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CHILD CARE IN PLANTATIONS

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Am/24/9.

Medical Officers' Conference
COONOOR

2nd September 1979



UPASI COMPREHENSIVE LABOUR WELFARE SCHEME

Glenview

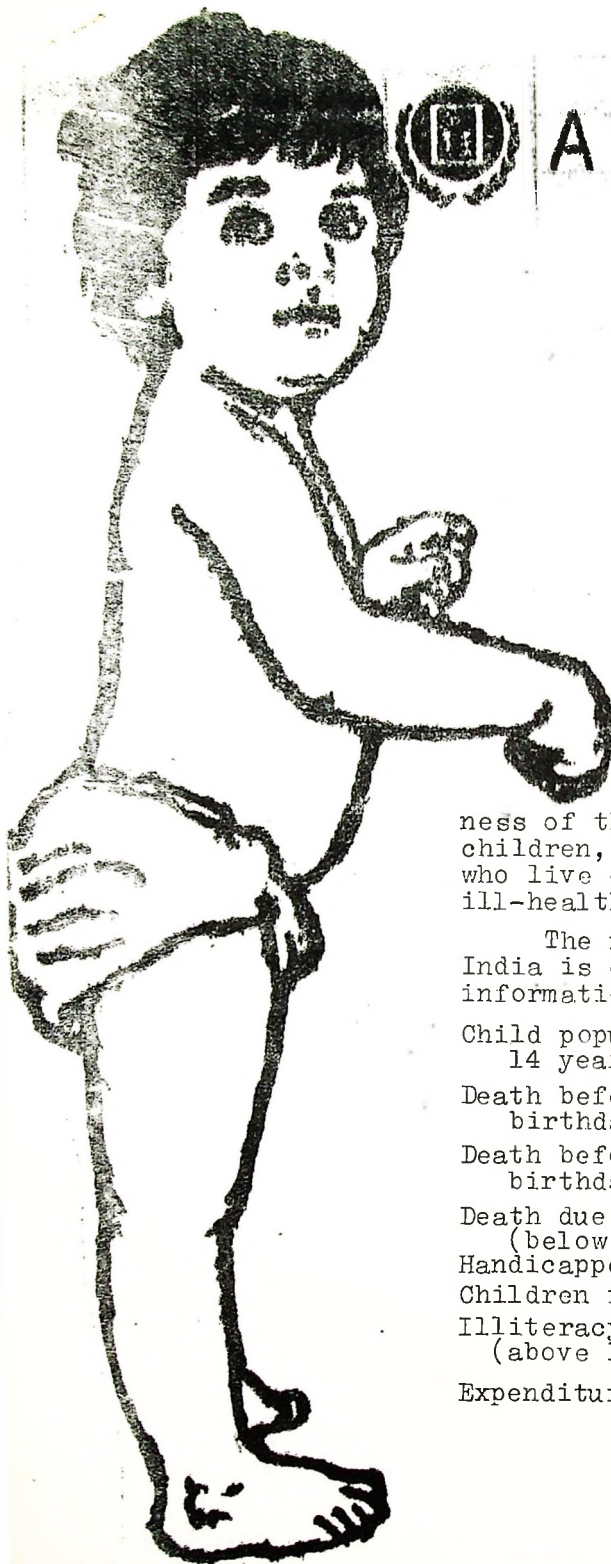
Coonoor-643 101

Nilgiris

| | | |
|-------------|---|--|
| At Birth | : | Child weighs 2500-3000 gms. Length-50.8 cms. or 20 inches Child in flexion attitude with all new born reflexes present |
| 0-4 weeks | : | Flexed attitude, headlag +, Turns from side to side. Doll's eye movement + ' Moro's Stepping, placing and grasp reflexes active. |
| At 4 weeks | : | Lies with legs more extended. Turns head tonic neck posture predominates, headlag + when brought to sitting position, watches mother able to follow objects for a few degrees. |
| At 8 weeks | : | Attitude general extension. Able to raise head slightly, tonic neck posture predominates head lag still persistent. Follows moving object. 180°. Smiles on social contact. Cooing + and listens to voices. |
| At 12 weeks | : | Able to lift head. Head control partial. Reaches towards and misses objects. Bobbing movement of head +, moro reflex-does not persist. Sustained social contact and cooing + |
| At 16 weeks | : | Lifts head and chest, symmetrical posture predominates, hands in mid-line, grasps objects and brings them to mouth. No head lag. Able to sit with trunk support. Able to focus on small objects makes a move for them, laughs, agitated when social contact is broken. Excited at sight of food. |
| At 28 weeks | : | Rolls over. May pivot. Lifts head, sits with pelvic support, bounces actively when made to stand. Reaches out and grasps larger objects. Transfers from hand to hand. Prefers the mother, babbles, responds to changes in emotional content of social contact. |
| At 40 weeks | : | Sits up indefinitely. Back straight. Pulls up to standing position. Crawls. Grasps object with thumb and forefinger. Picks up small objects with assisted pincer movement. Attempt to retrieve dropped toys. Starts saying mamma dadda, enjoys playing hide and seek. Waves bye bye. |
| At one year | : | Walks when held with one hand or with support of furniture. Picks up small objects with unassisted pincer movements and releases object to another person on request. Able to say two to three more words other than mamma or pappu. Able to play with toys and assists in dressing. |

Weight — Weech's Mnemonic

| | | |
|---------------|---|--|
| At birth | : | 7 lbs. |
| 3 - 12 months | : | Age in months + 11 lbs. |
| 1 - 6 years | : | (Age in years \times 5 + 17) |
| 6 - 12 years | : | (Age in years \times 7 + 5) |
| After 3 years | : | It can also be calculated (Age + 3 \times 5) upto 12 years of age. |



A YEAR FOR THE CHILD

"Ultimately, of course, it is the human being that counts and if the human being counts, well, he counts much more as a child than as a grown up." - Jawaharlal Nehru

February 26, 1979.

To

All Members

Dear Sirs,

1979 is the International Year of the Child (IYC). The decision to declare it as such was taken two years back by the United Nations in the pious hope that there will be a greater awareness of the needs of the large number of deprived children, especially in the developing countries, who live - and die - in poverty, malnutrition and ill-health.

The need for a greater concern for children in India is evident from the following statistical information:-

| | |
|--|--|
| Child population (below 14 years) | .. 250 millions or 40% of the population |
| Death before the first birthday | .. 14% of the children born |
| Death before the fifth birthday | .. 40% of the children born |
| Death due to malnutrition (below 5 years) | .. 4 lakhs per year |
| Handicapped children | .. 3 millions |
| Children in employment | .. 30 millions |
| Illiteracy among children (above 10 years) | .. 60% |
| Expenditure on education | .. 50 paise per day per child. |

Although various programmes have been drawn up by the Government for the IYC, they can, at best, touch only the fringe of what is a massively Himalayan task. Voluntary bodies have also joined in the cause of children but it could well be that the IYC - like the International Women's Year - turn out to be yet another listless ritual. We are, however, anxious that this does not happen in the plantation sector.

Apart from the statutory protection afforded to the child population on plantations, the UPASI Comprehensive Labour Welfare Scheme has, as one of its objectives, the CARE OF THE YOUNG and more than one-third of our membership already has an on-going programme for children. While efforts are under way to intensify efforts in this direction, it is our hope that those who do not subscribe to the CLWS will also be able to develop an action programme for the welfare of children on their estates. This could be on the following lines :-

- Estates having dispensary/group hospital could have an extended Maternal & Child Health and Family Welfare Programme.

This will enable the pregnant worker to deliver a healthy child. Further, the improved care will minimise the incidence of disease among children. It should also be possible to put all children under six years on a "Road to Health" chart.

- An Education Programme could be initiated so that mothers are induced to take better care of their children, particularly, in areas of correcting faulty dietetic practices and providing supplementary food to improve the calory intake.

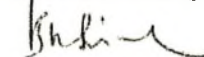
- Activating the Creche by :

- improving attendance
- using it as a centre for health monitoring
- providing supplementary nutritional inputs
- imparting basic education and preparing the children for school
- organising baby contests, games and group activities for the overall development of the child
- identifying handicapped children with a view to rehabilitating them
- early detection of preventable morbidity and organising eye camps, dental camps etc.

These are only our minimal suggestions and they could be acted upon straight away within the infrastructure already available and without any material cost increase.

In viewing the IYC as both "a challenge and an opportunity" let us not overlook the fact that a child development programme on the estates also has a long-term significance. Most of the children of today will be our workers of tomorrow and, as Mr. Maurice Pate, UNICEF's first Executive Director, observed : "Children are the future : they are the seed for the hoped-for harvest of the world - as precious and as rich in promise as the carefully nurtured wheat grains of the knowing farmer."

Yours faithfully,



SECRETARY



UNITED PLANTERS' ASSOCIATION OF SOUTHERN INDIA

POST BOX NO. 11

GLENVIEW

COONOR

CONDENSED FROM "NATIONAL PLAN OF ACTION FOR INTERNATIONAL
YEAR OF THE CHILD 1979".

CHILD HEALTH AND NUTRITION.

According to 1971 census, the child population of India was 42% of total population, infant mortality rate is as high as 122 per 1000 live births, suggesting that lot of effort is still needed to promote child health.

Importance of first 6 year of age of child for its growth and development is well known, there is no doubt that malnutrition either directly or indirectly is the biggest single contributor to child morbidity and mortality in our country; caloric-protein malnutrition has been identified as the crucial problem amongst under sixes.

The prevailing Indian situation in relation to Maternal and Child Health (MCH) reflects on woeful inadequacy of achievement and leaves out immense ground yet to be covered.

The importance of health and nutrition in the overall development of the child should be given greater emphasis. The problem calls for formulating a strategy with full broad goals:

1. Reducing infant and child mortality and morbidity;
2. Reducing maternal mortality;
3. Ensuring adequate maternal and child health care;
4. Preparing of boys, girls for wise parenthood;
5. Wider community education/health and nutrition./on

To realise these goal, a number of specific action programme as suggested below must be initiated, if in existence enhanced in the International Year of the Child:

Immunisation:

Preventive programme to reduce morbidity should be undertaken on a larger scale:

- a) Every newborn child should be protected against smallpox, TB, whooping cough, Diphtheria, Tetanus and polio by immunisation.
- b) Mass immunisation campaign should be organised to cover the most weakest and vulnerable section of the population.

Nutrition:

Measures for detection of early cases of malnutrition must be intensified so as to prevent the cases from reaching a stage of No return.

Domiciliary of malnourished children encourage extended. All children below 6 years of age, especially those below 3 years, of weaker sections should be provided nutritional support in the form of inexpensive and nutritional supplementary foods produced locally.

Large scale distribution of Vitamin 'A', Ferrous Sulphate, Folic Acid should be undertaken to prevent blindness and nutritional anemia among children, pregnant and nursing mothers.

Community resources should be mobilised for raising kitchen gardens, poultry units and dairy farms to improve nutritional status of the community.

Nutritional education:

Mass educational programme should be launched to create health and nutritional awareness. Basic instruction in nutrition education should be imparted through elementary schools. Simple health and nutrition education materials, attractive posters and guide books or manuals should be prepared for teachers and health workers.

Family welfare planning is of crucial importance to child welfare and as such, to be promoted energetically in International Year of the Child.

School Health:

Health services for school children should be integral part of school activities. It should include supplementary nutrition, health check-up, referral services and immunisation.

Environmental Health:

Schools should have safe drinking water, sanitary latrines, adequate light and air and clean surroundings. Sale of unprotected eatables around the school premises and residential areas must be banned and such bans strictly enforced.

Supply of safe drinking water, periodic disinfection of wells, environmental and personal hygiene should be promoted. In order to promote environmental sanitation, the use of Bio-gas plants should be encouraged.

Documentation:

The present system of registration of vital events is truly the 'Achilles Heel' of community health programmes as assessment and evaluation of existing indices in a given area is the foundation on which preventive and curative services is provided.

The proper and complete documentation of all vital events of an individual or family or a community in relationship to the environments helps the doctor to provide the necessary preventive and curative services to the community.

Public health without statistics has been compared to a ship without a compass. Uses of health and vital statistics are several:

- 1) To measure the state of health^{of} a community and identify its health problems, the natures, their sizes and their distribution among the various population groups so that, available health and medical care used with make effect.
- 2) For planning and administration of health services.
- 3) To estimate future needs of the community and to fix suitable targets for achievements for evaluating the progress, success or failure of health programme and services.

sspd.
23.8.1979.

CHILD MENTAL HEALTH AND PSYCHOLOGICAL DEVELOPMENT.

At a time when we are still struggling to deal with high death rates and high physical morbidity in children a concern with psychological development and mental health problems might be thought to be a dispensable luxury. This is not the case.

In the 1st place different aspects of child's development should not be separated artificially, but, integrated into a holistic approach. Improvements in physical health will also aid psychosocial functioning. Thus relief of malnutrition would alleviate the apathy and misery that so often accompanies serious malnourishment.

In the 2nd place, mental health problems deserve attention in their own right. Judged by the criteria of frequency, seriousness, socio-economic consequences, and community concerns, they merit an important place in Health service planning.

Thirdly, unless steps are taken to prevent it from happening, the socio-economic changes taking place in developing countries are likely to bring an increase in the psychosocial problems. Increasing prosperity will not necessarily aid psychosocial development. Action is needed now or its may be too late.

The WHO expert Committee on child mental health and psychosocial development concluded that - a vast amount of knowledge at present remains unused, and that its application might substantially improve the fate of children. Action should be directed to the promotion of mental development and health in children because this would lead to overall developments. Such action would not require vast resources but, mainly the reorientation and co-ordination of activities undertaken by various sectors of community services and by the community as a whole.

Prevalence and need for action:

Persistent and socially handicapping mental disorders affect between 5 and 15% of all children aged 3-15 years. Far from being a luxury in developing countries, concern with childhood mental disorders is especially appropriate in these areas where children under 15 years account for about 40% of the population. Poverty and especially low socio-economic status appear to be associated with a greater incidence of mental disorders. The physical ill effects of poverty such as malnutrition and complications of pregnancy and child birth can themselves lead to increased mental and emotional disorders in children. Conversely the welfare of a country depends on the productivity of its people. Even mild and transient mental disorders in children can do irreparable harm to society.

For example, a child with emotional disorder may drop out of school and become educationally handicapped. In addition to being nonproductive mentally handicapped adults may perform so poorly as parents that the next generation is born into severe deprivation.

Mental health problems prevalent in children may be considered under 3 main groups:

- 1) Emotional disorders (e.g., fears, anxiety, depression, obsessions, hypochondriasis) occur with the same frequency in boys and girls.
- 2) Conduct disorders (poor peer relationship and destructiveness constitute the main features) are significantly more common in boys. Particularly in younger children a sub-group can be identified in which over activity correlates with a serious lack of attention paid to the child.
- 3) Impairments or delays in development are markedly more common in boys than in girls. Developmental disorders of speech and language occur in some 1-5% of children. Regular bedwetting is present in about 3% of children at the age of 10. Reading retardation in children of normal intelligence is found to be present in about 3-10% of children.

Other groups of disorders including epilepsy, organic brain syndromes, sensory impairments, communicable diseases and systemic physical disorders. Not only are they important problems in their own right but they may also occur in association with other mental health problems in children and aggravate their course and consequences.

Course of mental disorders in children:

In so far as most mental disorders in childhood constitute variations from normal psychosocial development rather than disease entities it might be thought that they are of little concern and need not draw on limited service resources. However, this would be a seriously mistaken view. Not only are such disorders a source of considerable suffering to the child but they are also associated with serious social problems and may be forerunners of serious psychiatric and social difficulties in adult life.

The great majority of children with specific delays in speech or language, talk normally by middle or later childhood. However, many of these children go on to have serious difficulties in reading and spelling. In some

cases, these are associated with emotional or conduct disorders. Although most individuals with mild mental retardation have scholastic difficulties in childhood, the great majority hold regular jobs in adulthood and rear ordinary families. On the other hand although they can be taught many useful skills, severely retarded individuals usually remain dependant, and very few of them become gainfully employed or produce offspring. Short-term followup studies of children who have suffered nonaccidental injury suggest that a high proportion have learning and behaviour problems at school. The same is probably true of less severe disturbances of early parent-child relationship.

Individuals who suffer rejection and discord during their childhood are more likely when adult to have both marital problems and difficulties in bringing up their own children. A background of seriously abnormal upbringing is often seen in the case of parents who deliberately injure or grossly neglect their offspring. The two other groups in whom intergenerational continuities are most evident are individuals with conduct and personality disorders and those with mild mental retardation. In both cases there is a higher risk of similar disorders in their children than is the case in the general population.

Factors influencing psychosocial development:-

1. Biological factors:

Individual differences between children are observed from birth onwards and these differences influence psychosocial development. Whereas very few mental disorders in childhood are inherited as such, genetic factors do play a role through their influence on personality and on vulnerability to environmental stress.

In all societies in which it has been studied, brain damage or dysfunction (such as indicated by cerebral palsy or epilepsy) has been found greatly to increase the risk of mental health problems. There is good evidence that brain dysfunction is important in its own right as a cause of mental illness, quite apart from the effects of psychosocial stress or disadvantage with which it may be associated and the intellectual impairment or specific cognitive deficits that may follow it. In addition, it is well known that the great majority of cases of severe mental retardation are a consequence of brain disease or damage. Organic brain dysfunction also plays a part in the genesis or some cases of mild mental retardation, but psychosocial influences are in this instance usually more important.

2. Cognitive factors:

In all literate societies in which the matter has been investigated, emotional and conduct problems have been found to be relatively common in both mentally retarded children and children with specific disorders of learning or language development. It is probable that several different causal processes are involved in these associations. Firstly, the mental disorders may stem from the same basic factors that led to the cognitive impairment i.e., the brain damage or the psychosocial deprivation. Secondly, the risk of poor mental health may stem in part from the experience of school failure. This is suggested both by the pattern of correlations and also by the association between cognitive impairment and mental disorder, which tends to be more marked in relation to children's behaviour at school than at home. In so far as the second mechanism is valid, it implies that the risk of poor mental health need not be inevitable. Alterations in the school environment to aid both the acceptance and functioning of the low-achieving child might bring benefits.

3. Ecological and social factors:

It has been found in developed countries that mental health problems in children are more common among those living in inner cities than among those in towns or rural areas. It appears that this is due in large part to the higher rates of family difficulties in inner city areas. Children's psychosocial functioning can be more frequently impaired in the cities because more children live in discordant unhappy homes or have depressed or deviant parents. However, relatively little is known about the specific features of city life that have this adverse effect on family functioning. It is not urbanization per se because many medium-sized towns have rates of disorder comparable to those in rural areas; nor is it a function of population density or of industrialization, because industrial areas may have relatively low rates of psychosocial disorder.

Migration is not necessarily associated with any increase in mental health problems, and population movement may sometimes involve psychosocial benefits. Nevertheless, in many countries migrants are at a disadvantage and there are mental health risks associated with major psychosocial change. The variables involved have not been well studied but it seems likely that the risks stem in part from the break-up of families and tribal or other community support systems, the lack of adequate child-care facilities which follows from this break-up, the alienation of migrants in the community or discrimination against them, and the very poor living conditions in the slums and shanty towns in which many migrants have to live.

4. Patterns of upbringing:

It has been well demonstrated in studies in both developing and developed nations that variations in the psychosocial development of children are strongly associated with qualities of parent-child interaction. In particular, it is known that when children are reared in homes where there is a lack of conversational interchange, where parents do not interact positively with their children, and where there is a lack of play opportunities, the development of language, intelligence, and scholastic skills is likely to be impaired. Similar associations have been found in children reared in institutions and it may be concluded that it is a causal connection. This pattern of inadequate parent-child interaction is sometimes discussed in terms of "lack of stimulation". Whereas it is true that children need stimulation, both experimental and clinical studies show that it is active experiences and interchange that matter. Parents need to be helped to understand that it is not enough to do things to their children; they must do things with them. Verbal stimulation is provided by talking with the child and not by turning up the volume of the radio. Similarly, it is helpful to provide toys, but children may also need to be encouraged to create their own play opportunities. There is good evidence from numerous studies in a variety of societies that there is a very substantially increased rate of mental health problems in children who are unwanted or who experience rejection, hostility, or serious family discord.

At one time it was thought that even temporary separation of a child from his parents created a serious psychosocial hazard. It is now clear that these arguments were to some extent mistaken. Children do need continuous relationships with a small number of parent figures but brief separations need not necessarily disrupt these relationships. Good-quality day-care and a working mother are both compatible with secure parent-child relationships.

Studies in many different countries on parental deviance have shown that children brought up by criminal or mentally disordered parents show an increased rate of mental health problems. While genetic factors may play some part in this association, it is clear that the hazards are in considerable part a function of the family discord and disturbed patterns of child-rearing that often accompany parental mental disorder.

In the past, great attention has been paid to certain patterns of child care such as timing of weaning and toilet-training, methods of discipline, and the like. It is now clear that these concerns were misplaced. Within quite broad limits the timing and mechanics of these aspects of child care are of little psychosocial concern. On the other hand, the quality of care (in terms of sensitivity and responsiveness to the child's needs), the relationship between the child and those who look after him, and the consistency

and efficiency of child-rearing methods are ^{of} some importance. Markedly inconsistent patterns of punishment, repressive or brutal handling, and a lack of concern all increase the risk of mental health problems.

It has already been noted that day-care for young children need not interfere with harmonious and secure parent-child relationships. It should be added that good-quality day-care can have positive psychosocial benefits, particularly in the case of children from poor or disordered homes. Obviously much is likely to depend on the quality of care provided, but so far there is no good evidence to suggest that either day-care or preschool education has such a lasting benefit to children's mental health that it could be advocated on these grounds alone.

5. Ameliorating influences and factors leading to
POSITIVE development:

The possible protective factors include the following:

1) Sex. For reasons that are ill understood (but which are probably both biological and social), girls appear less susceptible to most psychosocial stresses in childhood.

2) Temperament. Children with an adaptable temperament are generally more resilient in the face of deprivation and disadvantage.

3) Isolated nature of the stress. It appears that surprisingly little damage is done by even chronic stresses provided they occur in isolation (e.g., parental mental disorder in harmonious homes without social disadvantage). However, multiple stresses interact to potentiate the psychosocial damage.

4) Coping skills. One study found that children who were used to brief happy separation experiences (such as staying with friends or relatives) coped better with the stress of hospital admission. Presumably, children can acquire coping skills relevant to other stresses.

5) A good relationship with one parent. It has been shown that the risks to mental health that stem from an upbringing in a discordant, unhappy home are appreciably reduced if the child is able to maintain a good relationship with one parent. It is possible although not yet demonstrated that good relationships with other relatives in extended an family might have a similar beneficial effect.

6) Success or good experiences outside the home. It seems that good schooling can do something to mitigate the effects of a poor home environment.

7) Improved family circumstances. When a child is reared in a disturbed, quarrelsome family there is a substantial risk to mental health. However, if family circumstances improve and the later years of childhood are spent in harmony the risk is appreciably reduced.

These findings clearly indicate that there is much potential for preventing mental health problems and that a major improvement in children's psychosocial development is possible through actions that are feasible in most countries.

Some widely held beliefs about what can harm children are not supported by facts:

1) Antenatal disorders tend to be more common in inner city areas. However, it does not seem to be urbanization per se that interferes with children's psychosocial functioning, because many mediumsize towns have rates of disorder as low as those of rural areas.

2) Children are not inevitably damaged by receiving day care outside the home or by having mothers who go out to work, as was mistakenly argued by some previous WHO expert groups. It has been demonstrated that good quality day care can have positive psychosocial benefits especially for children from seriously disadvantaged or disordered homes.

3) Mental problems and abnormalities are persist across generation lines, but discontinuity is nevertheless more frequent than continuity. For example, about 5 out of 6 children reared by mentally retarded parents are of normal intelligence. And most individuals from unhappy discordant homes later go on to make stable marriages. Mental disorder is not a function of population density, although it does seem to be associated with serious overcrowding in dwellings.

Preventive measures:-

1. General health measures:

There is good evidence that children with any form of organic brain dysfunction (e.g., epilepsy, cerebral palsy, or encephalitis) or with mental retardation have a much increased rate of psychosocial and mental health problems. Accordingly, any measures that substantially reduce the rate of these conditions should also have mental health benefits. There is good evidence that children with chronic physical handicaps (of a kind that do not involve brain damage) also have a somewhat increased rate of mental health problems, although the increase is less than is the case with brain disorders. Thus, any improvements in the general physical health and wellbeing of children should also lead to gains in psychosocial functioning.

a) Improved maternal and obstetric care. This is important because perinatal complications may lead to cerebral

palsy and other forms of brain pathology, which in turn predispose to mental health problems.

b) Improved nutrition. Prolonged malnutrition in early childhood remains an important main or contributing cause of death in developing countries. In those who survive, it not only impedes physical growth but can impair mental development, especially if coupled with a poor psychosocial environment. In this connexion the encouragement of breast-feeding and concern with the psychosocial aspects of physical care are important factors.

c) Effective immunization programmes. In many parts of the world poliomyelitis, tuberculous meningitis, and tetanus (and to an important extent measles, rubella, and pertussis) remain both killers and causes of chronic mental and physical handicaps. Immunization greatly reduces both mortality and morbidity.

d) Reduction of accidents. Accidents are a principal cause of death in children. Survivors are often left with brain damage from head injuries. Steps to reduce accidents such as the imposition of speed limits on roads, road-sense training, and the provision of adequate play space for children are therefore important.

e) Improved physical and social conditions. The control of most infectious disease depends at least as much on better living conditions and pure water supplies as it does on improved medical treatment.

f) Better care of the chronically handicapped. Many of the adverse psychosocial sequelae to chronic physical handicaps are indirect consequences of the social restrictions, educational difficulties, inappropriate medical treatments, and stigmas associated with crippling conditions. Better support of families and improved medical, social, and educational care of chronically handicapped children would aid their psychosocial development and reduce mental health problems.

Social Welfare Measures:-

1. The extensive evidence showing associations between a wide variety of psychosocial stresses or disadvantages and mental health problems has already been mentioned. There is good reason to suppose that many of these associations reflect causal influences; hence measures to reduce these stresses or to foster development would improve children's psychosocial functioning.

2. The avoidance of unstable and discontinuous patterns of parenting:

Studies have indicated that children who repeatedly go in and out of children's homes or foster homes, or who frequently change from one home to another, or who live with their own parents in a severely unstable and unsettled family environment have a high rate of difficulties in their psychosocial development. There is also evidence that children from stressful, discordant homes are more likely to develop normally if they are adopted than if they remain in a disturbed family environment. Accordingly, it is suggested that, in the case of young children whose parents seem unlikely to be able to look after them, an early decision should be taken with respect to adoption or long-term fostering.

3. Improved conditions in day-care facilities:

It has been shown that children's development is influenced by their experiences in day-care centres or nursery schools. It is important that there be continuity in the staff who take care of the children so that each child has only a limited number of people who look after him. However, there should be sufficient staff to provide them with play, talk and activities, and the centre should provide an adequate range of experiences and learning opportunities. An interaction between the day-care centre and the parents will help to increase parental skills and confidence.

4. Improved conditions in hospitals and other residential institutions, together with a reduction in admissions:

Children's behaviour and development varies systematically according to the social and psychological conditions in the institutions in which they live for either short or long periods of time. There is some evidence that a change in institutional conditions (as shown by hospital studies) leads to changes in children's mental health functioning. Parents should be strongly encouraged to visit children daily in hospital and be able to do so without restrictions; in the case of very young children it should also be possible for a parent to be admitted with the child.

It has been found that repeated hospital admission are associated with an increased risk of psychosocial and mental health problems in later childhood. The risk is probably much greater among children already experiencing psychosocial stress or disadvantage. The implication is that every effort should be made to treat ill children as outpatients or day-patients unless there are strong reasons for admitting them to hospital. Emphasis must therefore be placed on the development of community and outpatient facilities.

5. Reduction in the number of unwanted births:

Children whose conception is unwanted by their parents may subsequently become loved, but this is less likely to occur than in the case of children whose births were desired from the beginning. Children who are rejected are more likely than other children to have mental health problems. Clearly, it is no straightforward matter to take action to reduce the number of unwanted births. The free availability of family planning services is one important step but it is not enough. The individuals most likely to produce unwanted children (young single people and people with many personal and social problems) are just those least likely to avail themselves of services. Usually it is not that they wish to conceive a child or that they have ethical objections to contraception but rather that their difficulty in planning a family is merely one facet of a general problem in planning all aspects of their lives. Accordingly, family planning must form part of a wider community service that is educational in the broadest sense.

6. Enhanced public awareness of children's needs:

It is essential that high priority be given to the health and welfare of children and thus to the task of rearing children. Children learn through play, conversation, and experiences, on all of which emotional and intellectual growth depends. Parents need to appreciate the distress often experienced by very young children when away from home and to recognize that their clinging on return is a normal response. Sensitivity to differences in individual children's needs is important at all ages, as is the requirement for the right balance of emotional support and social control. It is also important for parents to realize that children are influenced by the emotional climate in the home. These are but a few of the general principles that people should know about children's needs so that they will be better able to respond appropriately as parents, as teachers, or indeed as anyone who comes into contact with young people.

sspd.
29.8.1979.

MORBIDITY AND MORTALITY AMONG THE 0-6 YEAR OLDS.

By

Dr.(Mrs.) Sulochana Unnikrishnan,
UPASI.

INTRODUCTION:

The plantation community may be divided into 3 broad categories - the workers, the medical personnel and the management. It was felt that a study into the patterns of morbidity and mortality among the vulnerable 0-6 population here would, apart from being appropriate in 1979, be of value to all three sections mentioned above - the working mother whose suffering is both mental and financial, the doctor, one-fourth of his outpatients being made up of worried mothers and wailing children and the manager, concerned about his mounting absenteeism figures. Viewed from a larger perspective, there is no exaggerating the importance of such a study as we are talking about our human resources whose health has a bearing on the economic and social growth of the country. Plantations due to their isolated situation and closed nature of the community would perhaps be the best suited for a study of this type. Hardly any deaths go unreported here; when someone is ill and convinced of the need of medical care he invariably has to go to the estate hospital. The very same reasons make plantations ideal for a 'total health care' approach.

METHODOLOGY:

Statistics from 4 estates were collected for this purpose. These are estates where an intensified total health care delivery system is being adopted for more than a year now. The reasons for giving them special attention were their high mortality and birth rates as well as increased incidence of water-borne illnesses when compared to other estates in the area. The 0-6 population of these 4 estates was 1389 in 1978 making up 18.55% of a population of 7489. 1976 was taken as the base year for this paper. Statistics for 1978 were collected for comparison. The objective of this effort may said to be two fold:-

- 1) To study the pattern of morbidity and mortality in the 0-6 year olds.
- 2) To find out the impact, if any, of the total health care approach on the incidence of illnesses and deaths among these children.

Morbidity figures were taken out agewise from the out-patient registers of the respective hospitals. The diagnoses and age entered therein by the peripheral medical staff was accepted. When the complaints were charted out they were found to fall into the major categories of respiratory tract infections, common water-borne illnesses and fevers including influenza. Upper and Lower respiratory infections, bronchitis, bronchopneumonia, cold and cough were put down under respiratory tract infections. In water-borne illnesses, diarrhoea, dysentery and gastro-enteritis were included. One single case each of Typhoid and Hepatitis has been recorded which was left out from this category. The diagnoses given as Pyrexia of Unknown Origin, influenza and fever come under fevers.

Mortality and still-births statistics are from death and still-birth reports.

Population figures for 1976 are from a survey conducted by UPASI early that year. Those for 1978 are from estate office records except the 0-6 population which is from an actual enumeration of the CARE feeding beneficiaries.

The outpatient figures for 3 months were further analysed in detail to study the pattern of repetition in these cases. It was felt that it would be extremely difficult to do such a study for the full year and so this was done for the months of March, April and May for diarrhoeas because previous statistics have shown their incidence to be maximum during this period. Similarly for respiratory complaints, May, June and July figures were taken.

OBSERVATIONS:

Out-patients:

Population and morbidity. (Table 1.)

| Year. | Population | | | Morbidity | | |
|-------|------------|---------------|--------------------|--------------------------------|-----------------------|-------------------------------|
| | Total | 0-6 yr. olds. | % of 0-6 yr. olds. | Total out-patients (New cases) | No. of 0-6 year olds. | % of 0-6 yr. olds in the O.P. |
| 1976 | 8369 | 1469 | 17.55 | 26383 | 3443 | 13.05 |
| 1978 | 7489 | 1389 | 18.55 | 27609 | 3227 | 11.69 |

The total population as well as the number of 0-6 year olds seem to have decreased in these two years.

The total number of new cases treated has gone up in 1978, from 26383 to 27609 in spite of which the 0-6 year olds treated has fallen from 3443 to 3227.

Frequency of attending outpatients among
0-6 year olds for a period of 3 months
1976 and 1978.

| COMPLAINT. | 1976 | | 1978 | |
|-------------------------------|---------------------|---------------------------|---------------------|---------------------------|
| | Number of children. | No. of times OP attended. | Number of children. | No. of times OP attended. |
| Diarrhoea | 181 | 1 | 135 | 1 |
| & | 13 | 2 | 13 | 2 |
| Gastro- | Nil | 3 | 6 | 3 |
| enteritis. | 2 | 4 | - | 4 |
| Respiratory tract infections. | 214 | 1 | 198 | 1 |
| | 16 | 2 | 23 | 2 |
| | 4 | 3 | 6 | 3 |
| | 2 | 4 | 1 | 4 |

The 1976 figures show that out of 196 cases who came for diarrhoeas, 13 had come twice and 2 had come 4 times for the same complaint within a period of 3 months. The total for 1978 has come down from 196 to 154 out of which 13 children came twice, and 6 children 3 times. A similar pattern is seen in the case of respiratory tract infections also.

Table 3* shows that respiratory tract infections, water-borne illnesses and fevers in that order were the major complaints that brought the 0-6 year olds to the hospitals. This was more so in the younger age group. These illnesses covered 88.84% and 67.09% of the morbidity in 1976 and 1978 respectively. Sex did not seem to make any significant difference in the morbidity rates. (* see Annexure)

Figure A gives the major illnesses as percentage of the total outpatients for each age group. It is seen that in 1978, respiratory tract infections were maximum in the infants and the incidence decreased as the children grew older. Water-borne illnesses are more in the 1-2 year olds and incidence of fevers seems to increase with age. In general the same trend was observed in 1976 also.

A comparison of incidence of the 3 major illnesses in 1976 and 1978 is shown in figure B. Reduction is observed in all cases, the most significant fall from 789 cases to 480 cases have been observed in the incidence of waterborne illnesses in the 1-6 year olds. Fevers did not show any set pattern.

Table 4 in the annexure gives the breakdown of table 3 as to the actual number of cases in the different categories. This shows that water-borne illnesses in the age group under study have shown the maximum reduction, from 1058 to 711. Respiratory tract infections have fallen from 1224 to 1086 and fevers from 585 to 513.

Inpatients:

Inpatient figures for both the years could be obtained for two estates only. Details are given in table 5. Total 0-6 year inpatients for 1976 was 128 and for 1978 it was 95. They formed 18.34 and 14.03 of the total inpatients for these years. Here too the major categories remained respiratory tract infections, diarrhoeas and fevers - 31.58% and 8.52% and 13.68% of the total 0-6 year admissions in 1978. (Table 5 annexure). Admissions for measles is observed to have gone up from 4 in 1976 to 38 in 1978 (Table 5 annexure).

Figure C shows that admissions for respiratory tract infections were lower in 1978 than in 1976. - 46 and 30 respectively; similarly the number of cases admitted for diarrhoeas has come down from 20 to 8 and fevers from 32 to 13.

Table 5 also shown that there were no admissions for dysentery in 1978 as against 7 in 1976. So too there have been no cases of malnutrition, Kwashiorkor or deficiencies, which category had 4 inpatients in 1976.

MORTALITY:

There were a total of 49 deaths in the age group under study in 1976; this figure has come down to 23 in 1978. (Table 8 in annexure and Figure D.)

Deaths in the first one week of life remained the same, 6, in both the years. Neonatal mortality after the 1st week seems to have come down from 9 to 5. Maximum reduction is observed after 1 year of age - from 29 in 1976 to 7 in 1978.

The number of still births were 11 in 1976 and 8 in 1978 (Table 8 annexure). Thus the number of perinatal deaths was 17 in 1976 and 14 in 1978.

0-6 year deaths/medical attention received.

(Table 9.)

| Year. | Male. | | Female. | | Total deaths. |
|-------|-------------|-----------|-------------|-----------|---------------|
| | Inpatients. | OP/lives. | Inpatients. | OP/lives. | |
| 1976 | 16 | 9 | 12 | 12 | 49 |
| 1978 | 8 | 3 | 10 | 2 | 23 |

Table 9 shows that majority of deaths took place in the hospitals in 1978, 18 out of 23. No significant difference is observed in the matter of hospitalisation as between the sexes. Mortality is almost equal in males and females - 25 and 24 in 1976 and 11 and 12 in 1978.

Prematurity and bronchopneumonia remained the major killers in the 0-6 year olds and does not show any reduction. There were 9 cases of prematurity for both years; 10 of bronchopneumonia in 1976 and 9 in 1978. There have been no deaths due to gastro enteritis, malnutrition, measles, enteric fever and neonatal infection or septicaemia in 1978 as against 20 deaths due to the above causes in 1976 (Table 8.)

DISCUSSION:

While collecting statistics for this paper the need for a standardised documentation system was keenly felt. With the present system of record keeping a great amount of time and effort are needed in extracting the relevant information. The value of such information in planning, accomplishing and evaluating one's health care delivery methods justifies the extra effort put in on documentation.

It would be relevant at this juncture to summarise the extra inputs covered by the term total health care, made on these estates. These were :-

1. Health education through link workers and health talks.
2. Supplementary CARE feeding programme in the creches.
3. Each child being put on a road to health card and regular monitoring of his/her weight.
4. Almost 100% coverage of these children with prophylactic Vitamin 'A'.
5. Supply of ferrous and folic acid tablets to selected children and all maternity cases.
6. Attention to water supplies and fly control measures.
7. A more purposeful MCH programme including immunisation.
8. Last but perhaps the most important, very good co-operation in the matter of all these between the management and the medical department.

The total population as well as that of the 0-6 year olds seem to have reduced in these 2 years. It may be argued that one cannot go by the total population figures which can fluctuate with the retireals and recruitment of labour. Yet a decrease in the 0-6 year olds may be of importance in view of the fall in the birth rate from 44.65 in 1976 to 39.30 in 1978. The birth rate on these estates

is still very much above the district average which is only 30.75/1000 population.

The percentage of 0-6 year olds in the out-patient has decreased which may be attributed to better health in this age group.

Respiratory tract infections, diarrhoeas and fevers make up about two-third of the illnesses in the age group, under study. Incidence of all three categories has decreased, the most significant reduction having been observed in diarrhoeas. One of the reasons may be the fact that out of a total of 11 divisions on these estates 6 divisions have chlorinated water supply now. The total outpatients for water-borne illnesses on these estates came down from 3727 in 1976 to 2845 in 1978. Apart from this the educational efforts have resulted in improved hygiene and sanitation and an awareness in this regard among the workers. The maximum reduction is observed in the 2-6 year olds who incidentally must be the category who benefit most from the supplementary feeding programmes.

Respiratory tract infections charted agewise show a clear downward curve in both years. When the outpatient figures were taken out it was seen that there were hardly any cases in the 0-1 month group and so these were included in the 0-1 age group. The highest incidence was in the 1 month to 1 year group in the case of outpatients as well as inpatients. This was so far both 1976 and 1978. The possible reason may be the low natural resistance in these infants. There is a reduction in respiratory tract infections also from 1976 to 1978 both in the outpatients and the inpatients. Another finding is that more cases of measles were admitted in 1978 but there were no deaths due to measles. In 1976, 4 deaths in the 0-6 age group have been recorded as due to measles with bronchopneumonia. Perhaps prophylactic Vitamin 'A' and increased resistance due to supplementary feeding have contributed to the reduction in respiratory tract infections. One class for the link workers covered measles and its management; perhaps this has contributed to the increased admissions of measles cases to the hospital.

MORTALITY:

The number of deaths when charted agewise showed a downward trend in 1978. In 1976 there seemed to be more deaths in the 8 days to 1 month group than in the 1st week of life; the number of deaths in the 1st week of life has remained the same in both years. The reduction in perinatal mortality is due to the decrease in still births and so cannot be taken as significant. Prematurity and bronchopneumonia remained the major killers in both years. No reduction in this has been achieved. In the district,

prematurity accounted for 52.59% and bronchopneumonia for 24.14% of the 1978 infant deaths. The comparable figures for these 4 estates are 56.25 and 31.25 respectively.

Prematurity may be due to anaemia in the pregnant women, sexually transmitted diseases or lack of nutritional food during the last trimester of pregnancy. In view of these possibilities routine VDRL test is done for all antenatal cases these days. However, the figures point to the need for better and more purposeful antenatal and post-natal care, promotion of hospital deliveries and education of mothers. Incidence of bronchopneumonia may be aggravated by climatic conditions, state of repair of creches and lines, lactating mothers working in the fields during monsoon coming in their wet cumblies and clothes and feeding the babies etc. The chill a child may catch sitting and playing on the damp cement floor of the creches was behind our minds when 'attalais' or wooden platforms were provided in the creches for the children to sit and play and sleep.

From 1976 to 1978 there has been a good fall in the 8 days to 1 month mortalities. A noticeable reduction is observed in deaths after 1 year. This fact is reflected in the inpatient figures also where apart from the peak produced in the 2-3 age group due to the measles admissions, the total number of deaths have recorded a reduction in all age groups from 1 to 6. More significant is the fact that there were no deaths due to malnutrition, neonatal infections, gastroenteritis and measles. In general this shows better resistance and better health in the 0-6 year olds towards which the supplementary feeding, Vitamin 'A', health education, improved sanitation and waste disposal all could have contributed. No particular difference was noticed as between the sexes in the matter of hospitalisation when ill. A study of the mortality in the two sexes did not reveal anything significant.

CONCLUSION:

A study was made into the mortality and morbidity patterns of the 0-6 year olds on 4 estates. Morbidity and mortality cannot and should not be viewed as isolated entities divorced from their back ground and setting. In a developing country like ours nutrition, education, housing, hygienic disposal of waste, water supplies, all spell health, in different ways. This is the rock on which the more affluent nations have built their medical edifice. It would be folly to envisage vertical growth without a solid base. This country of ours with its myriad problems cannot afford to have its educated intelligent minority wearing blinkers. In an industry illhealth also means, substandard work, absenteeism, and mandays lost. So when we say that in 1978, 3227 0-6 year olds came to the outpatients it also means atleast double that number of working hours lost, mothers who did not get ration money for the week, whose earnings were reduced and lowered productivity.

There are two ways of dealing with morbidity and mortality rates. One is by stocking the hospital with more and more drugs and waiting for the patient to come to you. This will discharge our duty to the individual patient, but does not deal with the other problems attendant upon a sick child in the outpatients in an industrial setting. The other and more reasonable way would be to fight the battle against illnesses, making use of all available weapons in our armoury - in other words, the total health care approach. This latter has been basically accepted by the plantations as a philosophy, and it is to this end that the management, medical department and the UPASI are striving. Such a system of total health care includes a more purposeful ante-natal and post-natal programme with set targets and evaluation, educational efforts through link workers and health talks, regular monitoring of health of the children through under-five clinics and weight cards, the creche development programme, attention to water supplies etc. Here a streamlined system of documentation goes a long way in identification of priority areas, early interventions and improving the quality of services in general.

We find that the morbidity on these 4 estates have decreased from 1976 to 1978; mortality has halved from 49 to 23. Comparable figures for other estates, other states or all India were not available. Yet, higher mortality in the neighbourhood is no consolation nor should it make us complacent. The planning and efforts required to eliminate the last preventable death, within the existing constraints and resources are worthy of the highest intellect, the noblest heart.

ACKNOWLEDGEMENT:

1. Thanks to Dr.(Mrs.)V. Rahamathullah for her guidelines and help in preparing the paper.
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High Risk Infants.

These are babies with low birth weight, babies born after difficult labour, twins, babies with jaundice and babies with other neonatal complications. These high risk babies contribute largely to the perinatal, neonatal and infant mortality rates.

Low birth weight:

By International agreement any baby weighing less than 2,500 grams at birth is called a low birth weight baby. If the birth weight is not specified a live born infant with a period of gestation of less than 37 weeks is considered as equivalent of an immature infant. As it is difficult to know how immature a baby is WHO (1961) recommended that the concept of prematurity be replaced by that of low birth weight for all infants weighing less than 2,500 grams.

Importance:

A low birth weight baby is a 'Paediatric priority' because the baby has less chances of survival than babies weighing over 2,500 grams. Half of all perinatal and 1/3rd of all infants deaths are due to low birth weight. Further prematurity contributes to mental and physical handicaps especially in those who have not received expert neonatal care.

Causes:

The most important causes as far as India is concerned are maternal malnutrition and anaemia.

Other causes are,

1. Maternal - Syphilis, heart disease, toxæmias of pregnancy, diabetes, accidental injury, uterine malformations, ovarian tumors, incompetence of the cervix etc.
2. Foetal - Twins, hydramnios.
3. Placental - Premature separation and placental insufficiency, cephalopelvic disproportion.

Social:

1. Physical labour - Women who work late in pregnancy tend to have babies of lower birth weight than those who have no occupation during pregnancy or who stop work during the early weeks.

2. Smoking - Average weight babies of smoking mothers is about 250 grams less than babies born of mothers who do not.
3. Others - Poor housing, low economic status, repeated births are all associated with premature births.

Small for date babies:

Low birth weight babies may be immature or small for the period of gestation. Small for date babies are those having a birth weight below the 10th percentile for their gestational age. These are the babies where intra uterine growth has been abnormally low and whose birth weight is therefore low for the gestational age at which they are born. The advantage of this classification is that it will include most of the babies at risk associated with intra uterine growth failure.

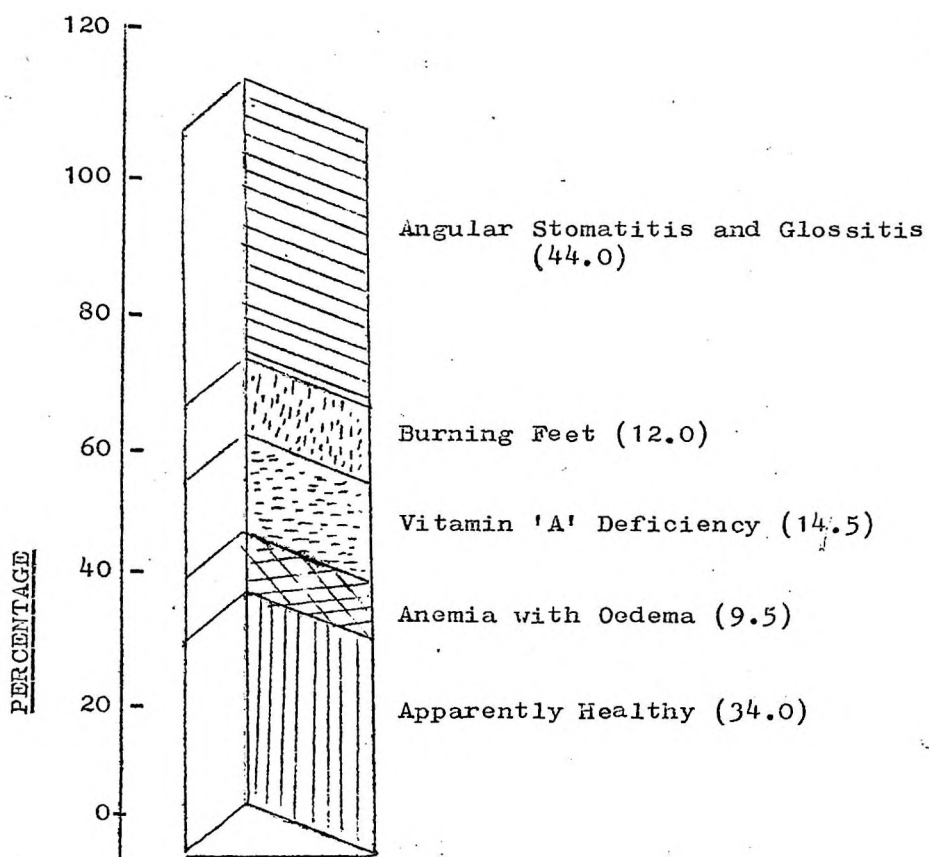
Causes of slow intra uterine growth:

1. Foetal - Poor growth potential - foetal malformations, intra uterine infections (rubella) and some forms of dwarfism where growth failure begins before birth.
2. Maternal - Severe malnutrition, severe toxemia or multiple pregnancy. There is a group of mothers who rather consistently bear small for date babies.

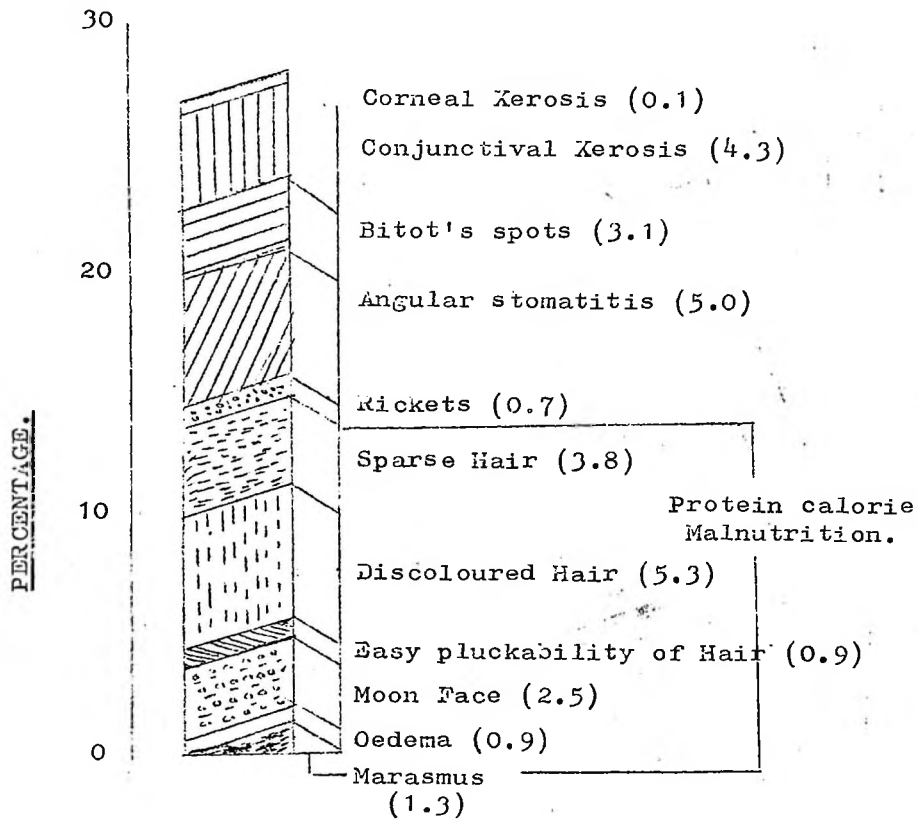
Measurement techniques.

Measurements of fetal mortality and perinatal mortality have frequently been carried out in which the populations used in the denominators consisted of either a count of live births or a count of total births, i.e., live births plus fetal deaths of 28 or more weeks of gestation (still births). To avoid confusion it has been urged that the terminology "fetal death ratio" and "perinatal mortality ratio" be reserved for those calculations in which the

population base is a count of live births; the terms "fetal death rate" and "perinatal mortality rate" should be used when the denominator is a count of live births plus fetal deaths of 28 or more weeks of gestation (still births). The rate is - in most cases - to be preferred to the ratio. While there are other useful measures of fetal and perinatal mortality, they may be given special names in order to avoid difficulties of interpretation.



Nutritional Deficiency Signs in Pregnant Women.
Percentage prevalence.



Nutritional Deficiency Signs in Preschool Children. Percentage Prevalence.

Developmental Milestones in Indian and British Infants.

| | Indian. | British. |
|----------------------------------|----------|----------|
| <u>HOLDING THE HEAD:</u> | | |
| Chin slightly off ground | 4 Weeks. | 4 Weeks. |
| Chin at 45° angle from ground | 8 " | 8 " |
| Chin at 45-90° angle from ground | 12 " | 12 " |
| Chin at 90° angle from ground | 16 " | 16 " |
| Sitting with support | 20.5 " | 28 " |
| Sitting without support | 26 " | 36 " |
| Standing with support | 22 " | 24 " |
| Crawling on belly | 26.3 " | 40 " |
| Crawling on knees | 32.4 " | 44 " |
| Walking with support | 45.1 " | 48 " |
| Walking without support | 52.4 " | 55 " |

Mental and Social Development.

| | |
|--------------|--|
| 2nd month. | : The baby smiles to himself; utters cooing sounds. |
| 4th month. | : He laughs. |
| 6th month. | : Cries on provocation, e.g., removal of a toy from his hand. |
| 7th month. | : Plays with feet to mouth. |
| 10th month. | : Starts to utter a few words - "ma-ma", "da-da". |
| 1 year. | : Enjoys simple tricks and games. |
| 1-1/2 years. | : He imitates. Likes to play with toys. He can speak a few definite words. |
| 2 years. | : Points to eye, nose and mouth. He desires to feed himself. He says "no-no" to everything. |
| 3 years. | : He feeds himself. Does not wet bed. He likes to be near other children. He begins to enjoy group play. He arranges his toys. He likes to dress himself. Much laughter with play. |
| 4 years. | : He repeats digits - one, two, three, etc. |
| 5 years. | : He repeats sentences; he loves his home.; he fears dark, ghosts; he wants to assume responsibility. |
| 6 years. | : He is restless; active all the time; he develops likes and dislikes. |
| 7-8 years. | : He does not like discipline; he forms groups. |


The Recommended temperatures and duration of storage for various vaccines along the cold chain.

| Vaccine | Central Store | Transport to region. | Regional Store. | Transport to district. | District Store. | Mobile team |
|------------------------|----------------------------|----------------------|-------------------------|------------------------|-------------------------|------------------------|
| Polio | 2 years at - 20°C | -20°C to +4°C | 3 months at - 20°C | -20°C to + 4°C | 1 month at +4 to +8°C | 1 Week at +4 to +8°C |
| BCG | 1 year at +4 to +8°C | +4 to +8°C | 3 months at + 4 to +8°C | + 4 to + 8°C | 1 month at + 4 to + 8°C | 1 Week at + 4 to + 8°C |
| DPT (never freeze) | 1 1/2 years at +4 to + 8°C | +4 to +8°C | 3 months at +4 to +8°C | +4 to +8°C | 1 month at +4 to +8°C | 1 Week at +4 to + 8°C |
| Tetanus (never freeze) | 1 1/2 years at +4 to + 8°C | +4 to +8°C | 3 months at +4 to +8°C | +4 to +8°C | 1 month at +4 to +8°C | 1 Week at +4 to + 8°C |
| Smallpox | 3 years at +4 to +8°C | +4 to +8°C | 3 months at +4 to +8°C | +4 to +8°C | 1 month at +4 to +8°C | 4 Weeks at +4 to + 8°C |

REMEMBER: Never use the vaccine after the expiry date on the packet; periods are quoted from the date of issue.

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V A C C I N E S

| Name of vaccine. | Effectiveness (% protection) | Period of immunity. | Minimal age to be given. | Reactions. | Complications. |
|--|---------------------------------|----------------------------------|---------------------------------|---|---|
| (1) | (2) | (3) | (4) | (5) | (6) |
| 1. Tetanus Toxoid | 100% | Long lasting > 10 Years | Antenatal and at any age. | Slight painful local reaction for 12-24 hours. | 1. Sterile injection abscess 2. Provocation Polio- myelitis |
| 2. DT (Diphtheria & Tetanus Toxoid) | - | Long lasting > 10 years | 1 month | -do- | -do- |
| 3. DPT (triple vaccine) | - | Pertussis approx. 4 years. | 1 month | Painfull local rea- ction for 24 hours. Mild to moderate fever for 12-24 hours. | 1. Local sterile inj- ection abscess (esp. with adsorbed vaccine under one month of age). 2. Occasional 'colla- pse' 1-3 hours after injection (pallor, sweat, slow pulse), uni- versal recovery after an hour or two. 3. Persistent crying for 12 hours. 4. Febrile convul- sions. 5. Encephalitis-very rare (1/million). 6. Provocation polio- myelitis. Vaccine associated paraly- tic polio in OPV recipients and their contacts 1/million (CDC* 1973). |
| 4. Poliomyelitis (oral) | 90% | Probably life long | 1 month | None  | |

| (1) | (2) | (3) | (4) | (5) | (6) |
|-------------|--------|---|-----------|--|--|
| 5. Smallpox | 90% | High level for 3 years & waning immunity for 10 years or more. | Birth | Primary vaccination Vesicle 3 days Pustule for 1 week Fever for 1-2 days. Scab at 2 weeks. Scar at 1 month. | 1. Vaccinia gangrenosa 2. Eczema vaccinatum 3. Generalised vaccinia 4. Post vaccinial encephalitis, 3/million in primary vaccination (Lane 1969). |
| 6. BCG | 70-80% | - | Birth | Papule 1 week Ulcer 2-6 weeks Healing by 12 weeks. | 1. Accelerated reaction in tuberculin sensitive individuals. 2. Deep ulcer with secondary infection. 3. Lymphadenitis - local 4. Keloid. |
| 7. Measles | 95% | Up to date more than 10 years follow-up-children still have high level of immunity. | 6 months. | 1. Slight to moderate fever from 6th-10th day. 2. Occasional rash. | Vaccine associated encephalitis (1/million.)(Encephalitis following natural measles is 1/1000.) |
| 8. Rubella | 95% | 5 years follow-up shows. Shows very little decline in antibody titre (CDC 1972) | 1 year. | 1. Rash 2. Lymphadenopathy 3. Arthralgia & transient arthritis. | Vaccine associated encephalitis. 1/million (CDC, 1971). |
| 9. Mumps | 95% | 6 years observations show very little decline in antibody titres (CDC 1974). | 1 year | 1. Occasional mild fever. 2. Parotitis. 3. Rash. | Vaccine associated neurological complication 1/million. (Encephalitis following natural mumps 2-4/1000) (CDC, 1974). |

* Centre for Disease Control, USA.

Contraindications:

1. Severe febrile illnesses

Vaccination of persons with severe febrile illness should generally be deferred until they have recovered. This is to avoid superimposing adverse side effects from the vaccine on the underlying illness or mistakenly identifying a complication of the illness as having been caused by the vaccine. Minor illnesses such as mild upper respiratory infections do not necessarily preclude vaccination.

2. Altered immunity

The virus replication following administration of live, attenuated virus vaccines can be potentiated by immune deficiency diseases and by the suppressed immune responses that occur with leukemia, lymphoma, or generalized malignancy or with therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation. Patients with such conditions should not be given live attenuated virus vaccines.

3. Poliomyelitis epidemic

During an outbreak of poliomyelitis, inoculation with multiple 'depot' antigens should be deferred. When an outbreak of diphtheria, pertussis, typhoid or smallpox occurs simultaneously with poliomyelitis, the appropriate specific inoculation should not be deferred. Fluid antigen rather than 'depot' antigen should then be used.

4. Skin disorders

Eczema and other forms of chronic dermatitis in the individual to be vaccinated or in a household contact are contraindications to smallpox vaccination. An inquiry into the family history for skin disorders should always take place before vaccination. If vaccination is required for an individual with dermatitis because of potential exposure in an endemic or infected area, Vaccinia Immune Globulin (VIG) should be administered to the vaccinee at the time of vaccination. VIG will not prevent successful vaccination. If there is real need to vaccinate an individual who may thus create a hazard for a household contact with dermatitis, consideration should be given to separating the vaccinee from his contact until the vaccination lesion has healed.

5. Pregnancy

On grounds of a theoretical risk to the developing fetus, live, attenuated virus vaccines are not generally given to pregnant women. With some of these antigens, particularly live, attenuated rubella vaccine, pregnancy is a contraindication to vaccination. With others, however, if there is a substantial risk of exposure to natural infection, vaccine should generally be given, taking whatever specific precautions are indicated—for example, giving vaccinia immune globulin with smallpox vaccine.

IRON DEFICIENCY ANAEMIA IN CHILDHOOD.

This is the commonest of the deficiency disorders in childhood. It is the commonest of anaemias in childhood, characterised by a greater proportional lowering of haemoglobin concentration than of the red cell count.

The prevalence of this deficiency is related to certain basic aspects of iron metabolism and nutrition. The body of the newborn infant contains about 0.5 gms. of iron in contrast to the iron content of the adult which is estimated at 5.0 gms. To make up this, 4.5 gms. discrepancy an average of 0.8 mg. of iron must be absorbed each day during the first 15 years of life. To this requirement, additional iron is necessary to balance normal losses through excretion. Hence, to maintain a positive iron balance in childhood, 0.8-1.5 mg. must be absorbed each day. As only about 10% of the dietary iron is absorbed, a diet containing 8-15 mg. of iron is necessary for optimum nutrition. During the first years of life because small quantities of iron rich foods are taken, it is often difficult to attain these amounts. At best, the infant is in a precarious situation with respect to iron. For this reason, the diet should include iron rich foodstuff.

Should the diet become inadequate or should abnormal external blood loss occur, anaemias ensue rapidly.

Etiology:

The etiological factors may act singly or more commonly in combination.

0-3 months - Physiological anaemia of infancy. Anaemia of prematurity. Poor ante-natal storage of iron.

3-6 months - Low birth weight and significant perinatal haemorrhage are associated with decreased neonatal Hb. mass.

Anaemia due to dietary deficiency is uncommon below the age of 6 months.

6-24 months - Dietary deficiency is the commonest cause.

After 24 months - Blood loss is a common cause. This may be due to occult bleeding due to a lesion of Gut e.g., Peptic ulcer, Meckel's diverticulum, Polyp or Haemangioma. In South India, it is mainly due to Helminthiasis and malnutrition. Anaemia could also be caused by chronic intestinal blood loss induced by exposure to a heat labile protein in whole cow's milk. This can be prevented by using heated or evaporated milk or a milk substitute.

Clinical Features:

Pallor is frequently all that is seen even with Hb levels as low as 5-6 gms./100 ml. With Hb levels less than this, child may have anorexia, irritability or

lethargy and frequent infections. There may be Tachycardia with a soft systolic murmur heard precordially. Not infrequently the heart may be dilated. The spleen may be palpable 1-2 cms. below the costal margin in 10-15% of the cases. The child may be obese or under weight, with other evidences of under nutrition. Plica is sometimes prominent. In long standing cases, widening of the diploe of the skull may be expected.

Laboratory data:

R.B.Cs. show hypochromia, microcytosis, anisocytosis and mild poikilocytosis. There may be leukopenia.

Bone marrow shows normoblastic, hyperplasia with increased pronormoblast and decreased or absent stainable iron. There will be low serum iron levels (less than 50 micro grams/100 ml.), and elevated iron binding capacity (over 350 micro grams/100 ml.) and a low saturation of transferrin protein (less than 16%).

Differential Diagnosis:

1. Iron deficiency must be differentiated from other hypo chromic microcytic anaemias.
2. In lead poisoning the red cells are morphologically similar but coarse basophillic stippling (punctate basophillic) is prominent.
3. The blood changes of Thalassaemia Trait resemble those of iron deficiency. But characteristic alterations in the levels of Hg b 42 and Hg b F are usually present whereas they are not in iron deficiency.
4. Thalassaemia major with its pronounced erythroblastosis and haemolytic component should present, no diagnostic confusion.

Treatment:-

The principles of treatment are -

- (a) correction of anaemia by transfusion and iron deficiency by iron medication.
- (b) elimination of associated ethiological factors.

The regular response of iron deficiency anaemia to adequate amounts of iron is an important diagnostic as well as therapeutic feature. Oral administration of simple ferrous salts (sulphates, gluconate, ferrous fumarate) provides inexpensive and satisfactory therapy.

There is no evidence that addition of any trace metal, vitamin or other haematinic substance significantly increases the response to simple ferrous salts.

A daily dose of 6 mg./kg. body weight of elemental iron in three divided doses provides an optimal amount of iron for the stimulated bone marrow to utilise.

Ascorbic acid containing juices aid in the absorption of iron from Gut. Iron medications are best administered 1 hour before meals. As foodstuff containing phytic acid interferes with absorption of iron.

Place of parenteral Iron:

The response to parenteral iron is no more rapid or more complete than that obtained with proper administration of oral iron. Parenteral iron can be given :-

- 1) When oral preparations are not tolerated.
- 2) When it is doubted that the oral preparation is faithfully taken over the required therapeutic period.

Place of transfusion:

Transfusion is indicated in severe anaemia or associated medical complications. Infections and anorexia would make indications for transfusion more immediate. Packed cells are preferred if increase in concentration of haemoglobin is immediately necessary. Along with transfusion, supplementation with iron medication is indicated.

ACUTE INFECTIVE DIARRHOEA IN INFANCY AND CHILDHOOD.

Infective diarrhoea is the major cause of death in infants and young children in developing countries but also produces considerable morbidity in developed societies. The success of the possibility of isolating the pathogen from stools of children, even with sophisticated laboratory techniques is only 30%.

Etiology

- 1) Enteric infections: (Virus, bacteria e.g., salmonella, shiga bacillus, staph, protozoa, cholera and amoebic dysentery).
- 2) Parenteral infection: Otitis media, pneumonia, pyelitis.
- 3) Dietary: Overfeeding, quantitatively or qualitatively, for example too much carbohydrate or fat.
- 4) Oral antibiotics may induce diarrhoea by direct action on the GIT or by inhibiting the normal bacterial flora and permitting overgrowth of pathogens.
- 5) Enzyme deficiencies, either primary or secondary to diarrhoea from any other cause.
- 6) Enteritis or colitis.
- 7) Gastrointestinal allergy.
- 8) Endocrine

Hypoadrenalism
Hyperthyroidism
- 9) Psychogenic

^mEmotional upset
Excitement
- 10) Vitamin deficiency - Pellagra.
- 11) Chemical poisoning - Arsenic sodium fluoride cadmium, zinc.
- 12) Food poisoning - Some types of mushroom newly sprouted potatoes.

The problems of proving the viral, etiology have been major as many viruses normally inhabitate the alimentary tract of healthy children who are free of diarrhoea and very few studies relating epidemics of diarrhoea to specific viruses have been documented. Recently, virus like particles have been identified by electron microscopy in epithelial

cells taken at intestinal biopsy and subsequently in stool concentrates of infants with diarrhoea, in the absence of a recognised bacterial pathogen. Such viral particles are absent from control infants without diarrhoea, and disappear when the diarrhoea subsides. The particular virus involved is currently termed the 'ROTA VIRUS'. Thus, although a viral aetiology of infective enteritis has not yet been proved, it does seem at the present time to be most likely.

Clinical Features:

Disinterest in feeding; associated with or without vomiting, is the initial clue. Diarrhoea rapidly follows and in severe cases upto 30 stools in a day. The stools are typically fluid, green in colour and have a typical 'musty' odour. Mucus is usually present. Blood - may or may not be occasionally, high fever and a convulsion precede the diarrhoea by a few hours - this is more common in the case of shigella infection.

The effects of these symptoms on the child are, loss of fluid and electrolyte imbalance. Sodium, potassium, chloride and bicarbonate are the particular electrolytes lost in the diarrhoeal fluid, the concentration or osmolality of which may approximate that of extra cellular fluid. Initially this fluid loss is from the extra cellular compartment because it is the most accessible. Later, equilibrium with the intra cellular compartment takes place so that, the cells share in the dehydration. As the loss increases, the plasma volume becomes depleted; resulting in poor tissue perfusion with tissue hypoxia. Oliguria and nitrogen retention follow because of poor renal perfusion and an associated metabolic acidosis, the result of anaerobic metabolism, is invariably present.

Physical examination reveals signs of dehydration, and if severe, with clinically recognisable acidosis. Dehydration is manifested clinically by restlessness, apathy, decreased skin turgor, depressed anterior fontenellae and dry mucus membrane in the earlier stages. If progressive, the pulse becomes rapid with poor pulse volume, cyanosed and signs of peripheral circulatory failure (extremities - cold, clammy and cyanosed) are present. Acidosis is clinically recognised by deep sighing respirations (Kussmauls' respiration); the breathe may have a sickly sweet odour. This combination of dehydration, circulatory failure and acidosis with oliguria will be fatal if not reversed rapidly by replacement therapy. Many other febrile illness present with vomiting and a few loose stools e.g., meningitis, pneumonia, septicaemia and renal tract infections. Particularly, small infants with infective enteritis develop abdominal distention rapidly and this should be differentiated from intestinal obstruction and peritonitis.

Management:

Management of infective enteritis basically involves the assessment of the degree of dehydration and correct fluid and electrolyte replacement.

Assessment of dehydration:

The very simple clue for dehydration is weight loss. Dehydration is assessed on the basis of weight loss. Clinical signs are not apparant if only less than 5% body weight is lost. Obvious signs of weight loss imply 7.8% of body weight signs of circulatory failure - 10% of body weight. Weight losses in excess of 12% are probably not compatible with life. For example -

- a) 5% dehydration in an infant weighing 5 Kg. is 250 grams. which is equivalent to 250 ml. of water and
- b) 10% implies 500 grams. equivalent to 500 ml. of water.

Classification according to severity.

| S.No. | Clinical Features. | Mild. | Moderate. | Severe. |
|-------|--------------------|---------------|-----------------------|-------------------------------------|
| 1. | Stools | 5-8/day. | Frequent fluid stool. | 15 or more/day. |
| 2. | Dehydration | Nil | Mild-moderate. | Marked. |
| 3. | Acidosis | Nil | No clinical acidosis. | Present. |
| 4. | Fever & Vomiting. | May be. | May be. | Present invariably. |
| 5. | Toxicity. | Nil | Slight. | Prostration and semicomatose state. |
| 6. | Weight Loss. | Less than 5%. | 5% | More than 5%. |

General rules for therapy:-

1. The aetiological factors eradicated if possible.
2. The gastrointestinal tract is rested.
3. Dehydration, electrolyte imbalance and shock are combated.
4. Adjuvant therapy.
5. Isolation.

Treatment of mild diarrhoea:-

- No food.
- Clear fluids are given (coconut water) ($\frac{1}{4}$ Qt. water + $\frac{1}{2}$ tsp. salt + $\frac{1}{2}$ tsp. sodium bicarbonate + 10 tsp. sugar).
- Then the gradual formula is started.
- When the stools have improved, there can be a gradual return to full strength formula and regular diet.

Treatment of moderate diarrhoea:-

- N P O for 12-24 hours.
- Water with added electrolytes (coconut water) and carbohydrates are given.
- Enough total fluid and electrolytes by oral and parenteral routes to prevent progression of dehydration and development of acidosis.
- Diluted milk may be used for interim feeding for 24 - 48 hours until the stools are improved.
- Gradual return to regular feeds.

Treatment of severe diarrhoea:-

- Immediate IV infusion.
- Blood culture and stool culture.
- Oral feeding started on 2nd day with clear fluid having electrolytes and carbohydrates.
- If oral fluids are tolerated, diluted milk be given followed by gradual return to normal feeds.
- Associated complications and aetiological factor treated promptly.

Adjuvants:

Antibiotics and chemotherapy are used according to the known or presumptive aetiologic organisms. Adsorbants (kaoline) and Antispasmodics (Lomotil) are of limited value. Water soluble vitamins are given to combat deficiency. As the infant improves, rarely tetany may occur and calcium can be given prophylactically. Trimethoprim is not preferred for children under the age group of 6 years because of associated side effects.

Isolation:

Isolation of the infected children are very important to avoid spread and reoccurrence.

Chronic Nonspecific Diarrhoea:

Chronic nonspecific diarrhoea is an ill-defined syndrome characterized by the appearance at 6 to 36 months of age of persistent, loose, foul-smelling stools containing mucus. Growth and weight gain are little affected by the chronic gastrointestinal disturbance that is frequently associated with an intercurrent infection. Partial response to diet and antibiotics may occur, but the administration of diiodohydroxyquin (Diodoquin), 0.32 to 0.65 Gm, daily in two or three divided doses is efficacious. Although the condition may last several months, the prognosis is very good.

CHOLERA:

Cholera is the result of infection with one of the pathogenic strains of cholera vibrio. Most cases are seen in the Indian sub-continent but outbreaks also occur in South-East and Central Asia. Infection occurs through the ingestion of contaminated food and water. Cholera carriers exist in areas where the disease is endemic and maintain endemicity through contamination of the environment. The cholera vibrio multiplies in the small intestine of man and produce toxins which cause damage and desquamation to epithelium. This results in the exudation of large volumes of fluid and electrolyte and the diarrhoea, dehydration and circulatory collapse which characterise this disorder. Blood stream invasion does not seem to occur.

The onset is usually acute with diarrhoea which rapidly increases in severity. Vomiting usually occurs soon after the onset. Within 24-28 hours the stools exhibit the characteristic 'rice water' appearances - thin whitish fluid containing denuded epithelial cells and mucus. Dehydration rapidly occurs and if fluid loss is not rapidly replaced, circulatory failure, anuria and death result. The diagnosis which can usually be made from the history and the typical appearance of the stools is readily confirmed bacteriologically. Management follows precisely along the lines described above for the severe case of infective enteritis but the acute and explosive nature of the illness is such that intravenous replacement must be rapid to prevent or correct circulatory failure. Tetracycline appears to reduce the diarrhoea and the duration of vibrio excretion in cholera. It is given in a dosage of 40 mg per kg per day in 4 divided doses. Cholera vaccination is indicated for persons intending to spend time in an endemic area. The immunity is however short - probably less than 4 months.

ORAL FLUID - A SIMPLE WEAPON AGAINST DEHYDRATION
IN DIARRHOEA.

By

N.F. Pierce & N. Hirschhorn.

:::

"Oral rehydration therapy provides an effective weapon in the fight against acute diarrhoeal diseases, including cholera. Recently, this method of treatment has been used in various situations and remarkable results in terms of controlling the severity of diarrhoea and mortality due to acute diarrhoea were obtained."

The aim of oral therapy is to prevent and treat dehydration, which is the main complication in any diarrhoeal illness. The treatment involves prompt replacement of faecal losses of water and electrolytes by an oral glucose-electrolyte solution. However, to understand the importance of oral therapy one must have a comprehensive view of the problem presented by diarrhoea.

Why is diarrhoea a problem?

Acute watery diarrhoea due to infections is second only in incidence to infections of the respiratory tract. In some developing nations diarrhoeal attacks may occur as frequently as once every month during a child's second year of life. In these countries acute diarrhoea is probably the most common cause of death;

it is certainly the major cause of mortality in small children. Cumulative mortalities of 25-40% among children up to the age of 5 years are common in developing nations; 40% or more of these deaths, which are caused by dehydration or chronic malnutrition, are associated with acute diarrhoea. Malnutrition is often initiated by acute diarrhoea and is aggravated by each subsequent attack of diarrhoea.

Thus in most developing countries, owing to the frequency of diarrhoeal illness (especially in young children) and the resulting morbidity and mortality, acute diarrhoea is a considerable health problem and every effort is needed to bring it under control and to prevent serious consequences.

What causes diarrhoea and the ensuing dehydration?

Acute watery diarrhoea is caused by a variety of infectious agents (see Table 1), whose actions alter intestinal function by different mechanisms. For example, viruses replicate within mucosal cells, produce patchy but transient mucosal damage, and cause water and electrolyte

secretion which is greatest during the healing phase. In contrast bacteria like Vibrio cholerae and enterotoxigenic strains of Escherichia coli are not invasive but colonize the mucosal surface and secrete an enterotoxin, which causes mucosal secretion without any apparent damage to mucosal cells. Although these mechanisms differ, the clinical and biochemical effects produced are similar in several important aspects:

Infectious agents that commonly
cause acute water diarrhoea.

| Bacteria | Viruses |
|----------------------------------|----------------|
| Salmonella species | |
| Shigella species 1 | Reo-like virus |
| Enterotoxigenic Escherichia coli | Norwalk agent. |
| Vibrio cholerae | |
| Non-cholera vibrios | |
| Vibrio parahaemolyticus | |

1 May also cause dysentery without watery diarrhoea.

(1) Normally the small bowel secretes, rather than absorbs, water and electrolytes. In severe diarrhoeal diseases, the volume secreted is large and cannot be fully absorbed by the colon, so that watery diarrhoea continues even if oral intake stops. In mild disease, diarrhoea may occur only when there is food and liquid intake, since the volume of intestinally secreted fluid is small.

(2) Diarrhoeal stool is usually isotonic with plasma but may differ greatly from plasma in electrolyte content (Table 2). Potassium and bicarbonate concentrations are usually higher than plasma; sodium concentration may be similar to plasma or lower. In general, sodium content approaches that in plasma when stool is watery and the rate of loss exceeds 50 ml per kg of body weight in 24 hours. Sodium content is lower when the rate of loss is less than this, and when faecal matter is present and food intake is continued.

(3) Disaccharidase enzymes of the bowel mucosa (especially lactase) are often damaged so that the ingestion of lactose in cow's milk by infants with lactase deficiency makes the diarrhoea worse. The lactose is not absorbed in the small bowel but passes into the colon along with the additional water required to maintain isotonicity. In the colon the lactose is fermented into smaller fragments, including organic acids, which apparently further increase the stool volume by their osmotic activity.

(4) Acute diarrhoea usually lasts 1 to 7 days and ends when the infection is controlled by the host's defences. In some cases (e.g., cholera and shigellosis), the duration may be shortened by antibiotics. Chronic or recurrent diarrhoea usually indicates intestinal abnormalities due to malnutrition, disaccharidase deficiency, or parasitism.

Dehydration is the result of fluid loss from the body. With diarrhoea this loss occurs almost entirely from the extracellular fluid compartment and leads to a progressive diminution of blood volume. When this fluid loss is less than 5% of the body weight, thirst is the only sign of dehydration (apart from the diarrhoea). When the deficit exceeds 5% of the body weight, the following symptoms and signs develop rapidly: tachycardia, decreased skin turgor, postural hypotension, irritability, oliguria or anuria, severe thirst, hypotension, and stupor or coma. Shock occurs when the deficit equals about 10% of the body weight; greater losses cause death. It is important to note that half of this lethal deficit can develop before the usually recognized signs of dehydration appear. Vomiting occurs but may not necessarily be due to fluid deficit. Some children develop serious hypoglycaemia, partly owing to fasting; this may cause coma, convulsions, and even death.

Hypertonic dehydration, which is due to relatively greater losses of water than salt, may occur in a few infants and, when serious, may cause coma or convulsions and death. This problem may occur when infants with diarrhoea are fed large amounts of cow's milk; larger volumes of stool with lower sodium content are then produced and a greater deficit of water than salt results. Hypertonic dehydration is relatively uncommon in developing countries, probably because more children are breast-fed (breast milk has a lower solute load than cow's milk and the feeding volumes are smaller) and because cow's milk, when used, is very much diluted, thus providing extra water to replace the stool losses.

Hypotonic dehydration (serum sodium concentration less than 130 mmol/l) may occur when stool loss is replaced orally by plain water. Hypotonic dehydration causes few specific symptoms and is usually much less dangerous than hypertonic dehydration.

Long-term effects of repeated diarrhoea;

The long-term effects of repeated diarrhoeal attacks are largely nutritional, children under the age of 3 years being the most frequent victims. Watery diarrhoea causes a negative nitrogen balance, which reflects protein catabolism due to the infection and to fasting. When the quality and quantity of a patient's food intake is marginal,

the protein losses during diarrhoeal attacks (and during other infections) are only slowly regained and the weight lost is only gradually recovered. The cumulative result is restricted growth and increasing protein deficiency. If this process is repeated, it is eventually complicated and accelerated by chronic diarrhoea and dietary malabsorption associated with atrophy of the small bowel mucosa. The final outcome, if not death from an intercurrent infection, is death with the clinical picture of protein-energy malnutrition.

Major objectives in treatment:

The two major objectives in treating acute diarrhoea are :

- very early replacement of water and electrolyte losses to prevent or treat dehydration.
- maintenance of adequate nutrition to prevent malnutrition.

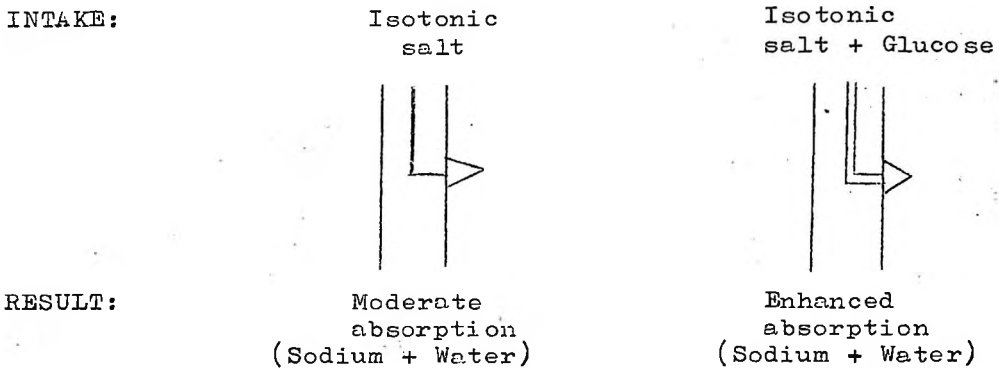
Early replacement therapy should begin promptly after diarrhoea starts. Early treatment has three important advantages. First, it avoids the risk of death from severe dehydration. Second, it minimizes the symptoms associated with increasing water and electrolyte deficit, e.g., vomiting, anorexia, lethargy, or coma, which interfere with continued feeding. And third, the treatment needed is simpler because it is given while two important homeostatic mechanisms (thirst and renal function) are still intact. When an oral glucose-electrolyte solution is taken under these conditions, thirst is one important guide to the amount required, and normal renal function permits the excretion of any excess of water or salt.

Maintaining nutrition during acute diarrhoea is essential to prevent the adverse effects of fasting. Moreover, nutrition can be maintained because the gut remains able to absorb a variety of nutrients, lactose being the most common exception. Apart from lactose, usually no other dietary restriction is needed. In fact, there is no physiological basis to the common belief that the bowel should be "rested" during acute diarrhoea.

The primary goal of treatment is not the immediate termination of diarrhoea. Although shortening the duration of diarrhoea would be desirable, most attempts are either ineffective (such as "routine" antibiotics or "antidiarrhoeal mixtures") or they interfere with goals of higher priority. For example, fasting may diminish stool loss but obviously prevents maintenance of nutrition. Moreover, treatment aimed at stopping diarrhoea often diverts attention from the major objective - fluid and electrolyte replacement - until a serious deficit has developed.

Other sugars, especially those that yield glucose when broken down in the gut, may be useful for enhancing salt and water absorption when glucose is not available. If their breakdown were incomplete, however, their effect would be reduced. A sucrose-salt solution containing 40 g sucrose per litre, for example, has been almost as effective as the glucose-salt solution for treating patients above the age of 5 years with severe cholera and other diarrhoeal diseases (D.L. Palmer et al., unpublished observations).

A. NORMAL SMALL INTESTINE



B. ACUTE WATERY DIARRHOEA

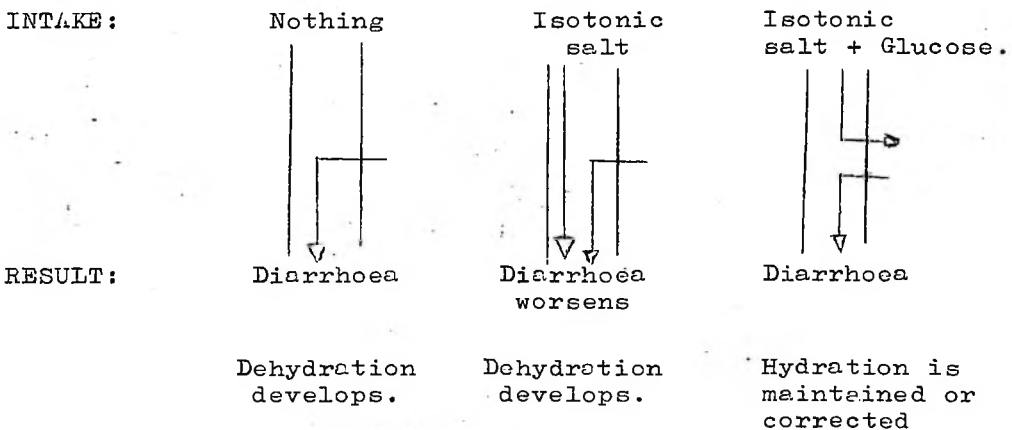


Fig. 1. Effect of glucose on intestinal absorption of salt and water (A) in the normal small intestine and (B) during acute watery diarrhoea. (See text for details).

For the successful implementation of oral therapy in local health centres and even in homes, the method employed must be uniform and simple. The use of an oral solution based on a single formula is essential in such an approach, the advantages outweighing any possible value of solutions of varying composition for use in different age groups or phases of treatment. Moreover, it should be emphasized that the water and electrolyte requirements are reasonably constant (and can thus be met by a single solution) in those clinical situations in which large amounts have to be given.

Table 2 and the accompanying box show the composition of a widely tested and effective glucose-electrolyte solution, which is approximately isotonic with plasma; it contains sodium and glucose in an approximately equimolar ratio and sufficient potassium and bicarbonate to replace a major portion of stool losses. The sodium concentration is sufficient to correct an initial isotonic deficit and to replace continuing stool losses when the rate of loss is moderate or severe. Normal renal function is essential so that any excess of salt or water may be excreted. In children with mild diarrhoea the stool sodium concentration is often lower than that in the oral solution; the water requirements of such patients are also met by the water provided in continued oral feedings.

Table 2. Electrolyte content of stool in acute watery diarrhoea compared with that of normal plasma, and the electrolyte and glucose content of oral fluid.

| | Na + | K + | Cl- | HCO ₃ - |
|-------------------------|------|-----|-----|--------------------|
| Cholera stool | | | | |
| adults | 140 | 13 | 104 | 44 |
| children (< 5 years) | 101 | 27 | 92 | 32 |
| Enteritis stool | | | | |
| children (< 5 years) | 56 | 25 | 55 | 14 |
| Normal plasma | 142 | 4.5 | 105 | 25 |
| Oral fluid ^b | 90 | 20 | 80 | 30 Glucose 111 |

a - Values, which are averages from several studies, are expressed in mmol/l. Widest variations are seen in Na and Cl content of enteritis stool in children. Sodium content drops rapidly from mean values above 90 mmol/l to a mean of about 60 mmol/l when the rate of stool loss falls below 50 ml/24 h per kg of body weight.

b - Values are expressed in mmol/l. The composition of oral fluid in grams per litre is given in the accompanying box.

Table 3: Guidelines for oral fluid therapy.

| | Amount given. | Time required. |
|---|--|--|
| 1. Rehydration | | |
| A. For mild dehydration (on examination, normal or diminished skin turgor, or sunken fontanelle; patient able to drink) | 50-120 ml/kg, the larger amount when turgor is diminished. Encourage patients to drink until they refuse. Adults may need up to 1000 ml per hour. If patients tire or drinking, use a continuous nasogastric infusion. | Usually 4 - 6 hours. |
| B. For severe dehydration (hypotension, shock, stupor or coma, absent radial pulse) | 100 ml/kg intravenous poly-electrolyte solution (e.g., Ringer's lactate ^a), or normal saline if nothing else is available. Do not use oral therapy until shock is corrected. | Give half rapidly (30-60 minutes), the remainder in 3-6 hours. |
| 2. Maintenance | | |
| A. For mild continuing diarrhoea (less than 1 stool every 2 hours) | 100-200 ml/kg oral solution. | Every 24 hours, until diarrhoea stops. |
| B. For severe continuing diarrhoea. | 15 ml/kg oral solution, sometimes more. Observe carefully to confirm adequate maintenance of hydration. | Every hour, until diarrhoea becomes mild or stops. |

^a An ideal polyelectrolyte solution is DTS (diarrhoea treatment solution), which is recommended by WHO. If half-strength Darrow's solution with 2.5% glucose is used, give 150 ml/kg.

Instructions for use:

The oral solution should be made fresh daily. It is practical to use foil packets containing pre-weighed mixtures of glucose and the salts to be dissolved in a specified volume of drinking water. These packets can be safely stored as long as they are moisture proof.

The guidelines for the use of oral therapy are simple (Table 3). The limits of its usefulness will be described below. It may be used as the sole therapy to rehydrate patients with mild or moderate dehydration (up to 7% loss of body weight) and to maintain hydration in almost all patients, after rehydration, until the diarrhoea stops. In general, most patients who are awake and able to drink well can be treated by the oral route.

Thirst is a very useful guide to the amount of oral solution required. Rehydration is often achieved by allowing the patient to drink as much fluid as desired, but patients with very rapid stool loss may have to be encouraged to drink sufficient fluid. Excess fluid intake may cause puffy eyelids (this is harmless), in which case the oral solution should be stopped until this finding disappears. If patients tire of drinking, the fluid can easily be given by continuous nasogastric infusion. If the initial dehydration is severe, rehydration must be performed intravenously with an isotonic polyelectrolyte solution (or normal saline if only that is available), after which the oral solution may be used for maintenance.

The maintenance requirements of oral fluid, after rehydration, should equal the rate of continuing stool loss. For adults with severe diarrhoea (e.g., in cholera), measurement of stool losses, separate from urine, by the use of a cholera cot helps to determine the maintenance requirements. Since accurate stool collections are not possible with infants, the stool losses should be carefully estimated by frequent observation of diapers. Stool losses are usually greatest in the first 24 hours of treatment and decrease steadily thereafter. Patients with mild diarrhoea may be given oral fluid for home use, returning each day for reexamination and more fluid, if needed. Those with frequent diarrhoea should be observed every 3-6 hours to determine whether oral intake is sufficient and hydration appears satisfactory. Valuable signs of adequate hydration include normal skin turgor, normal urine flow, normal pulse rate and volume, and a sense of well-being. If signs of dehydration reappear despite vigorous oral replacement, parenteral fluid therapy should be started.

Patients given the oral solution may vomit. This occurs most commonly when oral therapy is first begun. Unless vomiting is severe and repeated, oral therapy should

be continued, small amounts being given frequently. The volume of fluid lost by vomiting is usually a very small portion of that taken and retained by the patient.

If possible, patients with diarrhoea should continue to eat and drink to maintain their nutrition. Drinking of the oral solution can be alternated with food intake. Breast-fed children should continue to be nursed; those given cow's milk should take reduced amounts limited to 150 ml every 4 hours. If the diarrhoea worsens markedly, cow's milk should be stopped and other protein foods used. Staple foods such as cereals, bananas, cooked legumes (lentils, chick peas, etc.), and potatoes can be continued during the diarrhoea. Adults should resume a normal diet of well-cooked food as soon as their appetite returns. When the required amounts of oral fluid have been taken, patients may be given additional water if they desire it.

Antibiotics should not be given routinely. Oral tetracycline shortens the duration of diarrhoea in cholera. Antibiotics are also effective in severe shigellosis, but the appropriate choice can be made only by testing the sensitivity of the organism in vitro.

There are no other adjuncts to the treatment of diarrhoea that are of proven value.

Limitations:

There are some circumstances in which oral therapy is not successful or has not been fully tested and therefore cannot be recommended. In these situations, water and electrolyte replacement should be given parenterally:

Patients with severe dehydration, often with signs of shock. Such patients need very rapid water and salt replacement intravenously. Oral therapy is too slow.

Patients who cannot drink because of fatigue, stupor, or coma. The oral solution can be given to such patients by nasogastric tube.

Patients with prolonged oliguria or anuria, but not those with brief oliguria or anuria which often accompanies dehydration. The former require precise administration of water and electrolytes, usually parenterally.

Patients with severe and sustained vomiting.

About 3% of patients with acute diarrhoea have serious glucose malabsorption. In these patients oral therapy causes a marked increase in stool volume, the stool containing large amounts of glucose, and the dehydration worsens.

Patients with very severe diarrhoea (e.g., adults losing more than 800 ml of stool per hour) may be unable to drink enough fluid to replace the continuing losses.

Oral therapy has not been evaluated in premature infants or in babies less than one month old.

Oral therapy has been used with success to treat thousands of attacks of mild and moderate acute diarrhoea in children and adults in many parts of the world, and its usefulness has been carefully documented in a number of reported studies. When cholera and infantile diarrhoea were treated by this method by experienced workers, there was almost no mortality and the need for intravenous fluids was reduced by 70-90%. When oral therapy was used under the worst possible conditions to treat cholera among refugees from Bangladesh, most of the treatment being given by untrained family members, mortality was only 3% and half of these deaths occurred before the treatment could be given.

These observations and the rationale on which this approach to treatment is based argue strongly in favour of oral therapy as the single most effective therapeutic tool in the treatment of acute diarrhoeal disease.

Note:-

While intravenous rehydration is the best form of treatment when patients are in shock and are unable to drink, oral rehydration is the best procedure for treating mild and moderate dehydration and for preventing severe dehydration. The oral rehydration treatment of acute diarrhoea can be carried out in the homes of patients and thus saves expensive injectable fluids and hospital costs. The numerous instances of the use of intravenous fluids when the oral fluid could easily have been justified are an example of waste.

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Convulsions in Childhood.

Convulsive phenomena are more common in children and occur with a wide variety of CNS disorders. While the incidence in adults is less than 1%, the child suffers an incidence of 7%. Convulsions should be regarded as a symptom and not as a disease entity. There is a broad spectrum between those in whom a seizure may be provoked only after the administration of analeptic drugs and those whose seizures are spontaneous. In this latter group, the provoking stimuli are yet too subtle to be determined by present methods. Seizures may be provoked in certain individuals by hyperventilation, television, flickering of light, sunlight, sky, fire, water and even pain.

The basic pathophysiology involves abnormal neuronal discharge which may be recorded electrically as a seizure pattern on the E.E.G. and are manifested clinically as convulsions.

Incidence.

More common, during first 2 years than at any other period of life.

In very young infants $\left\{ \begin{array}{l} \text{Intra cranial birth injuries,} \\ \text{effects of anoxia and haemorrhage.} \\ \text{Congenital defects of brain, are} \\ \text{the most frequent causes.} \end{array} \right.$

In later part of infancy and early childhood. $\left\{ \begin{array}{l} \text{Acute infection.} \\ \text{(Extra cranial or intra} \\ \text{cranial).} \end{array} \right.$

The less frequent causes - $\left\{ \begin{array}{l} \text{Tetany,} \\ \text{Idiopathic epilepsy,} \\ \text{Hypoglycaemia,} \\ \text{Brain tumors,} \\ \text{Renal insufficiency,} \\ \text{Poisoning,} \\ \text{Asphyxia,} \\ \text{Spontaneous intra cranial} \\ \text{haemorrhage,} \\ \text{Thrombosis,} \\ \text{Post-natal trauma,} \\ \text{Hypocalcemia.} \end{array} \right.$

Mid childhood, convulsions due to acute extra cranial infection are not frequent. Idiopathic epilepsy is the commonest and it appears at the age of 3 years. Other causes in the post infancy period - congenital defects of brain, residual cerebral damage due to early trauma, infection, lead poisoning, brain tumors, acute and chronic glomerulonephritis degenerative disease of brain, drug ingestion.

Convulsion in newborn:

A clinical seizure at any age is associated with a paroxysmal bursts of electrical activity in CNS. In newborn, electrical activity of cerebral hemisphere is poorly developed but subcortical rhythm are present. Mass myoclonic movements have been said to occur in utero, but the tonic and clonic movement that characterise the grandmal seizures are rarely apparent during the first few weeks of life. The low incidence of G.M seizure reported during neonatal period probably reflects both the poor development of cerebral hemisphere and lack of uniformity in recognizing or classifying seizures or their equivalents.

After an episode of acute anoxia, a convulsion may take the form of a tonic spasm preceded by a few clonic jerks. The E.E.G becomes flattened.

Focal seizures may be associated with irregular jerky movements and nystagmus or staring, pallor and hypotonia, the E.E.G showing paroxysmal bursts of multiple spike and slow wave discharge.

In some instances respiration becomes slow and irregular with periods of apnoea and a feeble cry. The neck becomes rigid, pupils dilate and the child drools. Alteration of E.E.G may also occur with slight movement of fingers, toes or eyelids with a change in colour or with chewing.

The presence of a seizure suggests cerebral insult and should alert the physician. The possible metabolic use of drugs should be considered. A disorder of amino acid metabolism should be excluded through chromatography of urine or serum. Trial by pyridoxine and examination of urine for 'Maple syrup urine disease' may be life saving.

Earlier the onset, better the prognosis - unless other associated complication intervenes. The outlook is poor if heart rate is slow or if symptoms persists for more than 72 hours.

Supportive care is the primary treatment and management. This includes prevention of shock, maintenance of adequate air way and sedation. Diazepam and phenobarbital are the drugs of choice.

Acute convulsions in infants and children:

Generalised tonic and clonic convulsions similar to grandmal attack of epilepsy are by far the most common. Practically speaking all seizures resulting from extra cranial disorders are of this type.

3.5% of children have febrile convulsions most of which occur after first 6 months of life but within 2 to 3 years. The incidence decreases upto 6-8 years after which such seizures are rare. Males are more prone to this type and there appears to be an increased susceptibility in families. Febrile convulsion occur at the peak of the febril illness.

In late infancy and early childhood most of the seizures follow an acute febrile illness. Child who had an early episode should be examined thoroughly for the possible causes. Such disorders as Tetany, lead encephalopathy, Intra cranial haemorrhage or injury or tumor, poisoning strychnine, hypoglycemia, asphyxia, cerebral sinus thrombosis, acute nephritis and epilepsy should be considered. Age of the child, complete history and complete physical examination including fundoscopy will help to arrive at a diagnosis.

Determination of serum calcium, blood sugar and BUN (Blood urea nitrogen) will help in the differentiation of hypocalcaemic Tetany, hypoglycemia and acute nephritis respectively. Coexisting hypertension, albuminuria and cylindruria are evidences of nephritis.

In lead poisoning, X-Ray reveals lead line in long bones, multiple recent or healed fractures as in the battered child syndrome or thickening of the skull or widening of sutures in case of raised intra cranial tension.

In case of infection, the type of infection (extra cranial or intra cranial) should be determined.

Treatment:

In case of febrile convulsion treatment is 3 mg/kg phenobarbital and lowering of temperature is the ideal thing to do. After other causes of convulsion has been excluded a trial of pyridoxine would help.

Prognosis:

Every episode of convulsion increases the chances of nonfebrile epilepsy.

Developing Idiopathic epilepsy, is a high possibility in children who have more than 5 episodes in a period of 12 months or a single seizure more than an hour or persisting E.E.G abnormalities. Approximately 25% of epileptics give a history of febrile convulsions.

There is a wide range of opinions about starting daily anti-convulsant therapy. Some suggest anti-convulsant treatment after the first afebrile seizure and others suggest daily anti-convulsant therapy after the first

attack of convulsion (febrile or afebrile), because a great majority of epileptic give a H/o febrile convulsions. Any way both should be kept in mind before starting the daily anti-convulsant treatment. Consultation with child's parents is very necessary to avoid unnecessary complications.

Disorders stimulating epilepsy:

- Norcolepsy.
 - Abdominal epilepsy.
 - Breath holding.
 - Hysterical fits.
 - Syncope.
 - Apnoeic episodes during swimming.
 - Migraine (Hemicrainia).
-

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28-8-1979.

ETIOLOGIC CLASSIFICATION OF CONVULSIVE DISORDERS.

I. ACUTE OR NONRECURRENT FORMS:

"Febrile convulsions" (e.g. at onset of acute extracranial infections or in association with high environmental temperatures).

Intracranial infections (e.g. acute meningitis, encephalitis, sinus thrombophlebitis, cerebral abscess, tetanus, malaria, typhus fever).

Intracranial hemorrhage (e.g. from birth or other trauma, hemorrhagic disease, rupture of defective vessels sickle cell disease).

Toxic:

1. Convulsant drugs (e.g. aminophylline, antihistamines, camphor, propoxyphene, pentylenetetrazol, phenothiasine, hexachlorophene, corticosteroids, strychnine and thujone).
2. Tetanus.
3. Lead encephalopathy.
4. Shigellosis, salmonellosis.

Anoxic (e.g. sudden severe asphyxia, inhalation anesthesia)

Metabolic or nutritional (e.g. acute hypocalcemic and hypomagnesemic tetany, hyponatremia and hypernatremia, alkalosis, therapeutic hypoglycemia, pyridoxine deficiency, phenylketonuria, copper deficiency (Menkes), maple syrup urine disease, hyperammonemia, argininuria, argininosuccinic aciduria, hyperlysinemia, tyrosinemia, glycineemia)

Organic acidurias (propionic, lactic, green acyl dehydrogenase deficiency)

Acute cerebral edema (e.g. in acute glomerulonephritis or allergic edema of the brain)

Brain tumor

Miscellaneous (porphyria, systemic lupus erythematosus)

II. CHRONIC OR RECURRENT FORMS:

Epilepsy:

1. Idiopathic (primary, cryptogenic, essential or genuine epilepsy)
 - a) Hereditary or genetic type
 - b) Nongenetic or acquired idiopathic type (?)
2. Organic (secondary or symptomatic epilepsy-with residual brain damage from previous focal or diffuse injuries)

- a) Post-traumatic (e.g., from direct laceration of brain tissue)
 - b) Posthemorrhagic (e.g., from injury at birth or later, from hemorrhagic diseases, pachymeningitis, rupture of miliary aneurysm)
 - c) Postanoxic (e.g., from severe asphyxia neonatorum)
 - d) Postinfectious (e.g., following encephalitis, meningitis, sinus thrombophelebitis or abscess)
 - e) Post-toxic (e.g., kernicterus, encephalopathy following lead, arsenic or other chronic poisoning)
 - f) Degenerative (e.g., "idiopathic atrophy," cerebromacular degeneration, encephalitis periaxialis diffusa, intracranial neurofibromatosis, incontinentia pigmenti)
 - g) Congenital (e.g., cerebral aplasia, porencephaly, tuberous sclerosis, hydrocephalus, vascular anomalies such as the Sturge-Weber type and arteriovenous aneurysms)
 - h) Parasitic brain disease (cysticercosis, toxoplasmosis, syphils)
 - i) Posthypoglycemic injury.
3. Sensory (reading, touch, light, sound, music, self-induced)

Epilepsy-simulating states:

Narcolepsy and cataplexy
Hysteria ("psychogenic epilepsy")
Tetany:

- 1. Hypocalcemic (e.g., idiopathic, postoperative, neonatal, vitamin D deficiency, deficient intestinal absorption)
- 2. Of alkalosis (e.g., vomiting, administration of bicarbonate, hyperventilation)

Hypoglycemic states:

- 1. Hyperinsulinism (e.g., tumor or hyperplasia of islets of Langerhans)
- 2. Hypopituitarism (e.g., deficiency of adrenocorticotrophic, thyrotropic and growth hormones)
- 3. Adrenocortical insufficiency.
- 4. Hepatic disorders (e.g., von Gierke's disease)
- 5. Miscellaneous (e.g., leucine-induced, idiopathic ketotic)

Uremia

"Cerebral" allergy






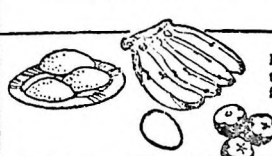







Cardiovascular dysfunction or syncopal attacks (e.g., simple fainting attacks, Stokes-Adams syndrome, hyperactive carotid sinus reflex)

Migraine.

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26.8.1979.

FEEDING & IMMUNIZATION CHART

| | | | | |
|--|---|--|---------------------------------|---|
| Mother's milk |  | New born child | Smallpox & BCO | |
|  | Rice & dal |  | 3 to 4 months | DPT Polio } I Dose |
|  | Rice, dal, iddli, greens, potato, tomato, banana |  | 4 to 5 months 5 to 6 months | DPT Polio } II Dose DPT Polio } III Dose |
|  | Rice, dal, iddli, greens, potato, tomato, banana, rice, vegetables, fruits, one egg. |  | 12 months | Measels (if available) |
|  | Rice, dal, iddli, greens, potato, tomato, banana, rice, vegetables, with emphasis on greens |  | 2 years | DPT Polio } Booster I |
| | |  | 3 years | Re-vaccination |
| | |  | 4 years | DPT Polio } Booster II |
|  | Rice, dal, iddli, greens, potato, tomato, banana, rice, vegetables, with emphasis on greens |  | 5 years (School going child) | BCG Booster |