoholism. So they should be considered as really sick person.
2. Neither the members of the families of alcoholics nor the general public have no/less knowledge about alcoholism and they are simply believe that the behaviour of alcoholics was intentional. Hence, hardly they could get out from the vicious circle.
3. While taking steps to treat alcoholics, first, one should accept the alcoholics as they are and should consider them as patients.

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4. They should be kept away from the members of their family, atleast for a period of 3 months and put them in half-way home to unlearn their behaviour of alcoholism and support them to ~~get~~ re-learn to live happily without alcohol.

5. For the first 10 days of the 3 months treatment, they should be detoxified in one of the nursing home under the observation of psychiatrist and treat them for their withdrawal syndrome and other physical ailments if any.

6. During their stay in the half-way home, they should be provided nurished food and should engage them with 24 hours tight programme.

7. They should be taught physical exersice and yoga to relax and reactivise their physical function.

8. The attitude of the alcoholics towards drinking should be changed through group therapy and one-to-one therapy in order to bring out their hidden feelings.

9. Since the denial is ~~the~~ one of the character of the alcoholics, confront them appropriately with documents and help them to accept that they are really suffering from the dis-ease of alcoholism.

10. Teach them the 12 steps envisaged by the Alcoholic Anonymous (AA) and motivate them to attend AA meeting every day.

11. With a strong confirmation and commitment to the AA concept, they said, the alcoholics are powerless. They lost control over drinks. No human beings could stop them from drinking. So they should depend on only God or Super power as one's own belief to maintain sobriety.

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12. Meeting the members of the family of alcoholics and the shop floor supervisors as and when the situation arises and help the family members to make aware of alcoholism and the way of dealing with them.

They suggested, the Management to start Employee Assistance Programme and identify the poor job performers and their causes for it, pick up the poor job performers whose causes are come under the group of Alcoholism. Since denial is part of the disease of alcoholics confront them with necessary documents and refer them for alcoholic treatment at half-way home.

CAIM also made it clear that Alcoholic Anonymous does not believe in pharmacholgical approach in resolving the problem of alcoholics, as there would be chances for causality or adverse effect while administring drugs. Instead of depending on alcohol the patient may depend on drugs.

Taking the suggestion of CAIM and analysing the condition in the factory, it was in principle agreed to send our alcoholic employees to the correctional institution for treatment and rehabilitation. Further to make the scheme more effective, it was also decided to involve the Trade Unions, Labour Welfare Fund, and the Departmental/Section Heads of various departments, particularly where there was more concentration of alcoholic employees. Subsequently many meetings were held with the representatives of the trade unions to make an awarness among them about alcoholism and its effect on the individuals as well as on the organisation. The Negotiating Trade Unions responded positively and appreciated the stand of the Management. They also assured that their co-operation, support and participation, would always be there for the good cause of rehabiliating the alcoholic employees and their family.

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c) Employee who expresses his inability to control his drinks and voluntarily approach the Management for help will also be sent for treatment after varification.

5. The Offer of assistance to help to resolve such problems shall be in an confidential manner.

6. Consideration would be given for the use of annual leave or leave without pay for those who are sent for treatment as is granted for ordinary health problems.

7. The programme aims at rehabilitation and not at elimination of an employee. It is neither pro-Management nor pro-union. It is just pro-patient.

8. Employees referred by the committee for treatment through the programme will be secured adequate medical, rehabilitative counselling, food, accomodation, or other services as may be necessary to resolve their problems.

9. The employee will be referred by the supervisor to a designated resource person who is professionally trained to diagnose problem and secure help.

10. Necessary counselling service also will be extended to the members of the family of alcoholic employees.

11. If the employees accepts the offer of help and the job performance or attendance problems improve to satisfactory level, no further action will be taken.

12. If the employee refuse the offer of help and job performance or attendance problems continue, the regular disciplinary procedure will apply.

SELECTION OF TREATMENT CENTRE:

Initially we desired to admit our alcoholic employees in St.Johns Hospital as it was the only hospital with the facility for treatment of alcoholics. But the condition put-forth by them was not acceptable to our organisation.

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COMMITTEE OF CONCERN :

After getting positive response from the Trade Unions, the Management decided to constitute a committee, exclusively for correcting alcoholics and helping other personal Maladjusted employees. The body was named as "the Committee of Concern in Bharat Electronics", with the Chief Administrative Manager as the Chairman, our representatives from departments with higher alcohol proneness, and the Trade Unions.

THE POLICY OF COMMITTEE OF CONCERN IN BEL :

1. The Company recognized that alcoholism is a disease.
2. Those employees of BEL who are affected by this disease will be identified and referred to an appropriate modality of care.
3. SOURCES OF IDENTIFICATION AND REFERRAL:
   a) When an employee's job performance or attendance is unsatisfactory and unable to change by himself with normal supervision will be refered, if the findings proves through him that such of his behaviour only on account of alcoholism.
   b) When an employee is observed by the Trade Unions as problem drinker, and they can also recommend him to be referred for treatment after confirming it through counselling.
   c) Based on the complaints of the dependants of the employee that his spouse/parent addicted to alcohol/drug, such employee/s will be referred for treatment after confirming it through counselling.

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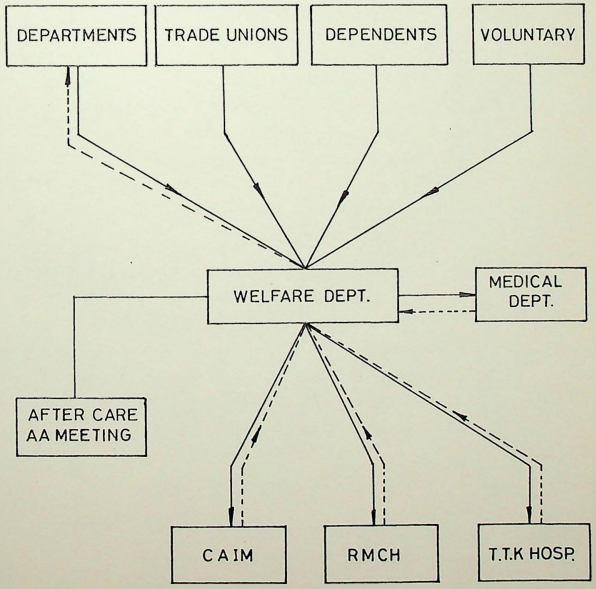
However we selected Varalakshmi Nursing Home, Rajajinagar for detoxification and treatment under psychiatrists for withdrawal panic of the alcoholics. Then after 10 days, admitted them to half-way home, Koramangala, which was run by CAIM.

IDENTIFICATION AND PROCEDURE FOR ADMITTING EMPLOYEES FOR TREATMENT.

As per the policy of the committee of concern, the procedure of identifying, motivation and referring the alcoholic employees is given in the form of diagram -



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Once the Welfare Department receives information either from the respective department or trade unions or dependants about one's poor job performance, chronic absenteeism or chronic alcoholism, the concerned employee is called and interacted by a professional social worker of the Welfare Department or counsellors of the various personnel divisions to identify the reason for such behaviour. If the reason is alcoholism , help him to be aware about the illness of alcoholism and how it ruins the individual and his family. Further he would be explained the programme of Committee of Concern and motivated to free himself from the disease of alcoholism. In case, the employee denies or is reluctant to go for treatment, he would be confronted with necessary documents. The social worker would also meet the dependents of the alcoholic employee and keep them aware of the disease of alcoholism and its effects and persuade them to admit him to the treatment centre. Once the employee/family agrees for Alcoholic treatment, he would be sent for medical examination at our medical department to confirm whether he is fit for alcoholic treatment or not. After confirmation, the employee would be sent to the treatment centre.

The first batch of our alcoholic employees were admitted to Varalakshmi Nursing Home on 5-9-1985 and later taken to half-way home. From 5th September, 1985 to 25th January, 1987, 150 poor job performers and habitual absentees were interviewed and about 90 were identified as alcoholic employees. Out of 90, 67 were sent for alcoholic treatment at half-way home, Koramangala, Bangalore.

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STATEMENT SHOWING THE ADMISSION AND DROP OUTS POSITION

Total no. of estimated poor job performance & habitual absentees	No. of suspected alcoholic employees	No. of employees counselled from 5.9.85 to 25.1.87	No. of employees identified alcoholics	No. of employees sent for treatment	No. of employees were discharged on medical grounds	No. of employees not willing to continue
1200	600	160	90	67	3	14

Over a span of one year and five months, we admitted 67 employees to half-way home. Out of which 50 employees had completed the full course of treatment and three of them were discharged on Medical Grounds. The rest of the fourteen employees discontinued their treatment as they could not cope up with the given situation, as the treatment procedure of the half-way home was more discipline oriented. The severe disciplinary standard led to increase of dropouts. Hence, we felt little difficult to motivate next batches of employees to go to the half-way home for treatment.

An alternative way was located at Ramaiah Medical College Hospital (RMCH), where Psycho-pharmachological therapy was being practised. The whole concept of alcoholism and the treatment of RMCH is quite different from the theory of Alcoholic Anonymous. Over a period of one year we have admitted 48 employees to RMCH for treatment and rehabilitation. At present option is given to alcoholic employees for treatment either at CAIM or T.T.K.Ranganathan Hospital at Madras.

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THE STATEMENT SHOWING THE POSITION OF ADMISSION, DISCHARGE, DROPOUTS, PERIOD OF TREATMENT AND COST OF TREATMENT PER EMPLOYEE:

Name of T/Centre	No. of Employees admitted.	No. of employees completed treatment.	No. of employees discharged on medical grounds	No. of dropouts.	No. of employees undergoing treatment.	Period of treatment in days.	Cost of treatment per employee in Rs.
HALF-WAY HOME	67	50	3	14	--	90	8,200-00
RMCH	48	48	--	--	--	60	6,000-00 (excluding food charges)
CAIM	17	11	1	--	5	90	7,500-00
TTKH MADRAS	5	3	--	--	2	30	3,500-00
Total	137	112	4	14	7	--	--

A third alternative was found in the T.T.K Research Foundation Madras.

EVALUATION OF THE ALCOHOLIC EMPLOYEE REHABILITATION PROGRAMME:

From our experience we concluded that a single agency could not attend to the physical, mental, psychological and moral problems of alcoholics. They required the attention of physician, psychiatrists, psychologists, social worker, and recovered alcoholics. We, therefore, felt that a team of professionals of different disciplines could help the alcoholic employees more effectively rather than single counsellor. Further, after experiencing with the procedure of Half-Way Home treatment, we have introduced two more hospitals (i.e., Ramaiah, Medical College Hospital and T.T.K.Hospital) having the facilities of treating alcoholic patients and kept them open to select the treatment centre at their choice.

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Over a period of 3 years the organisation gained rich experience in the field of helping, supporting and motivating the alcoholic employees. We also sent 10 employees including Personnel Officers, Welfare Officer and employees, Trade Union representatives to TTK hospital to attend a one-week alcoholic counselling course.

As on to-day we have admitted 137 alcoholic employees to various treatment centres. The following table shows the position of admission, recovery and relapse rate of the employees who were admitted to Half-way Home.

No. of persons sent to HWH.	No. of persons discontinued	No. of persons completed treatment	No. of persons left the service after treatment	No. of persons died after treatment	No. of persons sober	No. of persons reduced freq. of drinks	Total Relapse
67	17	50	10	03	25	08	23

The above table shows, out of 50 persons who completed full course of behaviourial training in the Half-way home, 25 (50%) continue to be totally sober for a period of minimum 2 years to 3 years. The other 8 persons have reduced their frequency of drinking. However, even among 17 dropouts, 3 persons have become sober. Apart from this, we could see a lot of qualitative change in their life style. Some of them are engaged spreading the message of the Alcoholic Anonymous and voluntarily assembling every day and sharing their feeling among their Alcoholic Anonymous Group.

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Since many of the alcoholic employees felt that the procedure of Half-way home treatment was very strict and discipline oriented, we found some sort of reluctence from our employees to go for treatment at Half-way home.

RAMAIAH MEDICAL COLLEGE HOSPITAL :

In one year two months, we have admitted 48 persons to RMCH. Here the psychiatrist and the psychologist, would conduct individual counselling, group therapy, relaxation therapy, and aversion therapy. Though it is a general hospital, a ward exclusively meant for treatment and rehabilitation of alcoholics had been set up.

The following table shows the position of admission, rate of recovery and relapse at RMCH :

No. of persons sent	No. of persons completed the treatment	No. of persons left the service	No. of persons become sober	No. of persons reduced drinking	No. of persons totally relapsed
48	48	2	17	8	21

Out of 48 persons, 17 recovered from the disease and continue to be sober for the last one year. It is important to note that none of them discontinued their treatment as the patients are allowed freedom, as in the case of other patients and the concept of their treatment itself does not believe in enforcing or brain washing the alcoholic patients to stop drinking.

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CAIM & TTK HOSPITAL:

Though recently we admitted 22 persons to CAIM & TTK Hospital and 17 of them have completed the treatment from the last two months, we could not assess their sobriety. However, the following tables show the admission and discharge position as on to-day.

Treatment Centre	No. of persons admitted.	No. of persons discontinued.	Number of persons complted treatment.	Number of persons undergoing treatment.
C A I M	17	1	13	3
T T K H	5	-	3	2

PROFILE OF ALCOHOLIC EMPLOYEES:

Alcoholic employees are distributed regardless of age, qualification, nature of work, marital status. The study of 137 alcoholics brings out the following profile. Along with the profile, the number of recovered alcoholics are also shown:

AGE GROUP	AGE GROUP							TOTAL
	20-25	26-30	31-35	36-40	41-45	46-50	51 & above	
NUMBER	-	8	16	37	42	26	8	137
IMPROVED SOBERITY	-	1	5	11	13	9	3	42/86
Percentage	-	12.5%	31.2%	29.7%	30.9%	34.6%	37.5%	48.8%

	EDUCATION						TOTAL
	Illiterate.	Primary.	Middle School	SSLC	SSLC+ ITI	DEGREE	
NUMBER	29	23	35	9	40	-	137
IMPROVED SOBERITY	08	07	14	02	11	-	42/86
Percentage	27.5%	30.4%	40%	22.2%	27.5%	-	48.8%

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Nature of work	Super-visor	Highly skilled	Skill-ed.	Non-Tech-nical	Semi-skilled operator	Un-skilled	Sanitary Helper	Total
	09	34	23	07	11	40	13	137
Improved Soberity	04	10	06	02	04	13	03	42 — 48.8% 86
	44.44%	29.41%	26.09%	28.57%	36.36%	32.5%	23.00%	

Year of service	0-5	6-10	11-15	16-20	21-25	26-30	31-35	36 & above	Total
	4	15	26	58	19	15	-	-	137
Improved Soberity	1	02	09	17	07	06	-	-	42 — 48.8% 86
	25%	40%	34.62%	29.31%	38.84%	40%			

MARITAL STATUS	Married	Un-married	Widower	Broken family	Total
	135	02	01	18	137
Improved Soberity	039	00	00	03	042 — 48.8% 86
	28.89%	-	-	16.67%	

(*) The total sobriety is considered only among 86 persons who completed the full course of treatment at half-way home and R.M.C.H.

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The study reveals the social, environmental and inter and intraconflicts are the contributary factors to alcoholism. Out of 137 persons, 60 come from lower strata of the Society. Most of them are living in bad housing condition. Recovery rate among them is also poor.

Among the group of broken family, the recovery rate is very poor. Even after treatment, 84% of them resume drinking.

AFTER CARE PROGRAMME :

The persons who recovered from the disease of alcoholism themselves conduct Alcoholic Anonymous meeting every day in BEL colony. They themselves meet the members of the family of employees who resume drinking after treatment. We are also getting feed back from the recovered alcoholics regarding progress and relapse cases.

CONCLUSION:

Alcoholism is a complex illness. Yet efforts are being made to evolve a systamatic therapy to cure it totally. As such our organisation has taken up the venture of helping/supporting both alcoholic employees and therapists by hoping for early cure from the illness of alcoholism. Though this venture is expensive in terms of money, its aims and objective to help our employees and their family to accomplish qualitative life, is humane, realistic and un-measurable.

Having high commitment and confidence, our Committee of Concern gave rebirth to many employees. Eventually their family members too got great relief from the world of tension, anxiety and stress etc.

Among the employees who underwent treatment through our scheme, 60% have shown improved sobriety, and good performance of attendance. We do receive the report of good job performance from their respective supervisors.

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Cases of drunkness while on duty, have reduced considerably.

Many employees who were at the early stage of alcoholism have become more alert and cautious and changed their attitude towards drinks. Increasing awareness programme on alcoholism through, pamphlets, brochure leads to falling alcoholic number.

A group of recovered alcoholics have already started spreading the message of alcoholic anonymous and sharing their feeling among themselves through AA meeting. We hope a time may come that the recovered alcoholic employees of BEL themselves will render a remarkable human service to the other alcoholic employees.

Despite 40% of relapse cases, the committee of concern of BEL has determined to march ahead till the eradication of the disease of alcoholism in Bharat Electronics without frustration and disappointment.

MH-2A.47

TTK HOSPITAL
T T RANGANATHAN CLINICAL RESEARCH FOUNDATION
IV MAIN ROAD, INDIRA NAGAR, MADRAS - 600 020.

EMPLOYEE ASSISTANCE PROGRAMME (EAP)

Facts about Employee Assistance Programme:

The first alcoholism assistance programme was started by E.I.Du Pont and Company in 1942. Dr.George Gehnman, the company's Medical Director set up the programme as an alternative to terminating employees with alcohol problem. The supervisors were trained to identify the alcoholics on the basis of certain symptoms like red eyes, trembling hands, frequent intake of cold water and use of breath sweetners. But this method was not very effective since the above mentioned symptoms were indicative of wide variety of illnesses. Further more the principal treatment modality employed to help the employees was through 'Alcoholics Anonymous'.

It was in 1950 that a new approach for providing help to employees with drinking problem was devised by Lewis Persnell. He employed a number of methods of case finding through observations of deteriorating job performance. From the results of these methods it was found that supervisors can only be given the responsibility of evaluating the job performance of an employee and not that of identifying alcoholics. If an employee's job performance has been deteriorating, then the supervisors can offer two choices to the employee - he can accept help on a confidential basis for whatever has been causing a decline, in his work capacity - he can face disciplinary action for his performance.

In 1971, the National Institute of Alcohol Abuse and alcoholism was formed to promote the Employee Assistance Programme in the American Industrial set up.

Employee Assistance Programme is neither pro-company not pro-union, but rather pro-patient. Both Union and Management share equal responsibility in evolving policies and in promoting this programme.

Objectives of the EAP Programme:

1. Early identification of alcoholic employees.
2. Effective motivation to help them accept treatment.
3. Prevention of alcoholism.

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Installing the programme:

a. Policies and procedures.

b. Procedures for case-handling.

c. Referral system

d. Supervisory training.

e. Need for line management involvement in programme implementation.

f. Employee Education component.

g. Record keeping system.

h. Insurance coverage for treatment of alcoholism.

i. Evaluation.

a. Policies and Procedures:

There is an unwritten policy regarding alcoholism in every company. This unwritten policy put in words reads like this. 'Any employee (including executives and members of top management) who can successfully conceal the disease of alcoholism from superiors will be entitled to full use of sickleave and payment of hospitalisation costs and will receive all pay and benefits including promotions and regular raises. When the employee can no longer conceal the disease, termination will result'. This unwritten policy leads to denial and concealment of the problem by all concerned.

A written policy on alcoholism provides a frame of reference for uniform administration throughout the organisation. The programme's acceptance and credibility depends on how well the policy provides clear cut answers to questions which are uppermost in the minds of the affected employees.

Some of the crucial questions that must be answered are:

1. Does the organisation really accept alcoholism as a disease?
2. How is alcoholism defined for programme purposes?
3. Will alcoholism be handled the same as any other disease?
4. Will request for an acceptance of treatment affect job security or promotional opportunities?
5. Will records be kept in strict confidence?
6. Will group health insurance benefits cover treatment for alcoholism?
7. What are the consequences of refusing to accept referrals or failing to respond to treatment?

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8. will there be an opportunity for self-referral on a confidential basis?

9. Is the primary purpose of the programme to encourage employee to seek diagnosis and treatment as early as possible?

b) Procedure for case handling:

Once the policy has been adopted by the management, a simple set of procedures for case handling should be set up. Individuals who seek help voluntarily, will not come under this section. Our concern is with those who will be referred to the programme because of poor job performance. The following are NCA's (National Council on Alcoholism) recommended procedures.

1. The supervisor should prepare a detailed documentation of the employee's job performance deficiencies - specific dates absent or tardy, specific deadlines missed, serious errors in work and incidents of unacceptable behavious.

2. The focus of the initial interview should be on job performance. No opinions or judgements as to the cause should enter the discussion.

3. The employee should have the right to have union representation at the interview.

4. After the job performance has been reviewed and the employee told what sort of standards must be met in the future, the employee should be informed of the professional services - including diagnosis and counselling - which are available on an absolutely confidential basis.

5. At this point, the employee may accept or reject the offer without prejudice. Neither the supervisor nor the union representative need know whether or not the employee has contacted the programme. No disciplinary action is involved.

6. If the employee's job performance improves to an acceptable level, there is no longer a problem.

7. If the initial interview involves discipline or if the job performance problem recur, the employee should be offered a firm, fair choice between accepting the offer of confidential help or accepting the disciplinary consequences of the poor performance.

8. In the overwhelming majority of cases, the

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employee when confronted with this sort of choice will ultimately agree to accept referral to the programme.

9. If the employee chooses termination, there is little that can be done to help.

10. If the employee accepts help, there should be clear understanding that this means following whatever course of treatment is prescribed.

C. Referral System:

Since an employee assistance programme is, in effect, a job performance action programme, those individuals who need professional assistance, regardless of the nature of their problems, will have to be referred to the resources in the community which are best qualified to help them. The treatment resources will range from detoxification facilities to out-patient treatment, including alcoholics Anonymous. Referral to programme is a major responsibility of the supervisors.

d) Supervisory Training:

For the success of any EAP, supervisors at all levels should be trained. Prior to the introduction of EAP, there should be an orientation programme for senior executives and union leaders. Training for supervisors should include reviewing the organisation's policy on alcoholism and explanation of procedures with which the policy will be implemented.

e) Employee Education Component:

An educational component designed to inform employees and management regarding the modern approaches to alcoholism, should be a part of all EAPs. The employees should be educated on the disease concept of alcoholism, and the treatment aspect of the disease emphasising the fact that alcoholism is treatable. Self assessing sings and symptoms of the disease should be made clear to them, as also the organisation's policy regarding the disease.

f) Record Keeping:

A record keeping system which assured confidentiality to the employee and maintains the progress of the programme should be included. Data to be recorded will include the name of the individual, department, various demographic details, education, job description, rate of pay, years of service, marital status and lastly his progress in the programmes.

g) Insurance coverage:

If the organisation's policy states that alcoholism is a disease, and that employees suffering from that diseases are entitled to the same consideration and

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treatment as those who suffer from any other disease, then the establishments group health insurance coverage should reflect that policy.

h) Evaluation.

The employee assistance programme should be periodically reviewed and evaluated. There should be an annual evaluation review of the EAP staff performance.

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The devil in the bottle

Dr Shide
Consultant Psychiatrist

"Candy is dandy but for liquor is quicker."

The irrepressible Ogden Nash said this in a very different context but nothing can summarise lethal effects of consumed substances more pithily. The present article will concern itself only with the "quicker" substance and leave candy aside.

Alcohol in its many consumable forms has been widely used all over the world over millenia. Even some of the most respected medical men have acknowledged its efficacy as a great social lubricant and many cultures set store by it as a symbol of well being, prosperity and hospitality.

And yet the "bottle" is the harbinger of great disasters and misery to an untold number of the world's citizens at any given time.

It is impossible to ascertain how many people consume liquor in a vast country like ours, but at best it could not be less than 40% of the adult males in towns and cities. Even more difficult to establish is the number of people who have a drinking problem, but on any day in a city like Bangalore atleast 3 new alcoholics are being brought for treatment to the medical Specialists! And this does not take into account the large number who have a problem but are yet to acknowledge it.

In India, the economic improvement in many families, the lifting of prohibition in many 'dry' states, the expense account culture, increased promiscuity among the youth and their poorer ability to tolerate frustration would all appear to have increased the magnitude of the problems. Also, increasingly, younger people seem to be becoming more prone to the habit.

Alcohol produces several physiological alterations which cause it to be used repeatedly. These include its effects on the mood, a moderate dosage often allays anxiety, and a slightly strong one causes a sense of euphoria. Alcohol seems to also diminish the inhibitory activity in the central nervous system. This loss of inhibition facilitates the ability to show hitherto suppressed aggression.

When one learns to use alcohol for these reasons, one gradually opens the lock of self control that keeps dependence in check. Most alcoholics indeed are individuals who have at some time been mere 'Social drinkers'. The slide from this state to that of an alcoholic is often so insidious that everyone is taken by alarm when it becomes obvious: the family, peers, even the family doctor and not the least, even the patients themselves!

The prolonged use of alcohol, as of many other abused substances, directly damages different parts of the nervous system and also causes grave injury to the mind of the individual. The effects include a state of trembling and unsteadiness even when the person is not drunk, a marked inability to coordinate the muscles, leading to clumsiness, a deterioration in memory and intelligence and burning sensations or tingling or numbness in many parts of the body. Unrealistic thoughts, especially of being persecuted by colleagues or of the spouse being unfaithful and vivid and disturbing visions and voices that nobody else experiences (hallucinations) can also result from chronic abuse of alcohol.

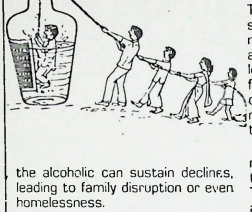
Alcohol adds insult to the brain injury it causes, by impairing the functioning of the liver where most of it is metabolised in the body. In the long run the liver can only totally breakdown if the load of alcohol becomes too much. This leads to the accumulation of substances that can no longer be detoxified, and are potentially poisonous. This causes further brain dysferection and such a doubly insulted brain is often in a fatal condition.

Alcohol has also been implicated in disorders of the gastrointestinal system (gastritis, peptic ulcer). A devastating effect is the fetal alcohol syndrome that results from a pregnant mother's drinking and can cause the child to die or be born with severe handicaps.

Apart from these tangible physical maladies, alcohol drains the individual's sense of self esteem and having earned the label of being an alcoholic he becomes a social derelict, a poorly respected family head or member, an incapable worker or bread winner and a social hazard, e.g., if he is at the steering wheel of a vehicle.

A significant number of road accidents is directly the result of drunken driving. Research also seems to indicate that the more serious the accident, the greater the chance that it has been caused by an inebriated driver. Alcohol seems to impair driving skills by interfering with one's judgement, attention to stimuli from the environment and also motor coordination. In Manchester, bus drivers who had received awards for safedriving were studied in one research project and it was reported that even for these experienced professionals there was no "safe" blood alcohol level below which their judgement could be guaranteed to be sure. Even in the period of the so-called 'hangover', perhaps due to irritability and fatigue, the driver who had a drinking bout, can be prone to cause accident. Equally important is the risk caused by intoxicated pedestrians!

The havoc that liquor can wage in family life is truly devastating. An obvious effect is the drain it makes on the family budget, and the well known fact that it takes more and more liquor to get a person drunk as his habit progresses ('tolerance') aggravates the problem. Debts tend to accumulate rather than be paid of. The quality of accomodation which



the alcoholic can sustain declines, leading to family disruption or even homelessness.

Sexual dysfunction is almost invariably a long term outcome of chronic alcohol abuse. This is ironic in the face of the popular notion that 'drinks' are aphrodisiacs (Sex stimulants). Perhaps there is some grain of truth underlying this notion, in that, alcohol being an inhibitor of inhibitions, helps remove some inhibitions even about sex; but even in people who use liquor frequently without being dependent on it, there is unequivocal evidence that alcohol causes a somewhat rapid degradation of hormones responsible for healthy sexual functioning leading to impotence. This is one important factor that contributes to the marital breakdown so common among alcoholics.

Even in the absence of such a sexual problem alcohol causes disturbed marital relations. In England and Wales, alcohol abuse contributes to as many as one third of divorces! The children of alcoholics are far more vulnerable to emotional and/or physical abuse and also to developing behavioral problems themselves. There is no doubt that in India as in many Western countries, alcohol has become a major scourge among the poor families already caught in the vice-grip of penury.

While drinking problems are extremely common among criminals an aspect of this phenomenon that is too often ignored is that crime can often be the *consequence* of this habit gripping a hitherto law abiding citizen. This is of course true for all forms of addictions.

Getting an alcoholic treated, is therefore of paramount importance. This is easier said than done, not the smallest reason being that very many alcoholics are unwilling to acknowledge that they have a problem at all! Even those that do, often fantasize that in some way they can control the problem but have chosen not to because they dont "yet" need to.

The treatment of alcoholism needs to pay sufficient attention to the physical and emotional aspects. This is best achieved by admission to a hospital/faculty geared for de-addiction and with the aid of selfhelp groups that can be run by reformed addicts under the surveillance of qualified personnel. Involving the family, the peers or employers when warranted, becomes important from the point of view of rehabilitation, because these individuals can be understandably embittered, sceptical and even unforgiving. Many alcoholics benefit from the use of a drug that by itself is largely harmless and non-addicting but in combination with liquor is a potential poison. Such a drug, (disulfiram) however, must always be administered under medical supervision with the patient's foreknowledge of the potential hazards. In some de-addiction units patients are made to experience the danger of the combination under a doctors' supervision, making sure that no life threatening situation arises. Some patients are exposed to these adverse reactions in another patient in the ward or to a video film demonstrating the same. The general principle is to form an aversion in the patients to allowing a very unpleasant situation arising from the consumption of liquor.

Apart from such chemical aversion, other forms of aversion to drinking can also be administered. These are procedures administered over a course of time by trained clinical psychologists. Many de-addiction units lay emphasis on group therapy wherein freshly admitted patients can gain confidence from alcoholics who have progressed through the difficult initial phase of being weaned from the bottle.

The ideal management of addiction of course is in its prevention, which means effectively restricting the availability of alcohol and educating the public, especially the vulnerable groups such as students on the ill effects of the drinking habit. For persons who have sustained physical dependence, the best advice is lifetime group such as Alcoholics Anonymous and encouragement to resume a creative life are aspects of treatment that can never be overemphasized.

Partly Precise:

Psychiatrist: *A person who deals with people who have the same problems we all have, but have more money.*

Commuter: *A man who shaves and takes a train, and then rides back to shave again.*

Conscience: *The thing that hurts when everything else feels so good!*

Contented: *A man who enjoys the scenery on a detour.*

Patience: *A minor form of despair disguised as virtue.*

Happiness: *The interval between periods of unhappiness.*

Puzzled

A magician working on a cruise ship had a pet parrot who was ruining his act. The bird would say to the audience, "He has the card in his pocket," or "The card's up his sleeve," or "It went through a hole in his top hat."

One day there was an explosion and the ship sank. The parrot and the magician found themselves together on a piece of wreckage. The parrot stared at the magician. Finally, the parrot said, "Okay, I give up. What did you do with the ship?"

Alcohol abuse among the women folk of Chakkiliyar community in the Tirunelveli district of Tamil Nadu

Prof AJA Ranjitsingh
Dr Padmalatha Ranjitsingh
Prof LM Narayanan

Alcoholism is the third largest killer disease of the world, next to heart attack and cancer. Such a dreadful disease is found to have affected a number of rural women belonging to Chakkiliyar caste, one of the schedule caste communities (Adidravida) of the Tirunelveli District of Tamil Nadu. Alcoholism is being fostered by this community as a social custom. Husbands bringing the inebriating drink to their spouses, mothers filling the feeding bottle with toddy and children drinking alcohol in the company of their parents is quite common in this community. On occasions like festivals, funeral functions etc., the entire family along with their kith and kin intoxicate themselves with alcoholic beverages. Moreover, drinking habit is traditional in this caste. Ontogeny of alcoholism in this caste is traced back to their traditional occupation of collecting dirt, garbage, faeces and wastes. *Once they drank to earn, but now they earn to drink.*

Because of the alcoholic 'infection' the entire Chakkiliyar Community could not make any progress in life. Socio-economic backwardness still prevails in them. Hence, an attempt has been made to probe into the various facts behind this contagion in this community. For the study, three villages viz., Reddiarpatty, Kadanganeri and Uthumalai of Tirunelveli District of Tamil Nadu were chosen, where more than 750 families of Chakkiliyars are living. From these villages a random selection was made comprising 200 females of Chikkiliyar caste in the age group of 15 to 65 years. Details of their alcoholic habits and work pattern were collected using a questionnaire and the data analysed on the basis of the information received. This data were collected before the promulgation of prohibition in Tamil Nadu.

Socio-economic status of the Chakkilyar community.

Chakkiliyars are one of the scheduled caste communities of Tamil Nadu. Their population exceeds 5 lakhs in the Tirunelveli District of Tamil Nadu. They are traditionally cobblers and scavengers. They speak a scriptless language which is a mixture of Telugu and Kannada. They are believed to have originated from Naikkar community. They speak Tamil fluently. Certain menial jobs viz., lavoratory cleaning, drainage cleaning, sweeping, garbage collection are exclusively done by the men and women of this caste in the human habitat. In many places they have to carry human excreta in a bucket as a part of latrine cleaning. Such undesirable jobs thrust on them in the name of social custom, has resulted in branding them "untouchables". They are also called THOTTIS or SCAVENGERS. In addition to performing this menial work, they are good agricultural labourers and beedi rollers. Each high caste family has atleast one Chakkiliyar family as their labourer to do menial jobs in their houses.

The socio-economic status of this society is very poor. Their chief source of income is from their traditional occupation and agricultural labour. Now-a-days the women have learnt to earn money by rolling beedies. More than 90% of the women folk of this community discharge one or more than one of these duties. Their average family size is 5—8. Except 9.1% of the families, all the other members earn below Rs. 1200/- month. About 40.3% of families earn Rs. 500—1000/- month. 50.6 percent families earn less than Rs. 500/- month. The income is not steady and it is high during paddy farming and harvesting seasons. Only 4% of the families could live in comfortable houses, while the others dwell in huts, small tiled houses or rented houses. The average age of marriage in women is 16.7 years. In this caste, women change their husband quite easily. Divorce is common in this caste. A woman without much hesitation or fear, joins another man who is already married and has his own children. The new man accepts her with her children without any hesitancy. Once a husband humourously said to his wife, "My children and your children are playing with our children". This saying is popular among the villagers even now. Extramarital relationship with their landlords and others is quite common in this caste. Pre-marital sex was found in 76.1% of the girls in this caste. More than 80% of the women of this caste were illiterates. The maximum education they had received was till VIII Std. Their working environment is exclusively dirty, filled with foul smell and it is extremely unbearable.

In spite of all these adverse socio-economic conditions, alcoholic beverages are consumed to a great extent, perhaps to forget their occupational hardships. 79.5% of the women of this community were ▶

found to be alcohol users. But the percentage was only 4.6% among the women in the other communities of the study area.

Drinking Pattern

Drinking pattern among the womenfolk of this community has been determined by categorising them into three age groups viz., 15—25 years, 25—45 years and 45—65 years. (Fig.1) In the age group 15—25 years, 63.1% of them were alcohol users. Among them 51.1% were heavy drinkers. In the age group 25—45 years, 90.8% were alcohol consumers. Of them 12.95 were light drinkers. 9.4% among them consumed alcohol more than once in a day. In the age group of 45—65 years 83.3% women were drinkers. Among them, the ratio of the light drinkers and heavy drinkers was 15.5% : 68.7%.

From the results it is clear that womenfolk in all the age-groups of this community freely used alcohol. The intensity of drinking is little less in the age group of 15—25 years and it is perhaps because of their initial hesitation in drinking liquor.

Drinking pattern varies with income. In the months of December — February, that is during the harvesting period, the women earn more money in the form of paddy and spend more than 33.6% of their earnings on drinking. During that season about 60 percent of women in all the age-groups consume alcohol once or more than once in a day. During week ends, the women beedi rollers get their salary and they freely spend a part of their income on alcoholic drinks.

Also, on occasions like festivals, funeral functions and other celebrations there is heavy consumption of liquor. During funeral functions it has become customary in this community to supply liquor to all the mourners. Hence, the entire community seems to encourage this habit.

In the study area, about 60% of these people usually take drinks in the evening. Other use alcohol at any time they like. 28% use alcohol while working. 20% consume alcohol when they indulge in extramarital affairs with each other, so as to maximise their enthusiasm.

Fig. 1 Drinking pattern of Alcohol in the different age groups of Chakkiliyar community (percentage)

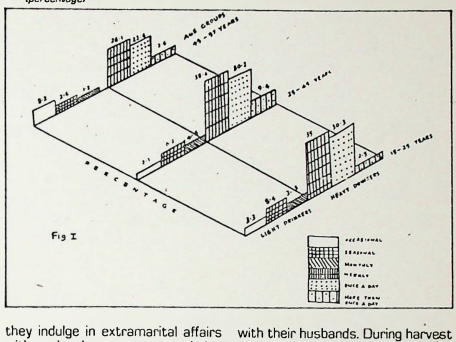


Fig I

The alcoholic drink popular among them is toddy. It is a palmyra product. About 80% of the women drinkers use this liquor. 69.6% women use arrack in addition to toddy. Only 16% of them use other varieties of IMFL (Indian Made Foreign Liquor) alcoholic beverages.

Alcohol is a family drink:

Women drink only with the knowledge of their husbands, parents and children. It is a family-accepted habit. 56.5% of husbands bring liquor for their spouses. 29.5% of women get the drink with the help of their family members. 14.5% of women even visit the liquor shops either alone or with their husbands. During harvest season 80% of the women directly get alcoholic drinks from toddy sellers in the field itself. 79.5% women who consume alcohol have extra-marital relationships either with their land-lords or with others. Extra-marital sexual relationship is even tolerated by some of their husbands. Thus alcoholism becomes a family-disease.

Ontogeny of Alcohol abuse:

Alcoholic usage has crept into the society of Chakkiliyars in the beginning as an occupational drink. To forget their hardship in the filthy working environment, they started drinking. under intoxication, they believe that they can discharge their menial labour more effectively. Also, after finishing their work they want to relax with a peg of alcoholic drinks to get relief from the tension. The people of this community used to eat the beef of the diseased and dead cattle which their landlords offer them freely. On such occasions every Chakkiliyar family in a village gets a free supply of beef. They consume the beef even though it is in a rotton condition. On such occasions also, they use alcoholic drinks. During harvest season their land-lords supply them free toddy. From drinking toddy slowly the people have ▶

Easy

A woman lion tamer had the cats under such control they took a lump of sugar from her lips on command. When a skeptic yelled "Anyone can do that!" the ringmaster came over and asked him, "Would you like to try it?"

"Certainly," said the man. "But first get those crazy lions out of there."

fallen into the hands of more toxic arrack and other liquor. Once they drank to work but now it has reached the stage of working to drink. Today non-menial servants are also habituated to alcoholic drinks. With the patronage of the elders of the community, alcoholism, has gradually affected every one of the members of this community including the women. Illiteracy is another factor to consider when dealing with alcohol abuse. Immoral sexual contact has also encouraged the continuation of alcoholic habit in 16% of the women studied. Lack of willingness to reserve their income for better living encourages them to waste a portion of their income on drinking liquor. Among the alcohol using women 60% use snuff which is deposited on teeth and 90% are betel chewers. The prevalence of other unhealthy habits also indicates their negligence with regard to their health. It is no doubt that alcoholism will become an uncontrollable disease among the women folk of this community in the days to come. It is the right time for the Government agencies and voluntary organisations to act and to rehabilitate the women folk of this Chakkiliyar community.

Checking out whether your drinking is a problem

— Do you feel the need to have a drink everytime you are exhausted, tense, depressed or lonely?
— Do you drink alone or early in the morning or more than thrice a week?
— Are you consuming more than 3 "larges" on any single occasion?
— Under the effect of liquor do you tend to become irritable, garrulous, silly, abusive or violent?
— Does stopping drinking cause you to become anxious/morose or have trembling hands/blackouts/unsteadiness/sleeplessness/lack of appetite?
— Has there been a parent/brother/sister/son or daughter who had a serious drinking problem?

If the answer is yes to any one of the these, please check with a doctor about your drinking habit.

HEART 1
Unstable Blood pressure, Irregular Pulse, Enlarged Heart

PANCREAS 2
Painful inflammation

LIVER 3
Severe swelling, Hepatitis Cirrhosis

MUSCLES 4
Weakness, Loss of muscle tissue

STOMACH 5
INTESTINES
Lining becomes inflammed, Ulcers

NERVOUS SYSTEM 6
Tingling and loss of sensation in hands and feet

BRAIN 7
Cell damage resulting in loss of memory, confusion, hallucinations

LUNGS 8
Greater chance of infections including T B

GENITALS 9
Temporary impotence

SKIN 10
Flushing, sweating, bruising

BLOOD 11
Changes in Red blood cells

— from Alcohol and Alcoholism. TT Ranganathan Clinical Research Foundation, Madras.

The new lotus eaters

The classics refer to a band of soldiers under Ulysses who lost themselves to the problems of the world by eating lotus stems which caused them to reach rapidly a state of bliss that was reinforced with fresh doses of the repast.

Young people today are alarmingly taking to chemical means of overcoming the problems in life, thus becoming the new lotus eaters. While experimental abuse of drugs is common among youth everywhere (more than 50% in urban Indian boys) if this occurs on the background of an unstable personality, a large number of stresses in the individual's life at that time or in the face of an emotional disturbance, a likelihood of *drug dependence* developing is considerably increased.

Not all Drug Addicts, however, come from disrupted or unhappy families — sometimes a drug problem in a previously healthy person can cause the family to become disturbed.

Not pattern of drug abuse frequently observed among youth is that initially legal drugs (such as tranquillisers) are abused, then alcohol and/or tobacco, progressing to cannabis or marijuana and finally to illicit substances like heroin.

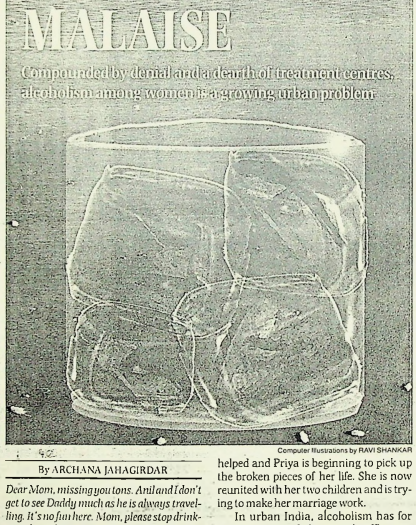
Peer pressure or persuasion by friends (the so called 'bad company') is not the most important factor in making a person an addict though some ruthless antisocial elements undoubtedly make a bad situation worse by taking advantage of an individual's weakness.

After the malady develops the best outcome arises in children of parents who do not allow their alarm over the habit to get the better of them, do not submit to their children's demands for more drugs/money and yet keep channels of communication open. Parents who delay their children's treatment or are highly secretive about the habit, often do their children harm — which *can* prove lethal.

WOMEN ALCOHOLICS

A HIDDEN MALAISE

Compounded by denial and a dearth of treatment centres, alcoholism among women is a growing urban problem



Computer illustrations by RAVI SHANKAR

By ARCHANA JAHAGIRDAR

Dear Mom, missing you tons. Anil and I don't get to see Daddy much as he is always travelling. It's no fun here. Mom, please stop drinking. If you do that, we will all be able to live together again. It will be so much fun. I hate school here. Write soon. Love you lots, Anjali.

MORE than anything else, this letter from her 11-year-old daughter put Priya Singh, a Delhi housewife, back on the road to sobriety. "That was my rock-bottom. I decided that I had to do something about my alcoholism." But after drinking heavily for almost two years—something that pushed her marriage to the brink—she knew getting back to normal wasn't going to be easy. She visited a rehabilitation centre, and after several relapses finally succeeded in giving up alcohol altogether. Frequent attendance at the Alcoholics Anonymous (AA) meetings helped and Priya is beginning to pick up the broken pieces of her life. She is now reunited with her two children and is trying to make her marriage work.

In urban India, alcoholism has for long been regarded as a male affliction. Not any more, as psychiatrists in the country's metropolises report. What began as a trickle of female patients coming in for treatment is now a widening stream. An estimated three out of 10 alcoholics in many established clinics across the country are now women.

Unfortunately, only now is the problem being taken seriously. At the National Institute of Mental Health and Neurosciences (NIMHANS) in Bangalore, the country's premier neurosciences research institute, around 10 female patients used to report for treatment every month just five years ago. Now, there are 30 to 35 every month and the institute has been forced to open a separate outpatient department for women alcoholics. Explains Dr I.A. Shariff, head of the Department of Psychiatric Social Welfare at NIMHANS: "Nobody speaks or makes public this problem because, in the Indian situation, for a woman to be alcoholic is considered worse than being a prostitute or a criminal. There is social ostracism. So, the way most women cope with it is by hiding their problem."

In Bombay, as the problem escalated, the Kripa Foundation, which has seven centres across the country for treating alcoholics and drug addicts, had to open a separate section for women. Part of the reason, according to Ossie Pereira, co-founder of the foundation, was that "most alcoholics, men and women, haven't been able to handle sex normally. So if I had a mixed centre, I would have to start a maternity ward instead." And Dr D. Mohan, head of the Department of Psychiatry, All India Institute of Medical Sciences (AIIMS), predicts that by the year 2000, overall India is likely to see one female alcoholic for every three male alcoholics—a ratio prevalent in the West today.

So, why exactly are women in India hitting the bottle in such alarming numbers? There is no clear answer. Research worldwide points a finger at a gene which could make alcoholism hereditary. Others talk of a certain percentage of people who are exposed to alcohol automatically becoming chemically dependent on it. For instance, the WHO estimates that one out of 25 people who drink will turn alcoholic as a result.

Part of the reason for the dramatic rise in women's dependency on alcohol is simple—they are now more exposed to alcohol socially than at any time in the recent past. It is no longer taboo for women to drink in public, and in parties it is the done thing. Confirms Rakesh Ghildiyal, associate professor of psychiatry at KEM, Bombay: "Alcohol is a bigger problem among women than any other drug because it now has social sanction."

Others, like Delhi-based psychiatrist Dr Avdesh Sharma, feel that it could be modern-day problems which are driving women to alcoholism. The most cited reason is stress. The balancing act required to keep a home and a career going has been known to take its toll. Conversely, a workaholic husband could lead to a depressed and lonely wife at home. Rachna Sharma, 38, would agree. Three grown-up children meant more time in her hands and a loving but hopelessly busy husband had no time to fill that. The bottle provided the initial solace and then the addiction took over.

If loneliness is creeping stealthily into

Nowadays, three out of 10 alcoholics reporting for treatment in established clinics in

Indian lives as joint families—and even nuclear ones—break up, it is compounded by an increasing identity crisis faced by urbanised Indians. Are we as cool and hip as our western counterparts? Hot-shot marketing executive Sonia Malik, 40, wanted to be all that and more. "I started drinking to deal with stress and to be in with the crowd." The only problem was that her drinking continued well after alcoholic faces are very different from what a man goes through—this needs to be addressed. Her drinking is even more hidden than his, thus making it all the more difficult to detect. Says Sonia: "I used to stay late in the office and drink secretly, but I was always very controlled in public—a woman has to be. I would fall apart the minute I reached home."

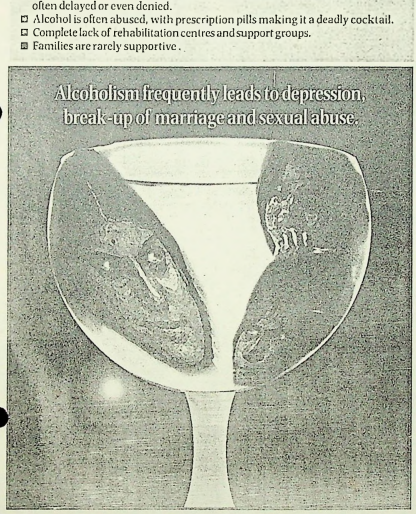
Home, unfortunately, is not where everyone went home. And often, just as others were beginning their day—a pattern common among alcoholics.

Denial and lack of empirical data have led to the problem turning even more serious. Says Ashwini Adawadi, an addiction counsellor in Delhi: "We have this neat policy of denial. Indian women do not have sex, they don't have orgasms and they don't drink. We simply solve the problem by denial." The issues a woman the support system is anymore. Husbands could be alcoholics or, at the very least, the initiating party. At the other end of the scale could be an alcoholic parent hovering in the background. Rahul Khanna, 40, became the target of his wife's criticism about five years ago. Her complaint? That he drank too much. In what she thought was an attempt at getting closer to him, Deepti, 35, too started drinking. "We began to share a few drinks together and things began to look more cheerful," she recalls. The cheer proved as short-lived as the fizz in a glass of beer. A truncated career and several violent fights later, the Khannas realised the severity of the problem.

Career and family life are not the only things at risk. Studies conducted in the US show that spontaneous abortions and early miscarriages are more likely to occur among alcoholic women. Pregnant women in their first trimester are at a risk factor of 1.2 per cent even if they are modest drinkers. And the risk of congenital abnormalities is estimated to be as high as 32 per cent for women who are heavy drinkers. Worse, children are found to be more seriously affected by alcoholic mothers than fathers.

WHY TREATMENT IS TOUGH

- Indian women are usually closet drinkers, so identification and treatment is often delayed or even denied.
- Alcohol is often abused, with prescription pills making it a deadly cocktail.
- Complete lack of rehabilitation centres and support groups.
- Families are rarely supportive.



THE problem also persists because there are no stereotypes, no target groups. And unfortunately, no safe groups either. At one end of the spectrum are groups like tribals and plantation workers, from whom come 48 per cent of women alcohol users. Women in the weaker economic strata have always consumed alcohol. Now, there is the new addition of the middle classes, which could include anyone from a college student to a bored housewife. Says Karl Sequeria, joint director of the Bangalore-based Freedom Foundation, a rehabilitation centre: "I find that more and more young college-going women are drinking heavily, a cause for grave concern."

So, while recognition is often myopic, treatment runs into a blind alley. Women face specific guilt trips and problems but rehabilitation centres and support groups are dominated by, and are often for, men. Rape, manipulation and sexual baiting are all part of a woman alcoholic's routine. With such a negative gender background, a woman with this problem finds it difficult to enter a predominantly male group. Even an immensely successful support group like AA faces this problem. There is now a crying need for separate rehabilitation centres for women, like the one SNEHAN has begun.

The silver lining to this dark cloud is that sobriety gives recovering women alcoholics an unparalleled high. Every experience is savoured with a new enthusiasm and talk of a new life. Sonia, who was considered dead for all practical and financial reasons, is today at the centre of all family decisions. Priya is the emotional anchor for her two teenage children. Most of them further the cause of sobriety by keeping their doors open to fellow alcoholics. —with ARUN KATHYAR in Bombay and SARITHA RAI in Bangalore

(Names of some people have been changed to protect their identities.)

Indian cities are women.

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ಆಲ್ಕೋಹಾಲಿಸಮ್

● 'ಪರಂಜ್ಯೋತಿ'

ಕುಡಿತದ ಚಟುವಿಂದ ತಮ್ಮ ಹಣ, ಆಸ್ತಿ, ಉದ್ಯೋಗ, ಗೌರವದ ಜಾಡಿಗೆ ತಮ್ಮ ದೈಹಿಕ ಹಾಗೂ ಮಾನಸಿಕ ಸ್ವಾಸ್ಥ್ಯಂ ಕೆಳೆದುಕೊಂಡು, ತಮ್ಮ ಕುಟುಂಬದವರನ್ನು ಕಷ್ಟಗಳಿಗೆ, ಅವಮಾನಕ್ಕೆ ಗುರಿಮಾಡಿ, ಸಮಾಜಕ್ಕೆ ಭಾರವಾಗಿ ಅಪಮಾನ ಜೀವನ ನಡೆಸುತ್ತಿರುವವರ ಬಗ್ಗೆ ಅಥವಾ ಈ ಚಟದಿಂದ ಬಿಡುಗಡೆ ಪಡೆಯಲು ಸಾಧ್ಯವಾಗದೆ ಸತ್ಪಪಡಲು ಬಗ್ಗೆ ನೀವು ಕೇಳಿರಬಹುದು ಅಥವಾ ಅಂಥವರನ್ನು ಕಣ್ಣಾರೆ ಕಂಡಿರ ಬಹುದು. 'ಆಲ್ಕೋಹಾಲಿಸಮ್' ಎನ್ನಲಾಗುವ ಈ ವ್ಯಾಧಿ ಪೀಡಿತ ವ್ಯಕ್ತಿ ನಿಮ್ಮ ಕುಟುಂಬದವರೇ ಆಗಿದ್ದಲ್ಲಿ ಒಬ್ಬನಾಗಿದ್ದರೆ ಈ ವ್ಯಾಧಿಯ ತೀವ್ರತೆಯ ಅಳವಿನ ನಿಮಗೂ ಅರಿವಾಗಿರುತ್ತದೆ.

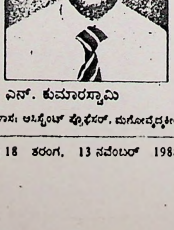
ಆಲ್ಕೋಹಾಲಿಸಮ್ ವ್ಯಾಧಿಯಾದರೂ, ಯಾವುದೇ ಘಟ್ಟದಲ್ಲಿ ಅದನ್ನು ಗುಣಪಡಿಸಬಹುದು–ಎಂಬ ಆಧಾರದ ನೆಲೆಗೆ ಕೆಲಸ ಮಾಡುತ್ತಿರುವ ಸಂಸ್ಥೆಯೇ ಮದಂಗಾಪ ಜಪ್ಪೆಮಾರಿನಲ್ಲಿರುವ ಟಿ. ಟಿ. ರಂಗನಾಥನ್ ಫ್ರಿಕಲ್ ರಿಸರ್ಚ್ ಸೆಂಟರ್‌ಜಪ್ಪೆ.

ಹೊಸ ವೈಜ್ಞಾನಿಕ ಸಮೀಪನ

ಆಲ್ಕೋಹಾಲಿಸಮ್‌ಗೆ ಚಿಕಿತ್ಸೆ ನೀಡುವವರಲ್ಲಿ ಇದು ಭಾರತದ ಏಕೈಕ ಬಗ್ಗಗಾಮಿ ಸಂಸ್ಥೆ. ವ್ಯಾಧಿಗೆ ವೈಜ್ಞಾನಿಕ ರೀತಿಯಲ್ಲಿ ಚಿಕಿತ್ಸೆ ನೀಡುವ ಮೊಲ ಅದಷ್ಟು ಗುಣವಡಿಸ ಬಹುದು ಎಂಬುದನ್ನು ಸಾಧಿಸಿ ತೋರಿಸುತ್ತದೆ.

1980ರಲ್ಲಿ ಪ್ರಾರಂಭವಾದ ಈ ಸಂಸ್ಥೆ ವ್ಯಾಧಿ ಪೀಡಿತರಿಗೆ ಮಾನಸಿಕ ಚಿಕಿತ್ಸೆ ನೀಡಬಹುತ್ತಿದ್ದು ಹಾಗೆ ಮೊದಲಿಗೆ ಚಿಕಿತ್ಸೆ ಪಡದವರು ಬರಿಯ ನಾಲ್ಕು ಮಂದಿ. ಮುಂದೆ ವೈದ್ಯಕೀಯ ಚಿಕಿತ್ಸೆ, ವೃಂದ ಚಿಕಿತ್ಸೆ, ವ್ಯಾಧಿ ಪೀಡಿತನ ಕುಟುಂಬದವರಿಗೆ ತರಬೇತಿ ಹೀಗೆ ಅದರ ಕಾರ್ಯವ್ಯಾಪ್ತಿ ವಿಶ್ವರಿಸಿತು. ಮಾದಕ ವಸ್ತುಗಳ ಚಟಕ್ಕೆ ಬಲ್ಪವರಿಗೂ ಈ ಸಂಸ್ಥೆ ಚಿಕಿತ್ಸೆ ನೀಡತೊಡಗಿತು.

ಶಾಂತಿ ರಂಗನಾಥನ್ ಈ ಸಂಸ್ಥೆಯ ಪ್ರೇರಕ ಶಕ್ತಿ ಹಾಗೂ ಜಾಲಕ ಶಕ್ತಿ. ಕೆಂದ್ರದ ಮಾಜಿ ಹಣಾಪು ಮಂತ್ರಿ ಟಿ. ಟಿ. ಎನ್. ಕುಮಾರಸ್ವಾಮಿಯವರು ಮನಶ್ಶಾಸ್ತ್ರದಲ್ಲಿ ಎಂ.ಎ. ಪದವೀಧರರೂ, ಚಿಕಿತ್ಸಾ ಮನಶ್ಶಾಸ್ತ್ರದಲ್ಲಿ ಎಂ.ಫಿಲ್ ಪದವಿಯನ್ನೂ ಪಡೆದಿದ್ದಾರೆ. ಪ್ರಸ್ತುತ ಕಸ್ತೂರ್‌ಬಾ ಆಸ್ಪತ್ರೆಯ ಮನೋವೈದ್ಯಕೀಯ ವಿಭಾಗದಲ್ಲಿ ಮನಶ್ಶಾಸ್ತ್ರಜ್ಞರಾಗಿ ಸುಮಾರು 10 ವರ್ಷ ಗಳಿಂದ ಸೆವೆ ಸಲ್ಲಿಸುತ್ತಿದ್ದಾರೆ. ವಿಶೇಷವಾಗಿ ಮದ್ಯವ್ಯಸನ ಮತ್ತು ಮಾದಕ ದ್ರವ್ಯ ಸೇವನೆಯ ಬಗ್ಗೆ ಮಂದಾಸ, ವೆಲ್ಲೂರುಗಳಲ್ಲಿ ನಡೆದ ಸಮ್ಮೇಳನ ಮತ್ತು ಕಾರ್ಯಾಗಾರ ಗಳಲ್ಲಿ ಭಾಗವಹಿಸಿ ಅನುಭವವನ್ನು ಪ್ರಯಾಸರು ಹಲವಾರು ವ್ಯಸನಿಗಳಿಗೆ ಸೂಕ್ತ ಸಲಹೆ ಮಾರ್ಗ ದರ್ಶನ ನೀಡುತ್ತಿದ್ದಾರೆ.



● ಎನ್. ಕುಮಾರಸ್ವಾಮಿ

ವಿದ್ಯಾರ್ಥಿಗಳ ಮಾನಸಿಕ ಸ್ವಾಸ್ಥ್ಯದ ಬಗ್ಗೆ ಸಲಹೆ ಮತ್ತು ಮಾರ್ಗದರ್ಶನ ನೀಡುವಲ್ಲಿ ವಿಶೇಷ ಆಸ್ಥೆ ಮತ್ತು ಅಭಿರುಚಿ ಇದೆ. ●

ಕುಡಿತದ ಚಟ ವಂಶ ಪಾರಂಪರ್ಯವೇ?

ಕೆಲ್ಲು ಗುಣ ಯಾವುದೇ ಇರಲಿ.ಅದು ಹುಟ್ಟಿನಿಂದಲೇ ಬಂದದ್ದೇ. ಇಲ್ಲಾ ಪರಿಸರದಲ್ಲಿದ್ದದ್ದನ್ನೆ ಕಂಡು ಕಲಿತದ್ದೇ. ಎಂಬ ವಿವಾದ ಯಾವಾಗಲೂ ಇದ್ದದ್ದೇ. ಮದ್ಯವ್ಯಸನವೆನು ಇದಕ್ಕೆ ಹೊರತಾಗಿಲ್ಲ. ವ್ಯಸನಿಗಳಲ್ಲಿ ಬಹುಮಂದಿ ವ್ಯಸನಿಗಳ ಮಕ್ಕಳು ಎಂಬ ನಂಡಿಯೂ ಕೆಲವು ಅಧ್ಯಯನಗಳಿಂದ ತಿಳಿಯ ಬಂದಿದೆ. ಹಾಗಾದರೆ ಕುಡಿತ ಚಟಕ್ಕೆ ಬಲ್ಪವರ ಪಕ್ಕಲ್ಲೂ ಮಕ್ಕಳೂ ವ್ಯಸನಿಗಳಾಗುತ್ತಾರೆಯೇ ? ಈ ಪ್ರಶ್ನೆಗೆ ಖಚಿತ ಉತ್ತರ ಹೇಳುವುದು ಕಷ್ಟ. ಕುಡುಕ ಮಕ್ಕಳು ಅವರಲ್ಲೂ ಗಂಡು ಮಕ್ಕಳು. ಕುದಿಯುವುದನ್ನು ಕಲಿತರೆ ವ್ಯಸನಗಳಲ್ಲಾದರೆವರೆ ಮಕ್ಕಳಿಂತ ಬಹು ಬೇಗನೆ ವ್ಯಸನಗಳಾಗುತ್ತಾರಂತೆ. ಇದಕ್ಕೆ ಸ್ಪಲ್ಪ ಮಟ್ಟಿಗೆ ಅವರ ದೇಹದಲ್ಲಿರುವ ಆನುವಂಶೀಯ ಗುಣಗಳು ಕಾರಣ ವಾಗಿಬಹುದು. ಮದ್ಯವ್ಯಸನಿಗಳ ಮಕ್ಕಳ ದೇಹ ವ್ಯಸನಗಳಲ್ಲಾದವರ ಮಕ್ಕಳಿಂತ ಹಿಚ್ಚಿನ ಆಲ್ಕೋಹಾಲ್ ಪ್ರಮಾಣವನ್ನು ಸಹಿಸ ಬಲ್ಲುದಂತೆ. ಎರಡೂ ಬಗೆಯ ಜನರ ರಕ್ತದಲ್ಲೂ ಒಂದೇ ಪ್ರಮಾಣದ ಆಲ್ಕೋಹಾಲ್ ಇದ್ದಾಗ್ಯೂ ವ್ಯಸನಗಳಲ್ಲದವರ ಮಕ್ಕಳಿಗೆ ಬೇಗನೆ ಮತ್ತೇರುವುದನ್ನೂ ಕೆಲವು ಅಧ್ಯಯನಗಳು ಕಂಡು ಕೊಂಡಿವೆ. ಈ ಸಾದೃಶ್ಯ ನಿರಾವಾರಿಯೂ ಆನು ವಂಶೀಯವಾಗಿಯೇ ಬಂದದ್ದೇ ಎಂಬ ಬಗ್ಗೆ ಭಿನ್ನಾ ಭಿನ್ನಾಯವೂ ಇದೆ. ಅಡಕ್ಕಾಗಿ ಇಲಿ, ಮೊಲಗಳಿಗೆ ಮದ್ಯ ಕುಡಿಸಿ ಸಂತೋಧನೆಗಳನ್ನೂ ನಡಸಲಾಗುತ್ತಿದೆ. ಆದರೂ ಮೇಲಿನ ಪ್ರಶ್ನೆಗೆ ಖಚಿತವಾಗಿ ಯಾವ ಉತ್ತರವೂ ಸಿಕ್ಕಿಲ್ಲ. ವ್ಯಸನಗಳಲ್ಲದ ಮಕ್ಕಳೂ ಕುಡಿತ ಕಲಿಯಬೇಕೇ ಇದ್ದರೆ ವ್ಯಸನಗಳಾಗಿಬಿಡಬೇಕೇ ಇಲ್ಲ. ಆದರೆ ಇಂತಹ ತಂದೆ ಯಾ ತಾಯಿ ಮನೆಯಲ್ಲಿರುವಾಗ ಅವರನ್ನೇ ಆದರ್ಷವಾಗಿಟ್ಟುಕೊಂಡು ಬೆಳೆಯುವ ಮಕ್ಕಳು ಆವಕಾಶ ಸಿಕ್ಕರೆ ಕುಡಿಯದೆ ಬಿಟ್ಟಾರೆಯೇ ? ವ್ಯಸನಿಗಳಾಗಲೇ ಉಳಿದಾರೆ ! ಆಂತಹವರು ಹೀಗೆ ಆನುವಂಶೀಯ ಗುಣವೆಂಯ ತಮ್ಮ ದೌರ್ಬಲ್ಯಗಳನ್ನು ಮುಚ್ಚುಮ್ಮುಕ್ಕೆಲ್ಲಲು ಯತ್ನಿಸ ಬಹುದೇನೊ ? ○

ವಿಳಾಸ: ಆಸ್ಪೆಟ್ರೆಲ್ ಪೌಲ್ಟ್ರಿಕಲ್, ಮಂಗೋದ್ಯೆದ್ಯಕೀಯ ವಿಭಾಗ, ಕಸ್ತೂರ್‌ಬಾ ಆಸ್ಪತ್ರೆ, ಮಂಗಳೂರು – 576 119, ದ.ಕ.

ಮದ್ಯವ್ಯಸನದ ಕೊನೆ ಹಂತ. ಈ ಹಂತವೇರಿದ ವ್ಯಕ್ತಿ ಬೆಳಗ್ಗಿನಿಂದ ರಾತ್ರಿಯವರೆಗೂ ಒಂದೇ ಸಮನೆ ಕುಡಿಯು ತ್ತಲೇ ಇರುತ್ತಾನೆ. ಕೈಯಲ್ಲಿ ಮದ್ಯ ಬಿದಿಯೊಂದೇ ಇನ್ನು ಮುಂದೆ ಕುಡಿಯುವುದೇ ಇಲ್ಲ ಎಂದು ಸ್ವಯಾಣವನ್ನು ಮಾಡುತ್ತಾನೆ. ಲೋಟ ಖಾಲಿಯಾದ ಕೂಡಲೆ ಮತ್ತಷ್ಟು ಬಗ್ಗಿಸುತ್ತಾ ಪ್ರಮಾಣವನ್ನು ಮರೆಯುತ್ತಾನೆ. ಒಂದೇ ಒಂದು ಲೋಟ ಕುಡಿದರೂ ಸಾಕು. ಸಂಪೂರ್ಣವಾಗಿ ಅಮಲೇರಿ ಬಿಡುತ್ತದೆ. ದೇಹ ಸ್ಥಿತಿಯೂ ಆತಿಯಾಗಿ ಹದಗೆಟ್ಟಿರುತ್ತದೆ. ಮದ್ಯವ್ಯಸನಿ ಇವನೆಷ್ಟು ದಾಸನಾಗಿರು ತ್ತಾನೆ ಎಂದರೆ ನೀತಿ ನಿಯಮಗಳಿಲ್ಲೆಲ್ಲಾ ಗಾಳಿಗೆ ತೂರಿ, ಕುಡಿಯುವುದಕ್ಕಾಗಿ ಏನೂ ಮಾರಲು ತಯಾರಾಗಿಬಿಡು ತ್ತಾನೆ. ಮನೆಯಲ್ಲಿನ ಪಾತ್ರೆ ಪಗಡಿಗಳು, ಮಲ್ಲು ಒಡವೆ ಗಳು ಅಷ್ಟೇಕೆ,ಹೆಂಡತಿಯ ತಾಳಿಯನ್ನೂ ಕೂಡ ಒಂದೊಂದು ಪೆಗ್ ಸಿಗುವುದಾದರೆ ಮಾರಲು ಹಿಂಜರಿಯುವುದಿಲ್ಲ. ಇದರೆಯಿಲ್ಲಿಗೇ ಕತ್ತ ನೆರಳ್ತೇ ಕುಡಿತ ಭಯಪಡುವನು ಯಾರು, ಅಂಜಕೆಯ ಭೂತ ಮನಸ್ಸನ್ನಲ್ಲಿ ಮನೆಮಾಡು ತ್ತದೆ. ಪಾಾನಸೇವನೆಯ ಎಷ್ಟವ ಈ ಶ್ರಿಯಲ್ಲಿ ಎಷ್ಟರ ಮಟ್ಟಿಗೆ ಹದಗೆಡಿಸುಬಹುದೆಂದರೆ ಕೆಲ ವ್ಯಸನಿಗಳು ಎಲ್ಲಿದ ರಲ್ಲಿ ಸಂದೇಹ ಪಡುವವರಾಗಿ ಬಿಡುತ್ತಾರೆ. ಪತ್ನಿ ಮಗಳೇ ಈಾ ಬಡಿದರೂ ಅದರಲ್ಲಿ ವಿಪರೀತವು ದೆಂಬ ಭಯ ಬರುಬಹುದು. ಯಾರೋ ನ್ಪಿಕ ಜಾಗೆ, ಕ್ಷಣ ಮುಂದೆ ನರ್ತಪಡಾ ಹಾಗೆಲ್ಲಾ ಭ್ರಮೆ ಉಂಟಾಗಲು ತೊಡಗುತ್ತದೆ. ನರಮಂಡಲ ಸಂಚರ್ಣವಾಗಿ ತನ್ನ ಚುರುಕುತನ ಕಳೆದುಕೊಂಡು ಸ್ಪಾ ಪ್ಪಟ್ಟ ಕೆಲಸಗಳನ್ನು ಮಾಡುವಾಗಲೂ ಕೈ ನಡುಗಿ ತಪ್ಪಲು ಪ್ರಾರಂಭಿಸುಬಹುದು. ಹೀಗಾದಾಗ ಗುಂಡಿ, ಕಂಡ ಲೀಸು ಪಾಾನವೂರೂ ಪತ್ಪ ವಾಗುತ್ತದೆ. ಇದರೆಯಿಲ್ಲೇ ಅವರಿಬಿತ ಮದ್ಯ ಉ�ಯು ಮಾಡಿದ ರಕ್ತದ ಒತ್ತಡದ ತೊಂದರೆಗಳು. ಯಕೃತ್ತು ಕರುಳಿನ ನಾಕ ಮುಂತಾದ ಆನಾರೋಗ್ಯಗಳು ಕಾಡತೊಡಗಿ ವ್ಯಕ್ತಿ ಜೀವಚ್ಛವವಾಗಿ ಬಿಡುವುದುಂಟು. ಇಂತಹವರಿಗೆ ಸಮಾಜ ಆತಿ ಹಿಚ್ಚಿಂದರೆ ಕನಿಕರ ತೋರಬಹುದೇ ಹೊರತು ಮತ್ತೇನೂ ಮಾಡುವುದಿಲ್ಲ. ಹೀಗೆ ಸಮಾಜದಿಂದ ದೂರೀ ಕೃತನಾಗಿ, ಕುಟುಂಬದಿಂದೆ ನುಂಗಲಾರದ ಕಡುಬಾಗಿ ಬಿಡುವ ಇಂತಹ ಮದ್ಯವ್ಯಸನಿಗಳನ್ನು ಕಾಣಾಲು ಚಿಕಿತ್ಸೆ ಆತ್ಯಂತ ಅಗತ್ಯ.

ಮದ್ಯವ್ಯಸನಿಯ ಚಿಕಿತ್ಸೆಯ ವಿಧಾನವೇ ವಿಶಿಷ್ಟವಾದುದು. ಇದು ಕುಟುಂಬದ ವೈದ್ಯರೊಬ್ಬನಿಂದಲೇ ನಡೆಸಲಾಗುವ ದಿಲ್ಲ. ವೈದ್ಯರು, ಮನಶ್ಶಾಸ್ತ್ರಜ್ಞರೂ, ಮನಶ್ಶಾಸ್ತ್ರ ಸಮಾಜ ಸುಧಾರಕರೂ, ಕುಟುಂಬದವರೂ ಒಟ್ಟು ಸೇರಿ ಚಿಕಿತ್ಸೆ ನಡಸಬೇಕಾಗುತ್ತದೆ.

ಬಾಳಿದವರು ಈಕೆ. ಇನ್ನು ಯೌವನದಲ್ಲಿ ತನ್ನ ಪತಿಯನ್ನು ಕಳೆದುಕೊಂಡ ಇವರ ದುಃಖ ಮೇರೆ ಇಲ್ಲದ್ದು. ನಿಜ. ಈ ದುಃಖ ಇವರ ಚೈತನ್ಯವನ್ನು ಉಡುಗಿಸ ಬಿಡಲಿಲ್ಲ. ಕುಡಿದ ಚಟು ಆಂಟಿಸಿಕೊಂಡು ದಡೂಡಿಯ ದಾರಿ ಹಾಗದೆ ತೊಳಲು ತ್ತಿರುವವರಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡುವ, ಆ ಮೂಲಕ ಅವರ ಕುಟುಂಬ ಗಳಿಗೆ ನೆರವಾಗುವ ಸಾಧ್ಯತೆಗಳ ಬಗ್ಗೆ ಆಲೋಚಿಸತೊಡಗಿದರು.

ವಿದೇಶಿ ತರಬೇತಿ

ಜಗತ್ತಿನ ಬೇರೆ ಬೇರೆ ಕಡೆ ಈ ಸಮಸ್ಯೆ ಎಷ್ಟು ತೀವ್ರ ವಾಗಿದೆ, ಅಲ್ಲಿ ಇದಕ್ಕೆ ಯಾವ ಬಗೆಯ ಚಿಕಿತ್ಸೆ ನೀಡಲಾಗು ತ್ತಿದೆ ಎಂಬ ವಿವರ ಸಂಗ್ರಹಿಸತೊಡಗಿದರು. ಇವರ ಆಲೋಚನೆ ಕಾರ್ಯರೂಪಕ್ಕೆ ಬರುವ ಕಾಲ ಬಂದಿತು. 1979ರಲ್ಲಿ. ಆಲ್ಕೋಹಾಲಿಸಮ್‌ಗೆ ಚಿಕಿತ್ಸೆ ನೀಡುವುದ ರಲ್ಲಿ ಜಗತ್ತಿನಲ್ಲೇ ಆಗ್ರಗಾಮಿ ಸಂಸ್ಥೆಯಾದ ಅಮೆರಿಕದ ಹೆಜೆಲ್‌ಡನ್ ಇನ್‌ಸ್ಟಿಟ್ಯೂಟ್‌ನಲ್ಲಿ ತರಬೇತಿ ಪಡೆದು ಬಂದರು. ಬಂದವರೆ ಉದ್ದೇಶಿಸಿದಲ್ಲಿದ ವಾಲಂಟರಿ ಸಂಸ್ಥೆಯಾಗಿ ಇದನ್ನು ಸ್ಥಾಪಿಸಿದರು. 1987ರ ಅಕ್ಟೋಬರ 26ರಂದ ಇದು ತುಷ್ಟಿಕ ಸುಜ್ಞಿತ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ (ಟಿ.ಟಿ.ಕೆ. ಆಸ್ಪತ್ರೆ) ತನ್ನ ಕಾರ್ಯಮಾಡುತ್ತಿದೆ.

ಮುಂಜಾನೆಯಿಂದ ಸಂಜೆಯವರೆಗೆ ಚುರುಕಿನಿಂದ ಆಸ್ಪತ್ರೆಯ ಎಲ್ಲ ವಿಜಯಗಳನ್ನು ಗಮನಿಸುವ, ನಗುನಗುತ್ತ ಆಗತ್ಯ ಸಲಹೆ ನೀಡುವ, ಮೃದು ಮಾತಿನ, ಸರಳ ವ್ಯಕ್ತಿತ್ವದ ಈ ತೆಳ್ಳಿನ ಮಹಿಳೆ ಕುಡಿದ ಚಟುವಂದ ಗುಣಮೂಂದಿದ ಅನೇಕರ ಪಾಲಿಗೆ ದೇವತೆ. ಮಕ್ಕಳಲ್ಲದ ಇವರ ಪಿಶು ಈ ಸಂಸ್ಥೆ. ತಮ್ಮ ಬದುಕನ್ನು ಇದಕ್ಕಾಗಿಯೇ ಮೀಸಲಿಟ್ಟಿದಾರೆ.

ತಾಂತ್ರಿಕ ರಂಗವಷ್ಟೇಗ ಜತೆಗೆ ಸುರಿತ ಡಾಕ್ಟರುಗಳು, ಸಲಹೆಗಾರರು, ಮನಶ್ಶಾಸ್ತ್ರಜ್ಞರು, ಮನೋರೋಗ ಚಿಕಿತ್ಸಕರು, ನರ್ಸ್‌ಗಳು, ಸಾಮಾಯರು ಸೇರಿರತೆ ಮಾಜಿತ್ಯೆಮ ಮಂದಿ ಸೇವಾ ಮನೋಭಾವದ ಟ್ರಾಂಬಿಗೆದ್ದಾರೆ. ಇವರೆಲ್ಲ ಹೆಚ್ಚಿನವರು ಮಹಿಳೆಯರು.

ಈ ಆಸ್ಪತ್ರೆ ಮದ್ಯ ಮತ್ತು ಮಾದಕ ವಸ್ತುಗಳ ಚಟಕ್ಕೆ ಬಿದ್ದವರನ್ನು ಗುಣಪಡಿಸಲು ಆಗತ್ಯವಾದ ಎಲ್ಲ ಆಧುನಿಕ ಸೌಲಭ್ಯಗಳನ್ನೂ ಹೊಂದಿದೆ.

ಚಿಕಿತ್ಸೆ

ರೋಗಿ ಆಸ್ಪತ್ರೆಗೆ ದಾಖಲಾದ ಕ್ಷಣದಿಂದಲೇ ಚಿಕಿತ್ಸೆ ಪ್ರಾರಂಭವಾಗುತ್ತದೆ. 'ಡಿಟಾಕ್ಸಿಫಿಕೇಷನ್' ಅಥವಾ 'ಡಿಟಾಕ್ಸ್' ಎನ್ನಲಾಗುವ, ರಕ್ತದಲ್ಲಿ ಬೆರೆತುಹೋಗಿರುವ ಮದ್ಯದ ನಂಜು ಇಳಿಸುವ ವೈದ್ಯಕೀಯ ಚಿಕಿತ್ಸೆ. ಬೇರೆ ಅಂಗಗಳಿಗೆ ಹಾನಿಯಾಗಿದ್ದರೆ ಅದಕ್ಕೂ ಇವರು ನೆರವಾಗುತ್ತದೆ. ಇದು ಏಳರಿಂದ ಹತ್ತು ದಿನಗಳ ಚಿಕಿತ್ಸೆ. ಯಕೃತ್ತಿಗೆ ತೀರಾ ಹಾನಿಯಾಗಿದ್ದರೆ ಚಿಕಿತ್ಸೆ ಮೂರು ವಾರಗಳ ಕಾಲ ಮುಂದುವರಿಯುತ್ತದೆ. ರೋಗಿಗೆ ಪುಷ್ಟಿಕರವಾದ ಆಹಾರ ವನ್ನು ಆಸ್ಪತ್ರೆಯಲ್ಲೇ ನೀಡಲಾಗುತ್ತದೆ.

ಇದ್ಕಕ್ಕಿದ್ದಂತೆ ಮದ್ಯ ಸೇವನೆ ನಿಲ್ಲಿಸುವುದರಿಂದ ಈ ಅವಧಿಯಲ್ಲಿ ರೋಗಿಗೆ ನಿದ್ರಾಹೀನತೆ, ವಾಕರಿಕೆ, ನಡುಕ ಹಾಗೂ ಬಡಬಡಿಕೆ ಕಾಣಿಸಿಕೊಳ್ಳುವುದು ಸ್ವಾಭಾವಿಕ. ಮಾದಕ ವಸ್ತುಗಳ ಚಟದವರಿಗೆ ಈ ಹಿಂದಿನಿಂದೆ ಲಕ್ಷಣಗಳು (ವಿತ್‌ಡ್ರಾಯಲ್ ಸಿಂಪ್ಟಮ್ಸ್) ಬಹಳ ತೀವ್ರವಾಗಿರು ತ್ತವೆ. ಆಗ ನ್ಯೂರೋಲೆಪ್ಟಿಕ್ಸ್ ಎಂಬ, ಚಿಟುವಾಗಿ ಅಂಟಿ ಕೊಳ್ಳದ, ಔಷಧ ನೀಡಲಾಗುತ್ತದೆ.

ರಕ್ತದಲ್ಲಿರುವ ನಂಜು ಸಂಪೂರ್ಣವಾಗಿ ತೊಳೆದು ಹೋಗಿ ರೋಗಿಯ ದೈಹಿಕ ಹಾಗೂ ಮಾನಸಿಕ ಸ್ಥಿತಿ ಸುಧಾರಿಸಿ ಸಹಜ ಸ್ಥಿತಿಗೆ ಬಂದ ತರುವಾಯ ಮಾನಸಿಕ ಚಿಕಿತ್ಸೆ ಪ್ರಾರಂಭ ವಾಗುತ್ತದೆ. ರೋಗಿಯ ಮನೋಭಾವ, ವ್ಯಕ್ತಿತ್ವವನ್ನು ಸುಧಾರಿಸುವುದು ಮತ್ತು ವ್ಯಕ್ತಿಯಲ್ಲಿ ಪರಿವರ್ತನೆ ಉಂಟು ಮಾಡುವುದು ಇದರ ಉದ್ದೇಶ. ಆ ಸಂಧರ್ಭದಲ್ಲಿ ವ್ಯಕ್ತಿ ಯೊಬ್ಬ ರೋಗಿ ಒಬ್ಬಿಬ್ಬರು ಸಲಹೆಗಾರರ ಬಳಿ ಪ್ರತ್ಯೇಕ ವಾಗಿ ಸಲಹೆ ಪಡೆಯುತ್ತಾನೆ.

ಕುಟುಂಬ ತರಬೇತಿ

ಜತೆಯಲ್ಲೇ ವೈದ ಚಿಕಿತ್ಸೆ (ಗ್ರೂಪ್ ಥೆರಪಿ) ನಡೆಯು ತ್ತದೆ. ಮುಂಜಾನೆ ವಿಲ್ಲರೂ ಒಂದೆಡೆ ಸೇರಿ ವ್ಯಾಹಿಸು ತ್ತಾರೆ. ಲಲ್ಲಿ ಯಾವ ದೇವರ ಹೆಸರಿಲ್ಲೂ ಭಜನೆಯೋ, ಮತ್ತೊಂದೋ ನಡೆಯುವುದಿಲ್ಲ. ಬೇಸರ ಹುಟ್ಟಿಸುವ ರೀತಿಯಲ್ಲಿ ಯಾರೂ ಉಪದೇಶ ನೀಡುವುದಿಲ್ಲ. ಮನುಷ್ಯರ ಪರಸ್ಪರ ತಿಳುವಳಿಕೆಲ್ಲಿ, ಕುಡಿತ ಬಿಟ್ಟು ಸಹಜ ಜೀವನ ನಡೆಸುವುದಕ್ಕಾಗಿ ಸಂಕಲ್ಪ ಮಾಡಲು ಅವಕಾಶ ಕಲ್ಪಿಸಿಕೊಡುವುದು ಇದ್ದ ಉದ್ದೇಶ.

ಮುಂದಿನ ಹಂತದಲ್ಲಿ ರೋಗಿಯ ಕುಟುಂಬದವರೂ ಇದರಲ್ಲಿ ತೊಡಗಿಸಲಾಗುತ್ತದೆ. ರೋಗಿ ಗುಣಮುಖ ನಿಂತಿರುವಿದಾಗ ಮನೆಯಲ್ಲಿ ಹೊಸ ವಾತಾವರಣ ಇರ ಬೇಕಾಗುತ್ತದೆ. ಇದಕ್ಕೆ ಮನೆಯವರ ಸಹಾಯ, ಸಹಾನು ಭೂತಿ ಅಗತ್ಯ. ಈ ಬಗ್ಗೆ ಕುಟುಂಬದವರಲ್ಲಿ ತಿಳಿವಳಿಕೆ ಉಂಟುಮಾಡುವುದು ಇದರ ಉದ್ದೇಶ. ಆ ವ್ಯಕ್ತಿಗೆ ಮಕ್ಕಳ ದ್ರಲ್ಲಿ ಅವರನ್ನೂ ಇದರಲ್ಲಿ ಸೇರಿಸಿಕೊಳ್ಳಲಾಗುತ್ತದೆ.

ಸಾಮಾಜಿಕ ಕಾರ್ಯಕರ್ತರು ತರಬೆಯು ಪಡೆಯುತ್ತಿರುವುದು

ಪರಂಜ್ಯೋತಿ (ಕೆ. ಪಿ. ಸ್ವಾಮಿ): ನಲಿಗಿರಿಯ ಕೋರ ಣಿಂದ ವಿಕ್ಷೇಗರವರೆಗೆ 1936ರಲ್ಲಿ ಜನನ. ಪುತ್ತೂರಿನಿಂದ ಪ್ರಕಟವಾಗುತ್ತಿದ್ದ 'ಇಂದ್ರಧನುಸ್'ನಲ್ಲಿ ಮತ್ತು 'ಪ್ರಪಂಚ' ಬೆಂಗಳೂರು ಅವ್ಯತ್ಕಿಯಲ್ಲಿ ಸ್ವಲ್ಪ ಕಾಲ ಉದ್ಯೋಗ. ಈಗ ಮದರಾಸಿನ 'ಸೋವಿಯತ್ ಲ್ಯಾಂಡ್' ಕೇತೀಯಲ್ಲಿ ಉದ್ಯೋಗ. 'ನಲಾಲಾಂಬ', 'ಸ್ವಾಮಿ', 'ನಲ್ಲಿರಿಯಮ್ಮ'— ಇತ್ಯಾದಿ ಪದಬಂಗಳನ್ನೂ ಬರೆದಿದ್ದುಂಟು. ಪತ್ರಿಕೆಗಳಲ್ಲಿ ಪ್ರಕಟವಾಗಿದ ಕತೆಗಳು ಹಾಗೂ ಲೇಖನಗಳು ಸುಮಾರು ನೂರು. ಎರಡು ಕಥಾ ಸಂಕಲನಗಳು, ಮೂರು ಕಾದಂಬರಿ ಗಳು ಪ್ರಕಟವಾಗಿವೆ. ಕೆಲವು ಕತೆಗಳು ಹಿಂದಿ, ತಮಿಳು, ಮಲಯಾಳಂ ಭಾಷೆಗಳಿಗೆ ಅನುವಾದಗೊಂಡಿವೆ. ತಮಿಳಿ ನಿಂದ ಅನುವಾದಿಸಲ ಕೆಲವ ಕತೆಗಳು ಹಾಗೂ ಇತರ ಮೂರು ಕೃತಿಗಳು ಕನ್ನಡದಲ್ಲಿ ಪ್ರಕಟವಾಗಿವೆ.

● ಕೆ. ಪಿ. ಸ್ವಾಮಿ (ಪರಂಜ್ಯೋತಿ)
ವಿಳಾಸ: ನಂ. 15, ವಿ. ವಿ. ಕೋಯಿಲ್ ಲೇನ್,
ತೇಯ್ಕುಂಪೇಟೆ,
ಮದರಾಸು—600 086.

ತಂದೆಯ ಹಿಂದಿನ ವರ್ಷಗಳ ಮಳ್ಳ ಮೇಲೆ ಉಂಟುಮಾಡಿರ ಬಹುದಾದ ಕೆಲ ಭಾವನೆಯನ್ನು ತೊಲಗಿಸಲು ಪ್ರಯತ್ನಿಸ ಲಾಗುವುದೆ. ಅವರು ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಎಷ್ಟೊಗಾಗಿ ನಡೆಯುವ ಚಟುವಟಿಕೆಗಳಲ್ಲಿ—ಕಥೆ ಹೇಳುವುದು, ಚಿತ್ರ ರಚನೆ, ಆಂಗ ಸಾಧನೆ ಮತ್ತು ಕ್ರೀಡೆಗಳು—ಭಾಗವಹಿಸುತ್ತಾರೆ.

ಚಿಕಿತ್ಸೆ ಮುಗಿದ ವ್ಯಕ್ತಿ ಎಂದಿನಂತೆ ತನ್ನ ಸಹಜ ಚಟುವಟಿಕೆಗಳಲ್ಲಿ ತೊಡಗಿಕೊಂಡ ಮೇಲೆ ಮತ್ತೆ ಕುಡಿತದ ಚಟಲ ತಟ್ಟುವುದಿಲ್ಲವೆಂದು ಹೇಳಲಾಗುವುದಿಲ್ಲ. ಅನಸ್ಥ ತತೆಯಲು ಆತ ಕನಸ್ಸಪಕ್ಷ ಒಂದು ವರ್ಷದವರೆಗೆ ಪ್ರತಿದಿನ ಒಂದೊಂದು ರಿಫಿಲ್‌ಕ್ಕಿಂಗ್ ಅಥವಾ ಎಚ್ಚಿಲ್‌ಗಳ ಗುಳಿಗೆ ತೆಗೆದುಕೊಳ್ಳುವಂತೆ ಮಾಡುತ್ತಾರೆ. ಅಂಟಾಬ್ಯುಸ್ ವ್ಯಕ್ತಿ ಲಾಗುವ ಆ ಚಿಕಿತ್ಸೆಯ ಸಂಧರ್ಭದಲ್ಲಿ ಕುಡಿದ ಆತನ ದೇಹ ಮತ್ತು ಮನಸ್ಸಿನ ಮೇಲೆ ಉಂಟಾಗುವ ತಿವ್ರ ವರಿಣಾಮಗಳ ಬಗ್ಗೆ ಎಚ್ಚರಿಕೆ ನೀಡುತ್ತಾರೆ. ಆದರೆ ಕೆಲವರಿಗಾದರೂ ಇದನ್ನು ಪರೀಕ್ಷಿಸಿ ನೋಡುವ ಚಪಲ ವಾಗದೆ ಇರುವುದಿಲ್ಲ. ಎಚ್ಚರಿಕೆಯನ್ನು ಮೀರಿ ಕುಡಿಯು ವಾತ ಎಲ್ಲಿಯಾದರೂ ವ್ಯಕ್ತಿತಪ್ಪಿ ಬೀಳುವುದು. ಆಗ ಆತ ಚಿಕಿತ್ಸೆ ಪಡೆಯುತ್ತಿರುವ ಬಗ್ಗೆ ವ್ಯಕ್ತಿಯ ಜೇಬಿನಲ್ಲಿ ಒಂದು ಮಾಹಿತಿ ಚೀಟಿ ಇರುತ್ತದೆ. ಅದನ್ನು ಗಮನಿಸುವ ಯಾವ ಡಾಕ್ಟರಾದರೂ ಯಕೃತ್ತಿ ಚಿಕಿತ್ಸೆ ನೀಡುತ್ತಾರೆ ಅಥವಾ ವ್ಯಕ್ತಿಯನ್ನು ಸಂಸ್ಥೆಗೆ ಕಲಿಸಿಕೊಡುತ್ತಾರೆ. ಒಮ್ಮೆ ಈ ಅಮಲಿನ ಅಧ ಮೇಲೆ ಆತ ಮತ್ತೆ ಕುಡಿಯುವ ಸಾಹಸ ತೋರಿಸಿಲಾರೆ. ಇದು ದುರವಸ್ಥೆಗೂಗದ ವಿಶುದ್ಧ ಒಂದು ರೀತಿಯ ಪಾಕ್ ಟ್ರೀಟ್‌ಮೆಂಟ್.

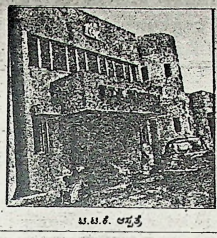
ತರುವಾಯದ ಫಾಲೊ-ಅಪ್ ಈ ಸಂಸ್ಥೆಯ ವರಾಂಬ ರೀಯ ಮುಖ್ಯಭಾಗವಾಗಿದೆ. ಇದು ಏದು ವರ್ಷಗಳವರೆಗೆ ಮುಂದುವರಿಯುತ್ತದೆ. ವ್ಯಕ್ತಿಯನ್ನು ಪ್ರತಿವಾರವೂ ಆಸ್ಪತ್ರೆ ಯಲ್ಲಿ ನಡೆಯುವ, ವರಾಂಬರೀಯ ತರುವಾಯದ ಕಾರ್ಯ ಕ್ರಮದಲ್ಲಿ ಭಾಗವಹಿಸುವಂತೆ ಪ್ರೋತ್ಸಾಹಿಸಲಾಗುತ್ತದೆ. ಪ್ರಾರಂಭದಲ್ಲಿ ಹದಿನೈದು ದಿನಕ್ಕೊಮ್ಮೆ, ಮುಂದೆ ತಿಂಗಳಿಗೊಮ್ಮೆ, ಎರಡು ತಿಂಗಳಿಗೊಮ್ಮೆ ಸಲಹೆಗಾರರನ್ನು ಭೇಟಿ

ಮಾಡುವಂತೆ. ಆಗ್ಗಾಗಾಗ್ಗೆ ವೈದ್ಯಕೀಯ ಸಲಹೆ. ಚಿಕಿತ್ಸೆ ವರೆಯುವಂತೆ ಪೌಷ್ಟಿಕಾಲಾಗುತ್ತದೆ. ಚಟ್ಟಿಯ ಪ್ರಗತಿಯನ್ನು ಐದು ವರ್ಷಗಳವರೆಗೆ ದಾಖಲುಗೊಳಿಸಲಾಗುತ್ತದೆ. ಸಂಸ್ಥೆಯ ದೃಷ್ಟಿಯಲ್ಲಿ ಕುಡಿತ ತ್ಯಜಿಸುವ ಮಾತ್ರವೇ ಅಲ್ಲ, ಆತನ ಉದ್ಯೋಗ ಬಿಡಿ, ವ್ಯಾಪಾರ ವ್ಯಾಪ್ತಿ, ಮಕ್ಕಳ ವಿದ್ಯಾಭ್ಯಾಸ, ಮಾನವ ಬಂಧ, ಮನೆಯಲ್ಲಿ ಪತಿಯಾಗ ಮಡದಿ ಇತ್ಯಾದಿ ಎಲ್ಲವೂ ಆತನ ಪ್ರಗತಿಯ ಸಂಕೇತಗಳೇ.

ಚಿಕಿತ್ಸೆಗಾಗಿ ಬರುವವರ ಬಗೆಗಿನ ಮಾಹಿತಿಗಳನ್ನು ಮುಂಜಿ ಯವ ರೀತಿಯಿಲ್ಲಿ ಆವರ ಪರಿಚಯಕ್ಕೆ ಹಾನಿಯಾಗದಂಬು ಕಾಳಜಿಯಿಂದ ರಕ್ಷಣೆಗಾಗಿಯೂ ಆಗುತ್ತದೆ.

ಸ್ವಯಂಸೇವಕ ದಳ

ಈ ಸಂಸ್ಥೆ ವ್ಯಕ್ತಿಗತ ಕರಪೇಕ್ಷಿ ಕೇಂದ್ರವನ್ನು ಹೊಂದಿದ್ದು, ಈ ರಂಗದಲ್ಲಿ ಸೇವೆ ಸಲ್ಲಿಸಲು ಆಸಕ್ತಿ ತೋರಿಸುವ ಸಮಾಜ



ಬ.ಟಿ.ಕೆ. ಆಸ್ಪತ್ರೆ

ಸೇವಾ ಸಂಸ್ಥೆಗಳು, ಉದ್ಯಮಗಳು, ಕಾಲಾ-ಕಾಲೇಜುಗಳ ಸ್ವಯಂಸೇವಕರಿಗೆ ತರಬೇತಿ ನೀಡುತ್ತವೆ. ಉದ್ಯಮಗಳಿಂದ ಬರುವ ವ್ಯಕ್ತಿವಿಧಿಗಳು ಆಲ್ಕೋಹಾಲ್‌ಮುನ್ನು ಸೇವಾರಲ್ಲಿ ಗುರುತಿಸುವ. ಆ ಬಗ್ಗೆ ಕ್ರಮ ಕೈಗೊಳ್ಳುವ ಬಗ್ಗೆ ತರಬೇತಿ ನೆಡೆಯುತ್ತಾರೆ. ಹಾಗೇ ಈ ರಂಗದಲ್ಲಿ ಸೇವೆ ಸಲ್ಲಿಸುತ್ತಿರುವ 'ಆಲ್ಕೋಹಾಲ್ಕ್ಸ್ ಅನಾನಿಮಸ್' ಮತ್ತು 'ಅಲ್ ಅನನ್' ಸಂಸ್ಥೆಗಳೊಂದಿಗೆ ಸಹಕರಿಸುತ್ತದೆ.

ಕುಡಿಬಿಂದ ತಮ್ಮ ಕೆಲಸ ಕಳೆದುಕೊಂಡವರಿಗೆ ಬೇರೆ ಬೇರೆ ಕಡೆ ವ್ಯಯಕ್ತಿಸಿ ಮತ್ತೆ ಕೆಲಸ ದೊರಕಿಸಿಕೊಡಲು ವ್ಯಯಕ್ತಿಸುತ್ತದೆ.

ಆಲ್ಕೋಹಾಲಿಸಮಿನಿಂದ ಉಂಟಾಗುವ ಹಾನಿಗಳ ಬಗ್ಗೆ ಧಿಕ್ಕಿ ವಕ್ಕ, ಕರವತ್ರಗಳ ಮೂಲಕ ಜನರಲ್ಲಿ ಜಾಗೃತಿ ಉಂಟುಮಾಡಲು ವ್ಯಯಕ್ತಿಸುತ್ತದೆ. ಈ ವಿಷಯದ ಬಗ್ಗೆ ಅನೇಕ ಪುಸ್ತಕಗಳನ್ನು ವ್ಯಕಟಿಸಿದೆ.

ಈ ಸಂಸ್ಥೆ ತತ್ವದೇ ಸಂಶೋಧನಾ ವಿಭಾಗವನ್ನು ಹೊಂದಿದ್ದು, ಜಗತ್ತಿನ ವೆರೆ ವೆರೆ ಕಡೆಗಳಲ್ಲಿರುವ ಇಂಥ ಸಂಸ್ಥೆಗಳೊಂದಿಗೆ ಈ ರಂಗದಲ್ಲಿನ ತನ್ನ ಅನುಭವವನ್ನು ವಿನಿಮಯ ಮಾಡಿಕೊಳ್ಳುತ್ತದೆ. 1987ರ ಆರಂಭದಲ್ಲಿ ಹಾಂಕಿ ರಂಗನಾಥನ್ ಹೆಂಡದಲ್ಲಿ ನಡೆದ ವಿಚಾರ ಸಂಕಿರಣದಲ್ಲಿ ಭಾರತದ ವ್ಯಕ್ತಿವಿಯಾಗಿ ಹೋಗಿ ಬಂದಿದ್ದಾರೆ. ಇಲ್ಲಿನವರನ್ನೂ ಅಲ್ಲಿಗೆ ತರಬೇತಿಗಾಗಿ ಕಳುಹುತ್ತಾರೆ. ಅಲ್ಲಿನ ತಜ್ಞರೂ ಇಲ್ಲಿನ ಚಿಟುಬಟಿಗಳನ್ನು ಮೆಚ್ಚಿದ್ದಾರೆ.

ಚಿಕಿತ್ಸೆಗಾಗಿ ರೋಗಿಗಳು ಆರಿಸಿಕೊಳ್ಳುವ ಸೌಲಭ್ಯಗಳಿಗೆ ತಕ್ಕಂತೆ ಚಿಕಿತ್ಸೆಗೆ ರೂ. 2000ದಿಂದ 7000ವರೆಗೆ ವೆಚ್ಚವಾಗುತ್ತದೆ. ಕೆಲವರಿಗೆ ಇದು ದುಬಾರಿ ಎನ್ನಿಸಬಹುದು. ಹಾಗೆಂದು ವೆಚ್ಚ ಮಾಡಲು ಸಾಧ್ಯವಿಲ್ಲದವರಿಗೆ ಅಥವಾ ಬರಿಗೈಯಲ್ಲಿ ಬಂದವರಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡದೆ ಕಳುವುದಿಲ್ಲ. ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಏರು ಉಚಿತ ಹಾಸಿಗೆಗಳಿವೆ. ಇವುಗಳ ವೆಚ್ಚವನ್ನು ಸಂಸ್ಥೆ ತುಂಬಿಕೊಡುತ್ತದೆ.

ಸಂಸ್ಥೆಯ ಸಂಶೋಧನೆ ದಾಖಲೆಗಳಂದ ತಿಂಯ ಬರುವಂತೆ, ವ್ಯತಿ ತಿಂಗಳು ಸುಮಾರು 50 ಮಂದಿ ಚಟ್ಟಸ್ಥರಿಗೆ ದಾಖಲಾಗುತ್ತಾರೆ. ಈ ಕೇಂದ್ರ ಈವರೆಗೆ 2500 ಮಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡಿದೆ. ಕೇ. 60ರಷ್ಟು ಮಂದಿ ಸಂಪೂರ್ಣ ಗುಣ ಹೊಂದಿದ್ದಾರೆ. ಕೇ. 30ರಷ್ಟು ಮತ್ತೆ ಮತ್ತೆ ಚಿಕಿತ್ಸೆಗಾಗಿ ಬಂದದ್ದುಂಟು. ಉಳದ ಕೇ. 10ರಷ್ಟು ಗುಣಹೊಂದಿರ ಬಹುದು ಅಥವಾ ಮಾಹಿತಿ ನೀಡಲು ಅವಕಾಶವಿಲ್ಲದೆ ಹೋಗಿರಬಹುದು. ಚಿಕಿತ್ಸೆಗಾಗಿ ಬಂದವರಲ್ಲಿ ಕೇ. 99 ರಷ್ಟು ಗಂಡಸರು. ಈವರೆಗೆ 20 ಮಂದಿ ಹೆಂಗಸರು ಚಿಕಿತ್ಸೆ ಪಡೆದಿದ್ದಾರೆ. ಕೇ. 50ರಷ್ಟು ಜನರು ಏರು ವರ್ಷಗಳ ವರೆಗೂ ತಪ್ಪದೆ ತಮ್ಮ ಬಗ್ಗೆ ಮಾಹಿತಿ ಒದಗಿಸುತ್ತಾರೆ.

ಬಡವರು, ಶ್ರೀಮಂತರು ಎಂಬ ತಾರತಮ್ಯವಿಲ್ಲದೆ ಎಲ್ಲ ವರ್ಗದವರು, ಯಶಸ್ವಿ ವ್ಯಕ್ತಿಗಳಲ್ಲಿರುವವರು, ಕೈ ತುಂಬಾ ಸಂಬಾದಿಸುವ ವ್ಯಾಪಾರಿಗಳು, ನಿರುದ್ಯೋಗಿಗಳು, ಸಾಧಾರಣ ಕೂಲಿಗಾರರು–ಹೀಗೆ ವ್ಯಾಧಿ ಪೀಡಿತರಲ್ಲಿ ಸಮಾಜದ ಎಲ್ಲ ಸದಸ್ಯರೂ ಇದ್ದಾರೆ. ರೋಗಿಗಳ ಸರಾಸರಿ ವಾಯು 250ಂದ 55 ವರ್ಷ. ಹೆಚ್ಚಿನವರು 350ಂದ 50ರ ವಾಯುದವರು. ಮಾದಕ ವಸ್ತುಗಳ ಚಟಕ್ಕೆ ಬಿದ್ದವರು ಹೆಚ್ಚಾಗಿ ಯವ ಜನರು. 200ಂದ 25ರ ವಾಯುದವರು.

ಕೇ. 83ರಷ್ಟು ವಿವಾಹಿತರು. ಕೇ. 43ರಷ್ಟು ಮಂದಿ ಒಂಟಿಯಾಗಿ ಕುಡಿಯುವವರು. ಕೇ. 57 ಕುಟುಂಬಗಳಲ್ಲಿ ಮದ್ಯದ ಆಭ್ಯಾದವರು ಯಾರೂ ಇಲ್ಲ. ಕೇ. 38 ಕುಟುಂಬಗಳಲ್ಲಿ ಆಭ್ಯಾವಿತ್ತು.

ಸಣ್ಣ ಮಟ್ಟದಲ್ಲಿ ಈ ಸಂಸ್ಥೆಯನ್ನು ಸ್ಥಾಪಿಸಿದಾಗ, ಇದು ಇಷ್ಟು ದೊಡ್ಡ ಆಗ್ಗಾಮಿ ಸಂಸ್ಥೆಯಾಗಿ, ಇಷ್ಟು ಮಂದಿಗೆ ಚಿಕಿತ್ಸೆ ನೀಡುವಷ್ಟು ಯಶಸ್ಸಿಯಾಗುತ್ತುಂಬಂ ಕಲ್ಪನೆ ಹಾಂಕಿ ರಂಗನಾಥನ್‌ರಿಗಿರಲಿಲ್ಲ.

ಸಂಸ್ಥೆಯ ಕರವತ್ರದಲ್ಲಿನ ಕೊನೆಯಲ್ಲಿ ಈ ಮಾತಿದೆ: ಸರ್ವೇ ಜನಾ ಸುಖಿನೋ ಭವಂತು. ●

ಮದ್ಯಪಾನ ಕೆಲವು ಮುಖ್ಯ ಅಂಶಗಳು

ಮದ್ಯಗಳಲ್ಲಿನ ಆಲ್ಕೋಹಾಲ್ ಅಂಶ

ಮದ್ಯ	ಪ್ರಮಾಣ	ಆಲ್ಕೋಹಾಲ್ ಅಂಶ
ವಿಸ್ಕಿ, ಬ್ರಾಂಡಿ, ಸಾಯಾಯಿರಮ್	30 ಎಂ.ಲಿ. (1 ಪೆಗ್ಸ್ ಅಥವಾ 1 ಡ್ರಿಂಕ್)	40 ರಿಂದ 50%
ವೈನ್ಸ್‌ಗಳು	60 ಎಲಿ (2 ಪೆಗ್ಸ್ ಅಥವಾ 1 ಡ್ರಿಂಕ್)	10 ರಿಂದ 20%
ಬೀಯರ್, ಹೆಂಡ	285 ಎಲಿ (10 ಪೆಗ್ಸ್ ಅಥವಾ 1 ಡ್ರಿಂಕ್)	6 ರಿಂದ 8%

ಮದ್ಯ ಸೇವಿಸಿದಾಗ 1 ಗಂಟೆಯೊಳಗೆ ಆಗುವ ಪರಿಣಾಮಗಳು

1–2 ಡ್ರಿಂಕ್ಸ್ – ರಕ್ತದ ವೇಗ ಹೆಚ್ಚಿ ಚರ್ಮ ಕೆಂಪಿಗಾಗುತ್ತದೆ. ಮಾತಿನಲ್ಲಿ ಆತಂಕ ಕಡಿಗೆ, ಸ್ವೀನವರಿತೆ ಹೆಚ್ಚುತ್ತದೆ.

3 ಡ್ರಿಂಕ್ಸ್ – ತಲೆ ತಿರುಗುವಿದೆ, ಅಂದಾಜು ತಪ್ಪುತ್ತದೆ. ಆಗ ಕೈಗೊಳ್ಳುವ ತೀರ್ಮಾನ ಹಾನಿಕಾರಕ ವಾಗಿರುತ್ತದೆ.

5–6 ಡ್ರಿಂಕ್ಸ್ – ದೃಷ್ಟಿ ಮಂದವಾಗುತ್ತದೆ, ಮಾತು ತೊದಲುತ್ತದೆ. ನಿಧಾನ ಪ್ರತಿಕ್ರಿಯೆ.

8–9 ಡ್ರಿಂಕ್ಸ್ – ಆಯ ತಪ್ಪತ್ತದೆ, ಕಣ್ಣಿಗೆ ಎರಲೆರಂತು ದೃಶ್ಯ ಕಾಣಿಸುತ್ತದೆ.

20 ಡ್ರಿಂಕ್ಸ್ – ವ್ಯಕ್ತಿ ಕೌಮ್ಯ ಸಂಭವ ಹೆಚ್ಚು.

ಸ್ವಲ್ಪ ಸಮಯದಲ್ಲಿ 20 ಡ್ರಿಂಕ್ಸ್‌ಗಳಿಗಿಂತ ಹೆಚ್ಚು ಕುಡಿದರೆ, ಆಲ್ಕೋಹಾಲ್ ಕ್ರಾಸವನ್ನು ನಿರ್ಬಂಧಿಸುವುದರಿಂದ, ಮರಣ ಸಂಭವಿಸಬಹುದು.

ಹದೆಗೆಡದೇ ಕುಡಿಯುವುದರಿಂದ ಶರೀರದ ಅಂಗಗಳ ಮೇಲೆ ಉಂಟಾಗುವ ಪರಿಣಾಮ

ಹೃದಯ
ಆಮ್ಲಿರ ರಕ್ತದೊತ್ತಡ. ಕ್ರಮ ತಪ್ಪಿದ ನಾಡಿಬಡಿತ. ಹೃದಯ ಉಬ್ಬುವಿಕೆ.

ಮೇದೋಜೀರಕ ಗ್ರಂಥಿ
ಉರಿಯೂತದ ವೇದನೆ.

ಯಕೃತ್ತು
ತಿವ್ರ ಊದುವಿಕೆ, ತೆಪಟಿಟಿಸ್, (ಯಕೃತ್ತಿನ ಊತ) ಸಿರೋಸಿಸ್.

ಸ್ನಾಯುಗಳು
ಕ್ಷೀಣತೆ. ಸ್ನಾಯುವೆರೆಗಳ ಸವೆತ.

ಜೊಟ್ಟೆ
ಕರುಳು ತುಡ್ಡು

ಪರಿಮಂಡಲ
ಜೋಮು ಹಿಡಿಯುವಿಕೆ. ಕೈಕಾಲುಗಳ ಸಂವೇದನ ಶಕ್ತಿ ಕುಂದುವಿಕೆ.

ಮೆದುಳು
ನೆಲ್‌ಗಳಿಗೆ ಹಾನಿಯಾಗಿ ಸ್ಮರಣ ಶಕ್ತಿ ಕಡಿಮೆಯಾಗುತ್ತದೆ. ಗೊಂದಲ, ಭ್ರಮೆ.

ಶ್ವಾಸಕೋಶ
ಕ್ಷಯರೋಗ ಸೆರಿದಂತೆ ತಿವ್ರ ಅಂಟುಜಾಡ್ಯ.

ಜನನೇಂದ್ರಿಯ
ಶಾಶ್ವತಿಕ ವಂಂತನ.

ಚರ್ಮ
ಬಳಿಚಿಕೊಳ್ಳುವಿಕೆ, ಬೆವರುವುದು, ವಿವರ್ಣವಾಗುವುದು.

ರಕ್ತ
ಕೆಂಪು ರಕ್ತಕಣಗಳಲ್ಲಿ ಬದಲಾವಣೆ.

SMOKING IS A HEALTH HAZARD

TOBACCO smoking is injurious to health because of nicotine and carbon monoxide contained in the smoke. These particles are drawn into the nose and throat during smoking and into the lungs by inhalation.

It is generally accepted that nicotine raises the blood pressure, heart beat, heart work and blood cholesterol. Nicotine action eventually makes a person prone to heart attacks and strokes. Researchers say nicotine reaches a smoker's brain in six seconds as opposed to 14 seconds when injected intravenously.

Carbon monoxide is equally harmful. It reduces the free oxygen available in the blood which is very essential for the proper functioning of not only the heart muscles but the entire body. It makes attacks, such as, the ischaemic heart diseases more common amongst the smokers.

Smoking leads to diseases:

Smoking is the single most preventable cause of lung cancer, emphysema (serious lung disease), chronic bronchitis, ischaemic heart diseases and peptic (stomach) ulcer. The trend of scientific evidence has been consistant to indicate that tobacco smoking particularly cigarette smoking shortens life. many a people, it is opined, needlessly become sick, disabled or die before their 'time' due to their smoking habit.

Lung Cancer

Cigarette smoke paralyses the tiny hair (cilia) which line the bronchial tubes, that sweep foreign particles out of lungs. Without this protective action, healthy lung tissues can be injured, even destroyed by particles which remain in the lung.

Statistics from various countries including India reveal that cigarette smoking leads to cancer of the lung. In fact, the larger the number of cigarettes smoked and the longer they are smoked, the greater are the risks to the smokers. On the other hand, studies show that in those who stop smoking, the risk of lung cancer decreases directly in proportion to the length of time they have stopped smoking. It has also been found that pipe, cigar and bidi smokers run a lesser risk of lung cancer. But they are more prone to cancers of the lips. tongue and mouth than non-smokers.

Bronchitis and emphysema

Bronchitis is inflammation of the air tubes. In chronic bronchitis, there is a continuous inflammation of the air tubes because of irrigation by the smoke. The lining membranes of the air passage get thickened and abnormal.

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Surface of the bronchial tubes is lined with hair like cilia (fine hair). These fine hair help the lungs expel irritating or poisonous particles. Smoking paralyses and then destroyes the cilia which play a very important protective function for the lungs. It is commonly agreed that cancerous lesions are most frequently found along the bronchial tube lines, particularly in places where the exposure of smoke is intense.

Emphysema is another respiratory disease which mostly affects the cigarette smokers. Damage to lungs is caused because the cilia (fine hair) tend to diappear leading to hyper-inflation of lung tissues. The patient has to struggle to keep the lungs working. He or she requires big effort at each breath.

Smoking also affects the larynx or voice box. The changes at the voice box are similar to those that occur in the air passages and in the lungs. These irriations cause swellings and result in 'smoker's cough'. Cigarette smokers usually pass on phelgm. It has been observed that symptoms of cough and expectoration are closely associated with smoking. These symptoms usually subside rapidly in those who stop smoking.

Ischaemic heart disease

This is condition in which heart muscles receive less blood supply through coronory arteries. Smoking is one of the factors which affects heart and the blood vessels. It is often seen that smoking increases the severity of angina pictoris-a condition which usually precedes heart attacks. Smokers die more often from coronary heart attacks. Mortality from ischaemic heart diseases is greater in cigarette smokers than in non-smokers. It increases with more cigarettes smoked. It is lower in cigarette smokers who stop smoking than in these who 'love' smoking.

Among the younger smokers the risk of death is two to three times more than in non-smokers.

Peptic (stomach) ulcer

Tobacco smoking delays the healing of stomach ulcer, increases the pain, and the size of the ulcer. Patients who smoke wh show a poor respons to the treatment.

Smoking in pregnancy

Smoking during pregnancy is harmful. A pregnant mother who smokes, can endanger the life of her child. She is likely to have an undersized baby and run a greater risk of death during child birth. The risk to babies of mothers who had smoked during pregnancy increases by two times. Babies may be still-born or die soon after death or there may be an abortion. In fact, one in five babies lost can be saved if their mothers do not smoke.

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Socio-psycho aspects

Smoking for fun or to calm one's nerves is a costly price to pay. Young people often take to smoking in imitation of older people or as an expression of a subconscious wish to be like them. Older children and youth want to be accepted by their friends and associates. Often friends dress alike, talk alike and have other behavious in common. Smoking may be part of this attempt to conform. Non-smoking could also be part of a group pattern. As a means of relaxation or fun on individual is responsible for his/her own decisions. But never forget that in a few years teenagers will be fathers and mothers. Then they will have a responsibility to their own children. They will have to do everything now to keep their children healthy and live as long a life as possible.

Let us look at facts:

Smoking is a big health hazard. The risk of contracting any one or more of the diseases through smoking increases with the number of cigarettes smoked. Evidence has proved that lung cancer is often fatal, unless found early. All medical scientists agree it is 'smarter' never to develop the smoking habit. Doctors advise that if one does not smoke, things are in his favour, healthwise. Moreover, smoking costs money.

To conclude, the hazards of smoking are Many:-

* The greatest single cause of preventable death is cigarette smoking.
* Cigarette smoking may lead to lung cancer.
* Cigarette smokers are more prone to heart disease.
* Children of women who smoke during pregnancy are likely to be adversely affected.
* The earlier the people start smoking, the greater the risk of heart trouble. Teenage years are very important to avoid smoking.
* The early effects of smoking may handicap teenagers who go in for competitive sports.
* Cigarette smoking can lead to the cancer of the lips, larynx and oesophagus.

Hence, smoking or health, the choice is one's own. To not to smoke is, however, a better and a wiser choice, in one's own interest.

Source: Swasth Hind - March/April 1980

Globalization and Increasing Trend of Alcoholism *

1. Introduction

Although alcohol consumption has existed in India for many centuries, the quantity patterns of use, and resultant problems have undergone substantial changes over the past two decades. Alcohol consumption produces individual health and social problems. The global burden of disease from alcohol exceeds that of tobacco and is on a par with the burden attributable to unsafe sex world wide (Global Status Report on alcohol, WHO, 1999). Although recorded alcohol consumption per capita has fallen since 1980 in most developed countries, it has risen steadily in developing countries and alarmingly so in India. The per capita consumption of alcohol by adults of 15 years and above in India increased by 106.67 percent between 1970-72 and 1994-96!

2. Alcohol industry

Based on beverage type the Indian alcohol industry has three prominent sectors: The IMFL (Indian Made Foreign Liquor) and beer sector, the country liquor sector, and the illicit liquor sector. The IMFL and beer sector is the most visible part of the alcohol Industry, with a few large companies with multiple production units and nation wide marketing networks. These companies control much of the market. They have been present in India for several decades and have established several brand names regionally or nationally. These companies aggressively advertise and promote their brands and their corporate identities, and constantly monitor and protect their products and market shares. They are also cash rich, since profit margins are high in this industry.

Beginning in 1992 under liberalized industrial laws, some Indian alcohol companies developed collaborative ties with international corporations. Joint ventures have been established to use local production capacity to manufacture international brands under a technology transfer and licensing system. These joint ventures have served a dual purpose: they have brought international alcohol brands to India, and they have utilized the existing production and marketing strengths of Indian Industry. Hence they have been mutually supportive. Nearly all of the major transnational alcohol companies now have a presence in India and many internationally popular brands of whisky and beer have become available. The upper middle and higher socioeconomic classes now purchase these 'famous' brands locally rather than having to carry these back from trips to other countries or to buy them from illegal importers. The price of these products remains high, but since they carry high social prestige value, there is good demand in this premium range.

With liberalization and globalization, foreign liquor has become freely available. The IMFL and beer industry spends much effort and money to promote and advertise their brands. Since direct advertisement of liquor was
not permitted in the print and electronic media, the industry has found methods to advertise indirectly (Saxena, 1994). Alcohol brands are advertised in the form of same or similarly named other products (e.g. mineral water, soda, and playing cards) made by the same company. The advertisements

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**Compiled by Mr. S D Rajendran, Community Health Cell for the Asia Social Forum, 2nd – 7th January 2003, Hyderabad, India.*

display the alcohol product prominently. In addition, beverage ads have become common on satellite cable television beamed to India from neighboring countries. IMFL and beer producers also financially sponsor major sporting events that attract sustained media attention, including live television coverage of the event. With its new international linkages, the Indian alcohol industry has also got into the entertainment and fashion world. It is now common for a liquor company to sponsor a fashion show or musical event. Hence the Indian IMFL and beer industry has initiated a high level of sustained marketing and promotional activities and these have become especially aggressive in the 1990s.

The Indian alcohol industry produces a large amount of revenue for the government. It has been estimated that direct collections of excise and sales tax are approximately US$ 5 billion per year for the country as a whole. In Karnataka, it is approximately Rs. 2400.00 crores per year. States derive as much as 25% of money from alcohol sales for their annual budget. Besides the generation of legal revenues for the government, the alcohol industry is thought to create an approximately equal sum in "black money" that takes the form of bribes, protection payments and profits from illicit alcohol. This gives the alcohol industry enormous political power and clout, which may be used to help influence and maintain government policies 'beneficial' to the industry but harmful to the people. Studies indicate that the losses borne by household, states and the nation out weigh financial gains.

Table 1: Annual Distilled Spirits Production in India, by Year (April to March)

Year	AMOUNT OF ABSOLUTE ALCOHOL PRODUCED (IN THOUSANDS HECTOLIRES)
1982 – 83	2862.55
1983-84	3104.75
1984—85	3310.64
1985-86	3407.49
1986-87	3204.80
1987-88	3432.48
1988-89	4190.45
1989-90	No data available
1990-91	No data available
1991-92	4895.00
1992-93	3467.00
1993-94	3626.00
1994-95	6056.00
1995-96	7888.04

Source: Alcohol and Public Health in 8 developing countries, WHO, Geneva, 1999.

3. Alcohol – Related Problems

It is probable, given equal amounts of drinking, that developing countries like India experience more problems than developed countries (Saxena, 1997). Among the reasons for this may be such things as a highly skewed distribution of drinkers in the society, the prevalence of nutritional and infectious diseases, economic deprivation, more hazardous and accident-prone physical environments, and lack of any organised support system. Although conclusive scientific evidence for alcohol related health and social problems is lacking for India, there are enough

indications in the available literature to infer that these are substantial. Women's sanghas participating in a women health empowerment training in several districts in Karnataka have consistently said that the biggest problem they face relate to alcohol abuse. Community health groups in different parts of the country also recognize the importance of the problem. The rapid rise in alcohol consumption in recent years has increased the likelihood of further growth of the following health problems in the years to come.

3.1 Health problems include

- Cirrhosis of the liver and premature death
- Cardiomyopathy
- Cancer of the upper gastrointestinal tract
- Pancreatitis
- Cognitive impairment or neuropsychiatric disorders
- Road traffic accidents and injuries
- Nutritional deficiencies and infections
- HIV infections and STD
- Hypertension

3.2 SOCIAL PROBLEMS

Excessive drinking produces a variety of closely inter related social problems in India. For ease of description these have been divided into the following broad categories.

3.2.1 *Violence and Crime*

Violence within and outside the home is frequent in India and a substantial proportion of it is alcohol – related. Wife beating and child abuse under the influence of alcohol are common, and street brawls and group violence happen often after drinking

3.2.2 *Workplace effects*

Heavy drinking affects work performance in a number of negative ways. When compared to their sober counterparts, drinkers are more frequently absent, are less efficient, have more accidents at work, and also show maladjustment with other workers which leads to over all decreased performance.

3.2.3 *Economic Effects*

While alcoholic beverages are less expensive in India, their purchase may still require a substantial portion of a poor persons meager income. With one in three people in India falling below the poverty line, the economic consequences of expenditures on alcohol attain special significance. Besides money spent on alcohol, a heavy drinker also suffers other adverse economic effects. These include reduced wages (because of missed work and lowered efficiency on the job), increased medical expanses for illness and accidents, legal cost of drink-related offences, and decreased eligibility of loans. Most individuals with severe alcohol dependence find it difficult to reduce their expenditure on drink, and hence their families often must do without essential necessities. Although the overall economic

effect of alcohol use at the national level has not been estimated, it is likely that it represents a substantial proportion of India's national income.

3.2.4 *Family Effects*

Excessive drinking by one or more family member results in several negative consequences for others in the family, especially for the wife and children of a male drinker. These effects are particularly serious for poor families. As has been mentioned above, much of the family income may be used to buy alcohol, wages may decline, and the drinker may eventually lose his job. In such situation the wife and children are forced into work, often in low paid, hazardous jobs. Children may be unable to continue their schooling and may also suffer from nutritional deficiencies because there is not enough to eat at home. Wife and child battering are common, which lead to physical and mental trauma. Failure of the man to use contraceptive methods often leads to unwanted pregnancies, further increasing family size. These factors contribute towards greater poverty, often to the point of destitution.

Strong family ties and social disapproval of divorce save many of these families from a formal breakdown, but the prevalence of intermittent or prolonged marital separation, as well as suicide, in heavy drinking families is high. Problems faced by wives of alcoholic men have been studied scientifically by Ganihat et al. (1983), but the many descriptive accounts by the lay press offer more vocal testimony of these phenomena. Wives of alcoholic men show a high degree of depression (Devar et al., 1983) and of suicide (Ponnudurai & Jayakar, 1980).

4. Govt. of India Response

Govt. of India should seriously think about the alarmingly increasing alcohol related problems and work towards developing a clear-cut and comprehensive Alcohol Policy.

The Indian Charter on Alcohol should be adopted with the following principles, which would be agreed upon by all the health ministries of the States:

1. **All people have the right to a family, community and working life protected from accidents, violence and other negative consequences of alcohol consumption.**

2. **All people have the right to valid impartial information and education, starting early in life, on the consequences of alcohol consumption on health, the family and society.**

3. **All the children and adolescents have the right to grow up in an environment protected from the negative consequences of alcohol consumption and, to the extent possible, from the promotion of alcoholic beverages.**

4. **All people with hazardous or harmful alcohol consumption and members of their families have the right to accessible treatment and care.**

5. **All people who do not wish to consume alcohol, or who cannot do so for health or other reasons, have the right to be safeguarded from pressures to drink and be supported in their non – drinking behavior.**

5. National Master Plan

The government of India formed an expert committee in 1986 to develop a comprehensive strategy for reduction of both supply and demand of all substances of abuse, including alcohol. The details of the master plan and its position on alcohol – related issues are not yet available. Again Govt. of India should review the National Master Plan and revise it for up to date condition. This plan should be implemented through Primary Health Centres and through health workers. It should contain the following broad areas:

1. Training to PHC doctors and Health Workers
2. Raise awareness of the effects of alcohol in rural areas
3. Arrange community based de-addiction treatment involving family members and the community
4. Proper after care should be provided with the family and community support
5. Introduce Life Skills programme in high schools to increase the ability of young people to meet the needs and challenges of every day life and avoid high risk behaviors
6. Provide and / or expand meaningful alternatives to alcohol and drug use and increase education, training and networking among community development workers ad organisations.

In monitoring and implementing the above plan, the local NGOs and community action groups should be encouraged to participate fully.

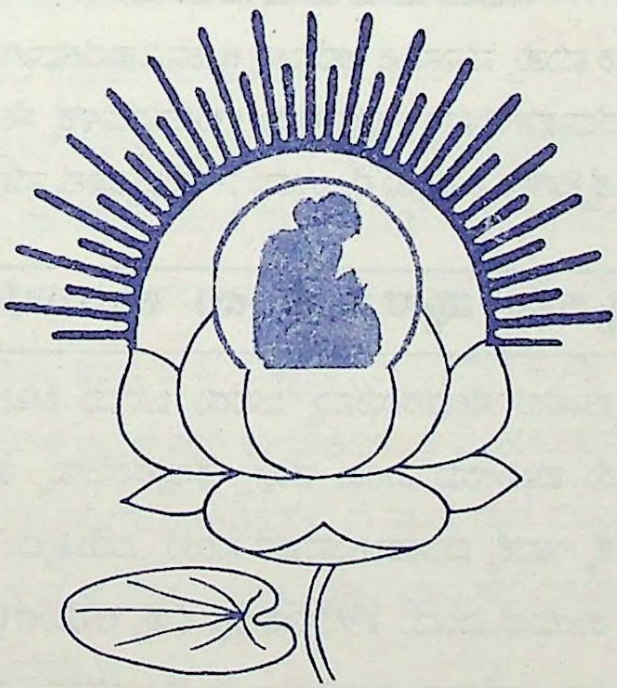
6. Conclusion

Globalisation is based on commercial interests, which want to increase the consumption of alcohol. They promote the expansion of drinking into new social context and situations. Their central perspective is that of the market, seeing developing countries as 'emerging markets'. Drinking is shown as a symbol of 'cosmopolitan outlook'. European and North American life styles are presented glamorously and attractively. We have to counter them. Globalisation has brought in global methods of manufacture, distribution, advertisements and promotion of alcohol consumption. We have to adopt or adopt global strategies to reduce alcohol consumption and its ill effects on the health and social life of our people. While interventions for primary prevention and community health based approaches are required along side medical deaddiction approaches, it is imperative that social movements also address the broader policy aspects and economic underpinnings of the problem.

MH-2

ಹಾರ್ಯೆಕೆ

ಮದ್ಯಪಾನ ದುಶ್ಚಟ ನಿವಾರಣಾ ಮತ್ತು ಆಪ್ತಸಲಹಾ ಕೇಂದ್ರ



ಸಂಪರ್ಕಿಸಬೇಕಾದ ವಿಳಾಸ

ಹಾರ್ಯೆಕೆ

ಮದ್ಯಪಾನ ದುಶ್ಚಟ ನಿವಾರಣಾ

ಮತ್ತು ಆಪ್ತಸಲಹಾಕೇಂದ್ರ

ಅಂಚೆ ಕಛೇರಿ ಮುಂಭಾಗ ದೊಡ್ಡಾಲಹಳ್ಳಿ, ರಸ್ತೆ

ಸಾತನೂರು-562126 ಕನಕಪುರ ತಾಲ್ಲೂಕು

ಬೆಂಗಳೂರು ಗ್ರಾಮಾಂತರ ಜಿಲ್ಲೆ

ದೂರವಾಣಿ : 080-75 51133 / 080-75 23550

ಸಂಸ್ಥೆಯ ಗುರಿ

ಸಮಾಜ ಕಲ್ಯಾಣ ಮತ್ತು ಅಭ್ಯುದಯಕ್ಕೆ ಮಾರಕವಾಗಿರುವ ದುಶ್ಚಟಗಳನ್ನು ನಿವಾರಿಸುವ ಮೂಲಕ ಗ್ರಾಮೀಣ ಮತ್ತು ನಗರ ಪ್ರದೇಶದ ಹಿಂದುಳಿದ, ಅಲ್ಪಸಂಖ್ಯಾತರ ಹಾಗೂ ಬಡ ಜನರ ಏಳಿಗೆಯನ್ನು ಸಾಧಿಸುವುದೇ ನಮ್ಮ ಗುರಿ.

ದುಶ್ಚಟಗಳೆಂದರೇನು?

ದೈಹಿಕ ಮತ್ತು ಮಾನಸಿಕ ಆರೋಗ್ಯಕ್ಕೆ ಹಾನಿಕಾರಕವಾಗಿರುವ ಕೆಲವು ರಾಸಾಯನಿಕ ಪದಾರ್ಥಗಳನ್ನು ನಿರಂತರವಾಗಿ ದೇಹದೊಳಕ್ಕೆ ಸೇರಿಸುತ್ತಿರುವ ಚಟಗಳನ್ನುದುಶ್ಚಟಗಳು ಎನ್ನುತ್ತಾರೆ. ಉದಾ :- ಮದ್ಯಪಾನ, ಮಾದಕ ವಸ್ತು.

ಮದ್ಯಪಾನ ವ್ಯಸನ ಒಂದು ಕಾಯಿಲೆಯೆ?

ಮದ್ಯಪಾನದ ಸೇವನೆಯನ್ನು ಜನರು ಒಂದು ಚಟ, ಅಭ್ಯಾಸ, ಮನರಂಜನೀಯ ಪಾನೀಯ ಎಂಬ ತಪ್ಪು ಕಲ್ಪನೆಯನ್ನು ಹೊಂದಿದ್ದು, ದಿನದಿಂದ ದಿನಕ್ಕೆ ಇದಕ್ಕೆ ದಾಸರಾಗುವವರ ಸಂಖ್ಯೆ ಏರುತ್ತಿದೆ. ಮದ್ಯಪಾನ ವ್ಯಸನ ಒಂದು ಕಾಯಿಲೆ ಎಂದು 1956ರಲ್ಲೇ ವಿಶ್ವ ಆರೋಗ್ಯ ಸಂಸ್ಥೆಯು ಧೃಡಪಡಿಸಿದೆ. ವಿಶ್ವ ಆರೋಗ್ಯ ಸಂಸ್ಥೆಯು ಈ ರೋಗವನ್ನು ಶಾಶ್ವತವಾದ ರೋಗ, ಕೌಟುಂಬಿಕ ರೋಗ, ದೈಹಿಕ ಮತ್ತು ಮಾನಸಿಕ ರೋಗ ಹಾಗೂ ಸಾಮಾಜಿಕ ರೋಗ ಎಂದು ಘೋಷಿಸಿದೆ. ಈ ಮದ್ಯಪಾನ ಸೇವನೆಯಿಂದ ವ್ಯಕ್ತಿಯ ದೇಹ ಮತ್ತು ಮನಸ್ಸು ದುಸ್ಥಿತಿಗೆ ತಲುಪುತ್ತವೆ ಮತ್ತು ಅವರುಗಳು ಆರ್ಥಿಕವಾಗಿ ದುರ್ಬಲರಾಗಿ ಸಾಮಾಜಿಕ ನಿಂದನೆಗೆ ಒಳಗಾಗುತ್ತಾರೆ. ಈ ವ್ಯಸನದಿಂದ ಹಲವಾರು ಕಾಯಿಲೆಗಳು ವ್ಯಕ್ತಿಯಲ್ಲಿ ಕಾಣಿಸಿಕೊಳ್ಳುತ್ತದೆ, ಉದಾ: - ಕಾಮಾಲೆ ರೋಗ, ಲಿವರ್ ಹಾಳಾಗುವುದು, ಫಿಟ್ಸ್, ನಡುಕ, ಊಟ ಸೇರದೆ ಇರುವುದು, ನಿದ್ದೆ ಬಾರದೇ ಇರುವುದು, ಮುಂತಾದವುಗಳು.

ಮದ್ಯ ವ್ಯಸನವನ್ನು ಬಿಡಬಹುದೇ? ಇದಕ್ಕೆ ಚಿಕಿತ್ಸೆ ಇದಯೇ ?

ಹೌದು, ಇದು ಒಂದು ಕಾಯಿಲೆ. ಚಿಕಿತ್ಸೆ ಪಡೆದರೆ ಖಂಡಿತವಾಗಿಯೂ ಬಿಡಬಹುದು. ಅದಕ್ಕೆ ನೀವು ತಿಳಿಯ ಬೇಕಾಗಿರುವುದು ಇಷ್ಟೆ ಆದಷ್ಟು ಬೇಗ ಚಿಕಿತ್ಸಾಕೇಂದ್ರಕ್ಕೆ ಕರೆತರುವುದು.

- ಮದ್ಯವ್ಯಸನಿಗಳೊಂದಿಗೆ ಚರ್ಚೆ, ಸಂವಾದ, ಘರ್ಷಣೆ, ಜಗಳ, ದೇವರ ಕಾರ್ಯ ಯಾವುದೂ ಸಲ್ಲದು.
- ಮದ್ಯವ್ಯಸನಿಗಳನ್ನು ಸಮಾಜ ಘಾತುಕರಂತೆ ಕಾಣಬೇಡಿ. ನಮ್ಮ ನಿಮ್ಮಂತೆಯೇ ಮನುಷ್ಯರು ಎನ್ನುವುದನ್ನು ಮರೆಯಬೇಡಿ.
- ಮದ್ಯವ್ಯಸನ ಬಿಡಲು ಪ್ರಯತ್ನಿಸಿ ವಿಫಲರಾಗಿದ್ದಾರೆಂಬುದನ್ನು ತಿಳಿಯಿರಿ. ಮದ್ಯವ್ಯಸನ ಬಿಟ್ಟ ತಕ್ಷಣ ಅವರಿಗೆ ಕೆಲವು ದೈಹಿಕ ಮತ್ತು ಮಾನಸಿಕ ಸಮಸ್ಯೆಗಳು ಕಾಣಿಸಿಕೊಳ್ಳುತ್ತವೆ ಎಂಬುದನ್ನು ತಿಳಿಯಿರಿ.
- ಮದ್ಯವ್ಯಸನಿಗಳು ಚಿಕಿತ್ಸೆಯಿಲ್ಲದೆ ಮದ್ಯಪಾನ ಬಿಡುತ್ತೇನೆಂದು ಮಾಡುವ ಪ್ರಮಾಣ, ಆಣೆ, ಆತ್ಮ ವಿಶ್ವಾಸದ ಮಾತುಗಳನ್ನು ನಂಬಬೇಡಿ.

ಚಿಕಿತ್ಸಾ ಸೌಲಭ್ಯಗಳು

- ದಾಖಲಾದ ದಿನದಿಂದಲೇ ಚಿಕಿತ್ಸೆ ಪ್ರಾರಂಭ.
- ವೈದ್ಯರಿಂದ ಮತ್ತು ಮನೋವೈದ್ಯರಿಂದ ವಿಚಾರಣೆ.
- ನುರಿತ ಅನುಭವ ಪೂರಿತ ಸಲಹೆಗಾರಿಂದ ಸಮಾಲೋಚನೆ.
- ಉತ್ತಮ ಹಾಸಿಗೆಗಳು, ಊಟ,ತಿಂಡಿ,ಕಾಫಿ, ಟೀ ಸೌಲಭ್ಯಗಳು.
- ಉತ್ತಮ ಮನರಂಜನೀಯ ಸೌಲಭ್ಯಗಳು.

ಬೆಳಗಿನಿಂದ ರಾತ್ರಿಯವರೆಗೆ ನಿರಂತರ ಕಾರ್ಯಕ್ರಮಗಳು

- ಯೋಗ, ಧ್ಯಾನ, ವ್ಯಾಯಾಮ
- ಶಿಕ್ಷಣ ತರಗತಿಗಳು
- ವೃಂದ, ಸಮಾಲೋಚನೆ
- ವ್ಯಕ್ತಿಗತ ಸಮಾಲೋಚನೆ
- ಕೌಟುಂಬಿಕ ಸಮಾಲೋಚನೆ
- ವೃತ್ತಿಮಾರ್ಗದರ್ಶನ
- ಎ.ಎ. ಸಮಾಲೋಚನೆ
- ಮಾತ್ರೆ, ಗುಳಿಗೆ, ಗೃಹ ಔಷಧಿ ನೀಡುವುದು.

ಗೃಹ ಭೇಟಿ

ಈ ಭೇಟಿಯಲ್ಲಿ ನಮ್ಮ ಕಾರ್ಯಕರ್ತರು ಹಲವಾರು ಸಲಹೆ, ಸಮಾಲೋಚನೆಗಳನ್ನು ರೋಗಿಗಳ ಮನೆಗಳಲ್ಲಿ ನಡೆಸಿ ಮಾತ್ರೆಗಳನ್ನು ನೀಡುತ್ತಾರೆ.

ಚಿಕಿತ್ಸೆಯ ನಂತರ ಮತ್ತೇನು ?

ಚಿಕಿತ್ಸೆ ಪಡೆದು ಹಿಂತಿರುಗುವಾಗ ಪ್ರತಿಯೊಬ್ಬರಿಗೂ ಗೃಹ ಔಷಧಿಯನ್ನು ಕೊಡಲಾಗುವುದು. ಏಕೆಂದರೆ ಅವರು ಜೀವನ ಪೂರ್ತಿ ರೋಗಿಗಳಾಗಿರುತ್ತಾರೆ. ಅಲ್ಲದೆ ಲಿವರ್ ಕೂಡ ಹಾಳಾಗಿರುತ್ತದೆ. ಆದ್ದರಿಂದ ನೀಡುವ ಮಾತ್ರೆಗಳನ್ನು ಸರಿಯಾಗಿ ತೆಗೆದು ಕೊಳ್ಳಬೇಕಾಗುತ್ತದೆ. ಅವರು ಸರಿಯಾಗಿ ಮಾತ್ರೆಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳುತ್ತಾರೋ, ಇಲ್ಲವೋ ಎಂಬುದನ್ನು ನೋಡಲು ನಮ್ಮ ಆಸ್ಪತ್ರೆಯಿಂದ ಗೃಹ ಭೇಟಿಯನ್ನು ನಮ್ಮ ಕ್ಷೇತ್ರ ಸಂಯೋಜಕರು ದಿನ ನಿತ್ಯವೂ ನಡೆಸುತ್ತಿರುತ್ತಾರೆ.

ಸಂಸ್ಥೆಯ ಇತರ ಕಾರ್ಯ ಚಟುವಟಿಕೆಗಳು

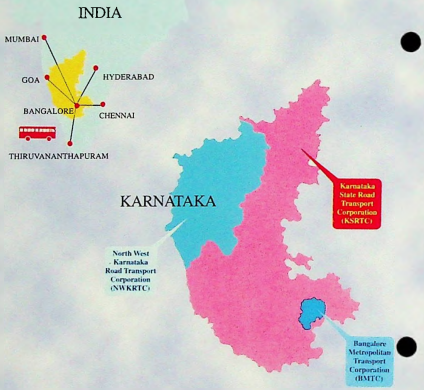
- ಹಾರ್ಮೆಕೆ ಸಂಸ್ಥೆಯು ಮದ್ಯವ್ಯಸನಿಗಳ ಚಿಕಿತ್ಸೆಯ ಜೊತೆಗೆ ಹಲವಾರು ಸಮುದಾಯ ಜಾಗೃತಿ ಕಾರ್ಯಕ್ರಮಗಳನ್ನು ಹಮ್ಮಿಕೊಳ್ಳಲಿದೆ.
- ಗ್ರಾಮೀಣ ಮತ್ತು ನಗರ ಪ್ರದೇಶಗಳಲ್ಲಿ ದುಶ್ಚಟಗಳ ಬಗ್ಗೆ ಜಾಗೃತಿ ಶಿಬಿರಗಳನ್ನು ಹಮ್ಮಿಕೊಳ್ಳಲಿದೆ.
- ಸಮಾಜದ ಮಾರಕ ರೋಗಗಳಾದ, ಏಡ್ಸ್, ಕ್ಷಯ, ಕಾಮಾಲೆ, ಸಕ್ಕರೆ ಕಾಯಿಲೆ ಮುಂತಾದವುಗಳ ಬಗ್ಗೆ ಸಮುದಾಯ ಜಾಗೃತಿಯನ್ನು ಮೂಡಿಸಲಿದೆ.
- ಇತರ ಸಂಘ ಸಂಸ್ಥೆಗಳೊಂದಿಗೆ ಸಹಭಾಗಿತ್ವದಲ್ಲಿಯೂ ಸಮುದಾಯ ಅಭಿವೃದ್ಧಿಗೆ ಶ್ರಮಿಸುತ್ತದೆ.

MH-2.

WAPPA

Workplace Alcohol Prevention Programme and Activity

KARNATAKA STATE ROAD TRANSPORT CORPORATION
BANGALORE



Statutory Warning!

None for the Road and NOT one for the road

should be the policy of transporters towards alcohol consumption notwithstanding their personal predilection

WAPPA to ARMADA

Workplace **A**lcohol **P**revention
Programme and **A**ctivity

to

Association of **R**esource **M**anagers
against **A**lcohol and **D**rug **A**buse

The path from WAPPA to ARMADA is the appropriate path for Industrial enterprises to follow for ensuring Accident Reduction, Increased Productivity, Reduced Absenteeism and Worker reach out programme

CONTENTS

INTRODUCTION

In KSRTC the Workplace Alcohol Prevention Programme and Activity (WAPPA) started on 30th January 1997 essentially as a Worker reach out programme. In view of the unsettled industrial relations obtaining at that time and the stand off between the management and a section of the labour who chose the path of militancy, the need was felt for a programme to reach out to the worker and look after his health and welfare and that of his family. This unique proactive vigilance programme was also aimed at curbing indiscipline at the workplace responsible for losses to the Corporation.

Inauguration of the De-Addiction Centre at Jayanagar

Goals and Objectives

Every enterprise is concerned with increased production, worker welfare, accident prevention in the workplace and improving its profitability and cutting down losses. A transport organisation having a statutory mandate to provide safe, reliable, adequate and economic transport service to the public at large is duty bound to ensure that passengers are transported from one place to another with utmost safety and comfort. The slogan **Driving and Drink Do Not Mix** needs to be enforced strictly in any public transport system.

For the Police and other Enforcement agencies concerned with Road safety, accident free roads mean better traffic regulation, less work for themselves and smooth passage for the road users. For the Industrial enterprises this means reduced monetary losses and compensation to be paid to the affected.

For the public of course this means reduced suffering and misery and physical injury.

At Tavaregere in Raichur District on 13-02-1998, 12 persons died and 35 got injured when a KSRTC bus with POLICE personnel on election duty had a head on collusion with a metador van. The KSRTC driver who also died on the spot, had consumed alcohol when the police party broke journey for food a little while before the accident.

WAPPA – KSRTC Initiative for worker welfare and passenger safety.

Karnataka State Road Transport Corporation (in short KSRTC or the Corporation) is a state owned public sector undertaking dedicated to meet the needs of the travelling people of the South Indian state of Karnataka. Established under the Central Road Transport Corporation Act, 1950 with the prime objective of providing the people of the eighth largest State in the country with economic, reliable, safe and adequate passenger transport facility, the Corporation is currently being restructured into smaller independent corporations for better administrative control. Together, the original monolith transported 6.5 million passengers running 11,227 buses over a total of 3.43 million kilometers every day. The Corporation had a total revenue collection of about Rs.12,080 million in a year. With a total strength of about 70,000 employees on its rolls, the combined Corporation was one of the biggest employers in the State. For ten years the Corporation made losses, year after year, accumulating to over Rs.4,500 Million. Rank indiscipline and militant labour union had stifled most activities in the Corporation and public demands for privatizing the nationalized transport services became strident over the years. The restructuring process was initiated in 1997 to set matters right and saw the birth of Bangalore Metropolitan Transport Corporation for the City of Bangalore and North West Karnataka Road Transport Corporation for the North West region of the State. The North East region too is to get its independent corporation soon. With the restructuring, several new initiatives were also launched to turn the Corporation round. WAPPA was one among them.

Indiscipline needed to be put down with an iron hand to improve the performance in various units of the Corporation covering one Printing Press, three body building workshops, 23 divisions, 124 bus depots and 300 plus bus stations. Alcohol consumption was diagnosed as one of the important factors that caused violent incidents time and again in the depots. **Workplace Alcohol Prevention Programme and Activity** (**WAPPA**), the unique preventive vigilance programme of the Karnataka State Road Transport Corporation was designed to assess the need and implement a programme of Anti Alcohol Policy in the public transport organisation. Formulated as a worker welfare policy to reach out to the workers in an atmosphere where tough measures were being taken to contain the indiscipline, the Workplace Alcohol Prevention Programme helped to win over the workers and their family.

Alcohol abuse among Industrial workers is recognised as a universal phenomenon, which brings down productivity and makes the life of the employees and their family members miserable. In the transport sector, the life of the passengers is made unsafe and unsecured in their journeys in public transport buses. Any number of incidents are available to substantiate the huge loss caused to the Corporation as a result of the employees developing a liking for the liquor especially while on duty.

In a transport organisation like the KSRTC, alcohol abuse among employees has a serious impact on passenger safety. Accidents due to drunken driving brings about avoidable suffering and bad name to the employees, exploitation and inferiority complex to the family members, loss of valuable life, permanent impairment to the passengers and bad image and loss of hard earned revenue to the Corporation. With over 400 fatal accidents in a yearly tally of 2500 accidents involving the death of about 500 persons and injury to over 5000 passengers in a year, the Corporation has claims of compensation running into Rs.3000 million filed against it. Therefore, the KSRTC had to urgently formulate a policy that would venture to arrest all the ill effects of these problems by adopting a humane and welfare oriented de-addiction programme.

Several studies and analysis were undertaken in 1996-97 in the Corporation to understand the full import and magnitude of the problems. It was seen that on an average one employee died in harness every day and the average age of the deceased employee was between 38 – 48 years. Each had a minimum of ten years service left and the Corporation was losing trained manpower every day. The principal causes of death were found to be heart attack, cancer and alcohol related in that order. Stress in the family as well as the work spot, conditions of the transport sector where peak efficiency and performance is demanded all through the work hours with zero tolerance for even a second of relaxation were identified as the causes of the transport workers seeking the bottle to relieve their tensions. The transport workers being forced to be away from their families and homes for long periods soon developed the habits of drinking and casual sex and brought upon themselves attendant consequences as well as causing a huge loss to the Corporation. The consequences of the alcohol habit had various consequences for the welfare of the workers and their families.

Implementation of Anti alcohol Policy started in January 1997 in combination with several other measures before the actual restructuring process to turn the monolith loss making State

Transport Undertaking into profitable public transport organisation came about. WAPPA has had its share in the resultant turn around. WAPPA helped reduce road accidents, curb absenteeism and indiscipline and increase productivity. Astounding results were obtained with the implementation of the overall strategy bringing down yearly losses of the Corporation from Rs.945 Million in 1996-97 to just Rs.26 Million in 1998-99. This proved that a large work force could be motivated to find cost-effective solutions to the perennial problems of absenteeism, indiscipline, low morale, road safety hazards, low productivity and loss of revenues plaguing the Corporation.

Unique features of the Policy of the Corporation

First Time : The Corporation spends Rs 5000 and gives leave to the employee to join a deaddiction centre of his choice and give up the habit.

Second Time: In case of relapse as assessed during a follow up programme, he is referred this time again to a deaddiction centre at his cost.

Third Time: Relapse yet again entails severe disciplinary action which may culminate in dismissal in view of the documentation of the case history done in the first two efforts.

DID YOU KNOW ?
⇨ 20% to 25% of accidents at the workplace are related to Alcohol and Drug Abuse
⇨ On the job fatalities related to Alcohol and Drug accounts for 15% to 16% of all accidents

Evolution of an Anti-Alcohol Policy in KSRTC

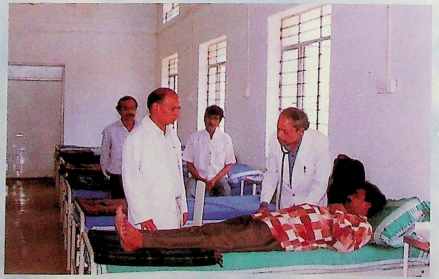
The De-addiction programme of KSRTC titled WAPPA (Workplace Alcohol Prevention Programme & Activity) evolved in 1996-97 necessarily as a worker reach out programme to motivate the workers and cut down the losses of the Corporation and bring about better discipline. WAPPA turned out to be a unique and unparalleled programme of its kind and size. There are numerous institutions both in the private and public sector in India as well as elsewhere in the world with a large labour force and Workplace Alcohol Prevention Programmes. Yet few undertakings have such a wide interaction with the masses and serious consequences for public safety. In India there are 68 other public transport Undertakings/Corporations and KSRTC is easily in the top five for its size of operations. One in thousand in the world is a KSRTC daily bus traveller. KSRTC's de-addiction programme is an example of sustained effort in the transport sector in India that was implemented with technical assistance from the International Labour Organisation and other Non-Governmental Organisations in the State and has proved successful.

Treating alcohol addiction as a health problem of its employees, KSRTC decided to spend time and effort to encourage its employees to give up the bad habit. The Corporation spends upto Rs.5000 from its coffers on each identified employee who is deputed for treatment of alcohol abuse. The problem of alcohol abuse is considered as a disease instead of a self acquired or inflicted malady. This perspective has a chain effect in reducing not only the sufferings and miseries of the employee and his immediate family but also has huge cost benefits to the organisation and ultimately benefits the society at large. Professionally

qualified people are the backbone and strength of this programme in which addicted employees are coaxed to give up their habits and borderline cases are stopped from going over the brink and becoming confirmed alcoholics. The treatment schedule is a mixture of good points of the medical and other models practised world-wide. Emphasis is given on identification of the employee with the Corporation. Employees are categorised into three Zones – RED (Chronic cases), AMBER (Social drinkers) and GREEN (Teetotallers). Separate strategies are devised to address the problem of the workers in each of the three zones.

Employees classifications in zones :

RED (Chronic cases)	: 9,248
AMBER (Social drinkers)	: 14,500
GREEN (Teetotallers)	: 48,252



The employees undergoing treatment at De-addiction centre.

DE-ADDICTION POLICY OF KSRTC APPROVED BY THE BOARD ON 22-12-1998.

POLICY OBJECTIVES:

a) To prevent Alcohol abuse among all employees as a part of the Corporation's commitment to the health and welfare of its employees, operational safety and the environment.

b) To educate the employees on the dangers and consequences of Alcohol abuse specially in the interest of commuter safety and Corporation and assist all employees so desirous to overcome this habit.

GENERAL:

It has been recognised that Alcohol abuse continues to be a major health hazard and safety problem. As a prudent and progressive organisation, the Corporation acknowledges that employees' suffering from Alcohol problems may need medical assistance and it cannot be dealt with by disciplinary measures alone.

The Karnataka State Road Transport Corporation's policy therefore is intended to meet the high level of safety requirements of the organisation to exercise reasonable control over its employees' consumption of Alcohol and to provide a positive approach to employees who seek assistance to overcome Alcoholism.

PREVENTION, EDUCATION AND TRAINING:

Within the scope of the Total Quality Management strategies of KSRTC, emphasis will be made on integrated Alcohol prevention education and training assistance module covering all the employees, supervisors and officers of the Corporation.

REGULATIONS AND CONTROLS:

The employees working in the Organisation at Depots, Workshops and Offices understand that intake of Alcohol can have detrimental effect on relationships, self control, judgment and ability, to make effective decisions and to provide the intended safe transport facility to the travelling public, and being under the influence of Alcohol is a gross misconduct, as a result of which the organisation can initiate disciplinary action on account of i) the consumption of Alcohol or being under its influence within the office or work premises which is prohibited. ii) No individual can report for duty under the influence of Alcohol.

SCOPE OF POLICY:

This policy will apply to all the employees of the Corporation, Supervisors and Officers working at various places like Depots, Divisional Workshops, Bus Stands, Control points and Offices etc. It shall be the endeavor of all Corporation servants to implement and follow this policy faithfully.

ALCOHOL RELATED MEDICAL PROBLEMS:

Being under the influence of alcohol while on duty, would amount to gross misconduct. The organisation would expect that any employee who considers that he/she has a problem should seek help and advise from both the organisation and medical sources.

Such employees will be provided the assistance and medical help. As a policy, the identified employees will be referred for medical treatment and counselling at the cost of the organisation. In the first instance by extending medical advance and leave to the credit of the employee. In case of relapse after the treatment, the employee will be referred for treatment, for a second time at his cost and leave to his credit. Even after the treatment for second time if there is a further relapse, severe disciplinary action will be taken against such employees under the C & D Regulations of the organisation. The KSRTC believes that the loyalty and commitment of its employees depends upon the quality of the life they are offered at work and home and the organisation is committed to creating on alcohol, and drug free environment at the work place. This would be achieved through the involvement of the employees from different Departments, Trade Unions and NGOs.

This process would include:

- raising awareness, education, training.
- assistance, treatment, follow up action.
- prevention programmes among the employees and their families.

As a measure of effective control, it is proposed to provide modern Breath Analysers to all Depots, Workshops and important Bus Stations to check the Drivers, Conductors and other Staff for consumption of alcohol before the resumption of their duties and also by the Line Checking Staff on line during the line checking programme extensively.

EXCEPTIONAL CIRCUMSTANCES:

There may be no variation to the above policy except with written permission of the Board of Directors.

KSRTC Strategies towards employees in different Zones

The employees in the RED Zone with acute alcohol problem were hitherto treated with harsh punishments and inhuman methods, instead of being shown sympathy, they were shown apathy, teased and shouted at. The presence of an employee in the work spot in a drunken condition invariably led to unruly scenes of disturbance at the work spot leading to stoppage of work. The supervisory staff had problems in preventing the situation from escalating. Red Zone strategies include identified employees being sent for detoxication to centres set up by the KSRTC itself at the state headquarter or other centres run by the NGOs. **Employees are sanctioned upto Rs.5,000 as medical expenses and leave to the extent of 30-50 days is sanctioned (even if not to their credit leave not due is sanctioned) to cover the treatment period.**

In the transport sector even social drinking cannot be encouraged and that is why the slogan ***driving and drink do not mix.*** Given the relaxed regulatory mechanisms at work, it has always been difficult to ensure that the staff does not drink while on duty. Presence of employees at the workspot after consumption of alcohol leads to trading of abuses and counter abuses, assaults and other violent incidents over petty matters. Several cases of indiscipline under the influence of alcohol have been reported in the past. Even Depot Managers have been seriously assaulted by employees who had consumed alcohol just before the incidents. At times of strikes, work stoppages and confrontation with the management and even at the time of routine gate meetings, it has been observed that several workers consume alcohol and several others are instigated or financed to consume alcohol to increase the shouting brigade.

As part of AMBER zone strategy, KSRTC has now equippped all the depots with breathanalysers and the supervisory and security staff conduct surprise checks to detect cases of alcoholism on duty not only at the depot or office premises, but also at the places of night halts of the crew and other enroute points in case of night services. Security and other Divisional authorities go in the night to far off places to check out the condition of their employees armed with the breath analysers.

KSRTC recruited over 10000 employees in the last three years. It was necessary to address the employees in the GREEN Zone to stay put in the Green Zone as buses driven under intoxication have resulted in serious road accidents leading to loss of human lives. The bus passengers have been inconvenienced and road safety seriously compromised. The other ill effects of alcoholism among KSRTC employees has been the worsening employee-employer, employee-family and passenger-Corporation relationships. Therefore armed with a formal policy resolution of the Board of Management making drinking on duty a serious misconduct, a concerted programme has been run since 1997 to keep the Greens Green. The results are there for every body to see.

ALCOHOLISM - Is a progressive disease

Any one who drinks can become an Alcoholic

If your best friend in drugs, choose a better one in 2000

For the benefit of other transport operators facing similar problems, the three zonal strategies are summarised below.

Red Zone Strategies

- Treatment at employer's cost
- Vocational rehabilitation – a second opportunity to reform followed by threat of dismissal.
- Reintegration into workplace
- Follow up and relapse prevention
- Use of self help groups
- Incentives and rewards for positive behaviour
- Flexibility-changed shift timing, transfer to place of residence, harnessing support from family, networking with other organi zations providing support

Amber Zone Strategies

- Facilitating self change
- Early identification
- Training staff in communication and assistance skills
- Motivating and encouraging supervisors to understand their roles
- Assessing and improving their awareness of early signs of non-performance
- Working with the families and medical personnel to identify early social and medical problems

Green Zone Strategies

- Awareness programme for all employees
- Consequences of alcoholism on the workplace shared in a simple and understandable manner

- Drama and skits organised by the workers for the workers and their families
- Seminars and workshops at all levels
- Regular review of WAPPA at all other staff meetings
- Inculcating a feeling of collective responsibility and a sense of ownership among workmen.
- Display of WAPPA Policy and printing slogans on tickets and other publicity material including in house magazine
- Awareness of the physical, psychological, social and occupa tional consequences of alcohol, Experts n the area, as well as recovered addicts.
- Stress management programme for various levels
- Information on WAPPA incorporated into all the Orientation/ Induction training programmes organized by the seven in house training institutes.

Recovered employees attending the follow-up programme.

Costing an Accident : The Hassan experience

Hassan is one of the 23 divisions of the Corporation, where WAPPA was implemented with full force and commitment. Actions taken to implement WAPPA sincerely in the division brought rich dividends by decreasing the number of road accidents. As an example of good practice, the Hassan experience is to be commended for the turn around achieved amidst organisational changes, protecting the workers from the social evils and assisting them in performing better. A systematic evaluation was done in 1997 in Hassan to assess the cost of an accident involving a public bus. All costs incurred by the Corporation concerning each and every accident that occurred in 1997 in Hassan was compiled and the average cost of an accident (major, minor or fatal) was worked out. Hassan division has both hilly regions as well as plains. In this division out of a total 539 schedules 278 were operated as urban and semi urban services. The division had 601 buses operating out of eight depots and there are 3367 employees consisting of 1284 drivers, 1032 conductors, 630 mechanical staff and 421 other support staff. In the year 1997 there were 141 accidents involving 36 deaths and 544 injured. One employee and 14 passengers travelling in KSRTC buses died while 15 passengers of other vehicles and six pedestrians were the other fatalities. Six of the injured persons were KSRTC employees and 399 were the bus passengers. 125 passengers of other vehicles involved in the accidents suffered injuries, as did 14 pedestrian road users. It is seen that for every three persons killed four other road users were killed in accidents involving KSRTC buses. In respect of injuries however the ratio was more favourable—for every three persons in the bus injured one person outside the bus was injured. Even so it is evident that in accidents involving the KSRTC buses, the other road user feels the severity of the accident. Of the 141 accidents during the year 17 were fatal 14 major and 110 recorded as minor accidents and it was found that in as many as 115 cases KSRTC driver was found to be at fault. Therefore majority of the accidents and fatalities associated with it and the pain and damages caused to the other road users can be minimised if the KSRTC drivers are made safety conscious and less accident prone. Though no proof is available about the actual no. of KSRTC drivers who were drunk at the time of these 141 accidents, atleast 80 of the drivers involved in these accidents were known to consume alcohol at one time or the other. As many as 90 had been involved in some accidents previously.

To arrive at the average cost of an accident to the Corporation, every item of expenditure incurred in connection with all the 141 accidents of 1997 and their handling was systematically costed in Hassan division. Wherever possible exact figures were taken into account and where actual figures were not yet fully available, approximations were made based on similar cases where such expenses were available. The expenditure involved in an accident begins with the first telephone call made to inform the authorities of the occurrence of the accident. Rushing to the accident spot, arranging to shift the injured to hospitals after first aid and rescue operations at the spot, locating the next of kin to come to take care of the injured or the dead and clearing the traffic jam are priority actions. Lodging police complaints, helping police investigations and visits by senior Corporation officials to the spot for assessing the cause of the accident are next in priority. Arranging for inspection by the motor vehicle inspector and the photographer, and towing the vehicle for repairs are also part of the immediate expenditure. Exgratia payments are made to the next of kin to meet their urgent requirements. Time and effort spent in repairing the vehicle, mandays lost due to injured employees being unavailable

for duty, their medical expenses and revenue loss to the Corporation for the period the vehicle is not available on road cost the Corporation dearly. Legal expenses in connection with the police cases and disciplinary proceedings if any launched against the defaulting employees are no doubt small compared to the compensation claims to be settled in favour of the accident victims. The Corporation paid over Rs 150 Million in a year as compensation and has claims totaling Rs 3000 Million yet to be settled.Hassan division has estimated that the total cost of the 141 accidents in Hassan division worked out to Rs 37.6 Million and cost of each accident works out to Rs 2.88 Lakhs to the Corporation alone.

Accidents involve expenditure towards :

- Accident, Spot IMV inspection, Transportation charges, Attendance and immediate relief : Rs. 2 Lakh
- Damages to vehicle and repair costs: Rs. 1.3 Million
- Loss of vehicle days and resultant revenue: 715 days Rs 4.4 million
- Injured on duty and loss of mandays
- Medical Attendance paid : Rs. 1 Lakh
- Public Compensation claims: Rs 31 million
- Workmen Compensation Claims
- Default case expenses : Rs.1.59 Lakhs
- Court case and lawyers expenses : Rs.3 Lakhs.

Hassan Compilation of all accidents of 1997 but the cost of each accident (Major or Minor) as Rs 2.88 lakhs to KSRTC alone. Costs incurred by others including the Police, Medical departments, Courts etc. may take the cost of each accident to Rs. 4 Lakhs or more. Since there are nearly 20,000 accidents per year, the notional loss due to accidents is equivalent to the entire budget of the Police Department in Karnataka. Each accident prevented is NET SAVING to the State.

Legal Provisions :

The Karnataka State Road Transport Corporation Servants (Conduct and Discipline) Regulations 1971 lays out very clearly the Corporation's stand on the use of intoxicants in the work place or consumption just prior to duty. The rule 7 of the C&D is as under.

A corporation servant shall-

Strictly abide by the law relating to intoxicating drinks or drugs in force in any area in which he may happen to be for the time being ; not be under the influence of any intoxicating drink or drug during the course of his duty and shall also take due care that the performance of his duties at any time is not affected in any way by the influence of any intoxicating drink or drug.

A corporation servant shall not-

Appear in a public place in a state of intoxication; if he is a Driver or Conductor have taken or used any intoxicating drink or drug within eight hours of the commencement of duty or take such drink or drug during the course of duty.

The recovered employee sharing his experience in the followup meeting.

Results achieved

Accident Reduction : The innovative programme to prevent alcohol abuse in the workplace launched with full vigor in 1997 embracing about 70,000 employees in the Corporation has in the last three years resulted in astounding results with accident rates dropping from 2.2 per Million kilometers to 1.7 per Million kilometers.

Losses : The overall losses of the Corporation have been brought down from Rs.94.05 crores in 1996-97 to Rs.2.46 crores in 1998-99 and the details are as under

Year	Losses
1994-95	-69.65
1995-96	-48.01
1996-97	-94.05
1997-98	-62.38
1998-99	-2.46

Strikes : The employee morale has been improved & productivity has gone up and the Industrial relation situation has been improved considerably which can be seen by the strike details and loss of mandays as indicated below:-

Year	No of strikes	Year	No of strikes
1989	8	1985	29
1990	18	1996	7
1991	15	1997	23
1992	12	1998	8
1993	49	1999	1
1994	21		

The Corporation has been awarded the Union Surface Transport Ministry's National Safety Award this year, which includes a Cash Prize of Rs. 5 Lakhs.

The Details of the Accidents/Rate of Accidents **before** and **after** implementing the project are as under :-

Sl. No.	Year	Fatal	Major	Rate of Accident /lakh kms
1	92-93	459	608	0.29
2	93-94	435	723	0.28
3	94-95	408	679	0.28
4	95-96	421	636	0.25
5	96-97	385	507	0.22
6	97-98	351	409	0.19
7	98-99	374	417	0.18

Improvement in attendance after treatment

Out of the identified employees in the Red zone 840 employees of KSRTC, BMTC & NWKRTC have been referred for treatment at various centers and the attendance particulars of 431 employees have been compiled for a period of six months before and six months after the treatment. The improvement of attendance of these employees are as follows:

No of days	No of employees
0 to 10	58
11 to 20	35
21 to 30	34
31 to 40	41
41 to 50	52
51 & above	38

Over 126 employees have remained sober for more than one year.

Conclusion :

The introduction of the de-addiction programme in KSRTC from 1997 emerged as a panacea for the many ill effects. The success of the programme resulted in tremendous change in relations in between the employees, passengers and the Corporation.This programme of the Corporation is a unique Human Resource Development Programme and the same is being implemented in all the divisions and depots of the Corporation. This programme has also resulted in reduction of accident rates. In a year all the money spent on the programme has been got back more than adequately in the form of improvement in productivity and the reduction in number of accidents.

In a labour intensive organisation worker reach out programmes such as WAPPA help both the organisation and worker to improve production, reduce absenteeism, reduction in accident rates and to improve the operations. Though the programme initially met with lot of criticism in KSRTC, concerted efforts of dedicated and committed managers in the project, helped to sustain the programme in the organisation. Evaluation of cost benefits results based on documentation helped to remove the doubts about the sustainability, validity and viability of the programme.

Wholehearted support from affected workers and their families, and the first hand information about the transformation brought at workspot led to the full acceptance of the programme. Constant innovation including computerisation of the records available for independent audit and inspection makes the programme highly credible. When accolades started coming from outside agencies the programme got all round support within the organisation. Seeing the success of programme in KSRTC, the same is now sought to be replicated in other organisations.

KSRTC & ILO

A memorandum of understanding was entered into with International Labour Organisation on 5-11-1997 for technical assistance and cooperation in running the Workplace Alcohol Prevention Programme. The International Labour Organisation has been associated with the Corporation in assisting implementation of the total de-addiction project by extending guidance, technical assistance in the training process, identification of NGO's, treatment process, formulation of action plan, formulation of organisational policy on de-addiction, conducting training programmes of officers, supervisory category etc.

National Project Manager ILO inagurating the training session for the supervisory staff.

Association of Resource Managers against Alcohol and Drug Abuse:

ILO, UNDCP & Ministry of Social Justice and Empowerment, Govt. of India have decided to establish and run projects all over India to mobilise community based organisations and enterprises to reduce and prevent drug abuse. ARMADA India is being formed to take this programme to all parts of India bymutual co-operation among all enterprises associated with this programme.

ADVANTAGES OF ARMADA

- ✓ TO SHARE EXPERIENCES AMONG COMPANIES ON THE IMPLEMENTATION OF PREVENTION AND EARLY ASSISTANCE STRATEGIES - BY THE COMPANY STEERING COMMITTEE MEMBERS THEMSELVES.
- ✓ TO MAKE EXPERIENCES IN PREVENTION AND EARLY ASSISTANCE AVAILABLE TO MEMBER COMPANIES.
- ✓ TO OFFER ASSISTANCE TO MEMBER COMPANIES REGARDING :
  - ❑ POLICY FORMULATION
  - ❑ CAMPAIGNS
  - ❑ PROCEDURES REGARDING REFERRAL OF RED ZONE PERSONNEL
  - ❑ APPROACH TO MANAGEMENT
  - ❑ EVALUATION OF THE PROGRAMME IN COMPANIES
- ✓ TO FOSTER AMONG COMPANIES A LINK
- ❑ TO THE FAMILIES OF WORKERS (TO ARRANGE JOINT FAMILY EVENTS AND FAMILY AND FAMILY SEMINARS)
- ❑ TO THE COMMUNITY (EG: ADOPTING A SCHOOL TO EDUCATE THE WORKERS OF TOMORROW ON THE WORKPLACE'S NORMS REGARDING ALCOHOL AND DRUG ABUSE)
- ❑ TO KEEP THE COST OF PREVENTION PROGRAMMES LOW AND TO OFFER ONGOING TRAINING TO NEW COMPANY STEERING COMMITTEE MEMBERS ON PREVENTION ACTIVITIES.

Programme of Action

An Enterprise adopting WAPPA could follow this programme of action.

- ⇨ Immediate survey, identification and classification of employees according to their propensity towards alcohol consumption specially in the workplace
- ⇨ Compilation of History of each worker including his drinking habits, medical and family problems and work performance and default history
- ⇨ Formulation of firm Policy in the organisation and creating an awareness among all the employees
- ⇨ Forming a Steering Committee under a committed leadership to run the programme with workers involvement
- ⇨ Networking with Non Governmental Organisations and other Experts involved in treating alcoholism / ARMADA
- ⇨ Counselling by dedicated team of doctors, Psychologists and trained counsellers to the workers and their families
- ⇨ Employee Assistance programme for treatment for RED Zone persons
- ⇨ Active Intervention and counselling for those in AMBER Zone / Health care programmes
- ⇨ Keeping the GREENs green by extended education and greater awareness
- ⇨ Making WAPPA/ARMADA a part of proactive vigilance programme and its regular monitoring through usual management information system
- ⇨ Strict follow up of all treated cases and their documentation
- ⇨ Spreading the programme to as many others as possible through audio-visual presentations, street plays, pamphlets, posters and other innovative techniques

RESOURCE PERSONS TO BE CONTACTED FOR FURTHER INFORMATION

1. SRI K.P. SINGH I.A.S. — PH : 2221125
   VICE CHAIRMAN & MANAGING DIRECTOR.

Police Housing Corporation - 5584402

2. SRI R. SRI KUMAR I.P.S. — PH : 2227491
   DIRECTOR (SECURITY & VIGILANCE). FAX : 2235251
   E-mail : r_sri_kumar@hotmail.com

3. SRI K.R. SASHIDHAR I.A.S. — PH : 2223038
   DIRECTOR (PERSONNEL & ENVIRONMENT) FAX : 2235251

4. SRI K.M. AURADHKAR — PH : 2235075
   CHIEF LABOUR WELFARE OFFICER

5. SRI S. MANOHAR — PH : 6347465
   CHIEF MEDICAL OFFICER

6. DR. G.Y. NARAYAN — PH : 6347465
   SENIOR MEDICAL OFFICER

SRI MUKHTIAR SINGH I.A.S., — PH : 4602101 (NEW DELHI)
NATIONAL PROJECT MANAGER FAX : 4602111
INTERNATIONAL LABOUR ORGANISATION, E-mail : delhi@ilodel.org.in
NEW DELHI.

NEW DIRECTIONS BEING TAKEN

- From bottle addiction to BYTE addiction
- All round computerisation effort including followup
- Having kicked the bottle habit - let us now root out corruption

Sri Sageer Ahmed, Hon'ble Transport Minister
Govt. of Karnataka with ILO Evaluation Team

VIEWS OF THE ILO EVALUATION TEAM :

'WAPPA is the most courageous and innovative programme initiated by the dynamic and visionary management conceived and implemented with total sincerity for the exclusive benefit of the employees who alone can ensure the productivity, profitability and excellent image of the corporation. It needs to be extended, expanded and replicated all over India with or without any support from outside'.

Hon'ble Minister of Industries, Govt. of Karnataka, inaugurating the meeting of Association of Resource Managers Against Alcohol and Drug Abuse in Bangalore on 19-02-2000

Meeting of ARMADA at KSRTC Central Office on 19/02/2000

KARNATAKA STATE ROAD TRANSPORT CORPORATION
Central Office, Transport Bhavan,
K.H. Road, Shanthinagar, Bangalore-560 027.
PH.: 91-80-2221321 FAX : 91-80-2237465
E-mail : r_sri_kumar@hotmail.com

MH-2.A.

DRUG-DEPENDENCE

The Blind Alley

The probability of an adolescent succumbing to the temptation of drug is not necessarily related to his knowledge about it

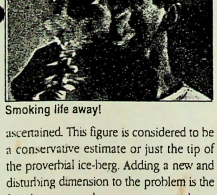
By Naveen Kumar

For thousands of years man has been using substances that have psycho-active effects. In some regions and countries the use of such substances was closely linked to the rituals and prevailing socio-cultural practices. For example, opium, coca-leaf, khat and alcohol have been regularly used in different regions of the world in a variety of ways. The apparent social acceptance of the use of such substances stemmed largely from the fact there was no abuse. Where there was, it was severely ostracized. Society had very clearly drawn the line and there was no question of condoning any abuse. Unfortunately, what we are witnessing today on a global scale is a virtual epidemic of drug abuse. According to the United Nations estimate, there are 15 million drug abusers worldwide.

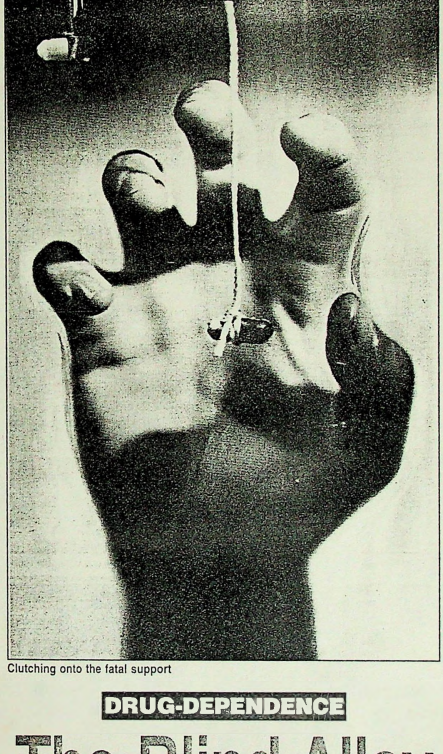
About 2.27 lakh drug addicts were registered with various de-addiction, counselling and after-care centres during 1990-91.

As no census of drug addicts has been undertaken, exact number of drug addicts in the country cannot be ascertained. This figure is considered to be a conservative estimate or just the tip of the proverbial ice-berg. Adding a new and disturbing dimension to the problem is the fact that more and more young people are being affected by what can only be described as the sinister network of global drug cartels. In view of the vulnerability of intravenous drug users to AIDS, drug abuse has now assumed even more dangerous proportions.

Besides, for the people seeking a



Smoking life away!



Clutching onto the fatal support

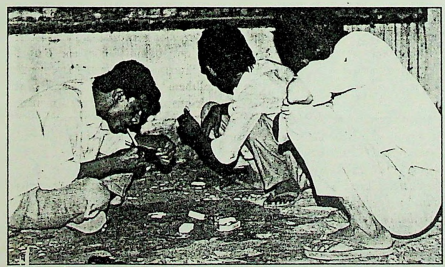
Drug addicts doing yoga exercises as part of their rehabilitation programme

euphoric state of existence, away from the maddening crowd around, the deadly consequences of substance abuse and dependence seem to be less important, if not an illusion. The dark future is washed away, at least for a moment, by the glaring "flash". Hence the rush for a "kuck" of a different life!

The phenomenon of substance abuse is the product of a complex interaction among the individual, substance and the environment.

"Drug-pleasure of a moment, debacle of a life-time", so goes a graffiti on the wall along one of the main thoroughfare of Delhi. Another hoarding exhorts, "say no to drugs the first time, every time". Looking at these hoardings one could sense that the problem of drug-abuse and dependence in our country is not an illusion but a reality that takes a heavy toll of human life.

It has been convincingly argued that people take to drugs because they are offered. It is very rare, at least for illicit drugs, that first drug contacts happen on the initiative of the user. The offer comes normally in circumstances where it is difficult to resist, in a situation which tends to be described not very aptly as social pressure or curiosity. More often, it is in a situation which is conducive to impetuous or precipitous behaviour, a mixture of peer-modelling, risk-taking and going for challenges. Or, it will simply be an offer of the opportunity to join with others in what appears to be the method of extending pleasurable aspects of a conventional recreational situation. So, in spite of being 'anti-drug' he or she evaluates the offer not in terms of the drug-education but in terms of the current situation and the normal rules of behaviour (sociability, enjoyment, reciprocity, keeping one's cool, etc) appropriate in such recreational situations. This is true both for early offers of legal drugs (cigarettes, alcohol) and for later offers of illegal drugs.



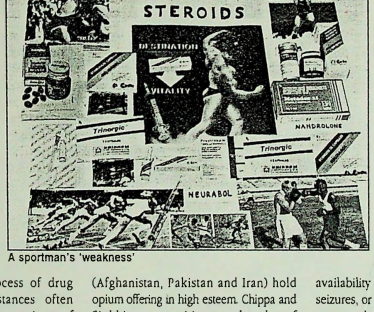
Seeking refuge in smack

Hardly any studies have been undertaken to elicit the circumstantial and emotional details of such situations of drug initiation. It is difficult to see how programmes of 'preventive education' can be effective if so little is known about the behaviour which is to be prevented. "Just say no" is certainly not the full answer. This lack of knowledge on the initiation into drugs has led to the generalisation of the medical model of dependence to drug use in general. We are asking for "cause" of using drugs. There is an evidence to suggest that dependence has a certain medical connotation in that there exists a genetic predisposition towards it.

However, the behaviour of taking a drug or accepting the offer of a drug does seem to resemble a medical condition about as closely as do other behaviours which imply a definite risk to health like skiing or mountaineering. All these behaviours are pleasurable. And in all of them risk-taking is one component of the pleasure. The fact that 'people do things which they enjoy doing' does not need further explanation.

However, it is important to keep in mind that the risk-taking can be fun, and especially so during adolescence. The physiological reactions to fear and fun are very similar. From merely observing hormonal and some other physiological changes we are normally not able to say whether a person is living through a frightening experience, is enjoying a good joke or is experiencing an orgasm. The smooth and virtually timeless undulations between fright and fun can well be observed on the faces of people on a roller coaster. It is therefore not surprising that the probability of an adolescent accepting the offer of a drug is not correlated to his knowledge about drugs. The component of pleasure experienced in the process of drug initiation in many instances often neutralises the unpleasant experience of the drug effect itself. This excitement permits, for example, adolescents to become smokers inspite of the initial unpleasant bitterness and cough provocation by cigarettes. They often have to literally work themselves into regular use. Like skiers, mountaineers or car drivers, drug users are convinced that they can overcome the risk. The facts, however tell a different story. The situation is so alarming in the N-E states that in Imphal there is rarely a home that has not been invaded by the drug menace.

The most widely used drug in these states is the most refined form of heroin, popularly known as *Number 4* among the locals. This brand of heroin is called Number 4 because it is fourth stage of refinement containing as high as 90 to 95 per cent heroin. From where do these drugs come? Immediately the query leads to the fact that National Highway No 39 connects India and Myanmar, a constituent country of the notorious "Golden Triangle" (Myanmar, Laos, Thailand) where opium is "grown like rice". Besides, Manipur shares a 352-km border with Myanmar which is sparsely guarded and it plays a crucial role in the availability of drugs in these areas. Apart from this, people in the districts of Rajasthan, (Barmer, Jodhpur) sharing its border with vicious 'Golden Crescent'



A sportman's 'weakness'

(Afghanistan, Pakistan and Iran) hold opium offering in high esteem. Chippa and Sinhhis communities attach a lot of importance to opium. The high frequency (22 per cent) of opium intake may be attributed to their frequent handling of opium in their professional life.

Famous psychiatrist (AIIMS). Dr Mohan has found in his study of school boys and girls in Delhi, that they use painkillers (49 per cent) followed by alcohol (12.7 per cent), tobacco (6.4 per cent), tranquilizers (3.4 per cent) and less than 5 per cent other drugs like *cannabis amphetamines, barbiturates*, LSD and opium.

School of Social Work reported that drug users are marked by features like insecurity, dependence, frustration and anxiety. Besides, more than three fourth of the respondents were dissatisfied with their family and social situation.

In a study conducted by Malhotra and Murty at the National Institute of Mental Health and Neuro-Sciences, Bangalore, it was found that drug addicts manifest neurotic traits and anti-social behaviour patterns.

Drug peddlers befriend before they offer drug for the first time to their victims, at the pockets in city slums and, in the face of gross apathy from administrative, medical and related agencies, their business flourishes smoothly.

One may well ask what role government could play in the control of drug abuse. Supply reduction is the job of the police and narcotics bureau. Demand reduction is the job of doctors in their treatment centres. To the extent that these administrative agencies can't stop the availability of drugs, let the health care service cure those who become drug addicts, in spite of all supply control efforts.

We have ample evidence in India and abroad that these traditional strategies alone do not work. Law enforcement will at times drastically reduce the availability of illicit drugs by spectacular seizures, or a vigilant narcotics police may prevent the establishment of a criminal distribution network. But such successes do not sustain. Clinics and drug de-addiction centres may cure large numbers of them but the rate of relapse of this vicious cycle has acquired menacing proportions.

Drug abuse is thus closely linked to health care, with health services rendering necessary support. But for successful prevention and care of the disabled and chronically ill, community involvement is necessary. Only people, friends, teachers, media and above all a commoner can prevent others to stay drug free. ■