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Brief Reports

Epidemiological Considerations on Age Distribution of Paralytic Poliomyelitis

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Summary

Despite a declining trend of poliomyelitis due to high coverage of OPV, the age distribution of poliomyelitis cases have not shown any change over the years in India. More than 90 per cent of the cases have continued to occur in children below 5 years of age; the median age of cases remained below 2 years of age. The authors examined the issue and suggest that any major shift in age at paralysis may not occur in India, in spite of high vaccine coverage with OPV unless there is concomitant improvement in sanitation and hygiene.

Introduction

The overall coverage with oral polio vaccine (OPV) in India increased to over 85 per cent in 1990–91.[1] Before OPV was introduced in the country in 1979, virtually all the polio cases occurred in children under-5. It was argued that with high OPV coverage levels there should be a right-side shift in age at paralysis otherwise either the coverage is not high enough or the vaccine is not effective.[2,3] Whether age shift has happened in India or not, and if not the reason thereof, is the subject addressed in this communication.

Materials and Methods

Studies included here were carried out during the period when OPV was not yet available to Indian children. The comparisons drawn with studies conducted when the coverage rates with three doses were reported to be over 85 per cent. The community based studies[4–15] include surveys carried out in 1981 in the absence of immunization,[4–10] surveillance data of 1990 and 1991[10] when the OPV coverage levels with the third dose were over 85 per cent,[1] and some of the outbreak investigations.[14,15] These have been summarized in Table 1. The institution based studies and the investigations[2,3,16–32] which provide the levels of immunization coverage in the areas surrounding these institutions have been grouped together in Table 2. Wherever available, the immunization coverage levels for three doses of OPV have been provided. Unless otherwise indicated, the OPV coverage levels have been estimated by the surveys.

Results

As shown in Tables 1 and 2, more than 90 per cent of cases of poliomyelitis occurred in children below 5 years of age. The median age at infection was found to be less than two in virtually all of the studies included in this article. These findings were observed irrespective of the source of data and place or period of studies.

Discussion

The argument that the increase in the immunization coverage will be associated with a shift in the age at paralysis in afflicted children, is based on the presumption that herd immunity provided by high immunization coverage will prevent the remaining unimmunized children from coming in contact with wild poliovirus. Over a period of time, a sufficiently large number of susceptible persons will accumulate and encounter poliovirus for the first time in late childhood or adulthood. However, this argument does not take into account the following facts.

Oral Polio Vaccine virus also multiplies in the gut of the vaccinee and is excreted in the faeces. Vaccine virus also spreads to the contacts. Moreover, OPV vaccinated children and children having naturally acquired poliovirus humoral antibodies resist re-infection to the same extent;[33] the resistance is not absolute.

Polio is a highly infectious disease. Major epidemics can occur in areas which have high overall community vaccination levels.[34] Therefore, very high immunization coverage levels will be required to prevent the remaining susceptible children from encountering wild poliovirus infection

TABLE 1
Age distribution of acute poliomyelitis cases: community data

Ref. no.	Year	State (area)	Source of data	No. ill	% Cases by age at infection						Median age	Immunization coverage
					<1	<2	<3	<4	<5	<6		
4	1978	Tamilnadu (1 rural block)	Survey (all ages)	247	53		80		96		<1	Available as research project coverage not given
5	1981	Gujrat (Whole)	Survey (5–9 years)	468	36	68	84	92	95		<2	NA
6	1981	MP (Whole)	Survey (5–9 years)	283	28		78		93	98		NA
7	1981	W. Bengal (urban)	Survey (5–9 years)	73	34	63	84	88	94	99	<2	NA
		(rural)		83	33	67	83	90	93	99	<2	NA
8	1981	Tamilnadu & Pondicherry (Whole)	Survey (5–9 years)	400	37	75	92	96	99		<2	NA
9	1981	Delhi (Whole)	Survey (5–9 years)	117	42	65	82	90	94		<2	NA
10	1981	10 States* (Whole)	Survey (5–9 years)	1466	35	70	87	93	96		<2	NA
	1990	10 States*	Surveillance	1425	23	55	75	87	93		<2	
	1991	10 States*	Surveillance	377**	20	54	77	87	93		<2	
	1990	Other than 10 States	Surveillance	2647	29	63	82	91	95		<2	
	1991	Other than 10 States	Surveillance	783**	27	62	81	91	96		<2	
11	1985	Haryana (1 rural block)	Survey (1–11 years)	219	45	77	87	91	93		<2	80% (3 months–2 years) in 1983 and 1984
12	1985	Rajasthan (1 city)	Survey (5–15 years)	104			69		92			Available, not mentioned
13	1992	Haryana (2 districts)	Surveillance (AFP)	87	32	68	87		97		<2	Coverage 81–97% (12–23 months)
14	1984	Maharashtra (1 city)	Outbreak	145		70	86				<2	
15	1992	Andhra Pradesh (urban)	Outbreak	180	34	71	84	95	98		<2	Coverage low

*The 10 States/UT are: Goa, Haryana, Himachal Pradesh, Karnataka, Kerala, Maharashtra, Punjab, Tamilnadu, Chandigarh, Pondicherry.
**The data for first 7 months of the year.
Note: NA = Immunization not available; AFP = acute flaccid paralysis.

TABLE 2

Age distribution of acute poliomyelitis cases: hospital/sentinel centre data

Ref. no.	Year	State/city (area)	Source of data	No. ill	% Cases by age at infection						Median age	Immunization coverage
					<1	<2	<3	<4	<5	<6		
16	1961–1975	Delhi	SN	7769	41		92		98		<2	NA
2	1976–1978	Delhi	SN	4106	45		95				<2	NA
	1979–1981			5374	52		96				<1	<10%
	1982–1984			4186	52		97				<1	
2, 17	1985–1987			4263	53		95				<1	Coverage survey in February 1988 (12–23 months) 72–91% in 11 zones
18	1989	Delhi	SN		30	74	86	93	98		<2	
	1990			616	31	66	87	95	97		<2	
19, 20	1991	Delhi	SN	406	27	65	85	94	98		<2	Coverage in slums 53–76%
21	1992	Delhi	SN	576	28	62	82	92	96		<2	
22	1980s	Maharashtra (rural)	Hospital (camps)	5554	41	65	81	87			<2	NA
23, 24	1982–1986	Bombay	SN	5587	43	81	93	97	98		<2	Coverage 37–45%*
	1987–1988			1839	40	79	92	97	98		<2	
	1989			581	36	76	89	94	97		<2	Coverage 80% in <2
13, 15	1992	Bombay	SN	376	28	62	82	92	96		<2	Coverage 90%
25	1981	Madras	SN	516	53	84					<1	
26	1983	Madras	SN	1158	37	78	91	97	98		<2	
3	1988–1989	Madras	SN	614	31	79	93	98	99		<2	Coverage >80%
13, 27	1992	Madras	SN	307	36	73	87	95		99	<2	Coverage 99%
28	1973–1979	Vellore	Hospital	201	24	56	77	89	92	95	<2	
29	1977–1984	Manipal	Hospital	158	27	65	82	89			<2	Coverage 67–72%
30	1981	Calicut	Hospital	119	18	52	69	77	84	90	<2	
31	1987	Trivandrum	SN	231	33	69	81	90	95		<2	Coverage 44, 59, 94, 100, 91% (1983–87)**
32	1980–84	Calcutta	SN		24				92			Very low coverage

*Based on the number of doses of OPV vaccine administered to children aged under 12 months.

**Estimated from vaccine distribution data.

Note: SN = sentinel centre; NA = immunization not available.

in communities with poor sanitation and personal hygiene. With such a high immunization coverage, the vaccine virus will be so widespread that it is likely to infect the remaining unimmunized children also.

In the last few decades overall sanitation and hygienic conditions have not changed much, while rail and road transport has increased greatly providing the opportunities for transmission of virus even in remote areas. Even if there was a possibility, however small, of some isolated populations escaping exposure to poliovirus in childhood in the past, it is almost unlikely that a child will not get the (wild or vaccine) infection in the present era. Children will either get the immunity or suffer from the disease.

The results shown in Tables 1 and 2 are consistent with this reasoning. Despite the decline in the incidence of polio due to high immunization coverage,[1] most of the paralytic poliomyelitis cases continued to occur in children below 5 years of age. Although a small shift in age at infection was noticed in some of the studies in the form of comparatively less cases in children below 1 and 2 years of age, the median age at infection continued to be less than 2 years.

The situation is not much different in most of the other developing countries. They also have the same experience even with high immunization coverage. For example, in a recent outbreak of poliomyelitis in Oman,[35] the age distribution of cases was typical of that reported for countries with endemic poliovirus transmission, even though most infants and young children had received at least three doses of OPV by 12 months of age. Perhaps, age shift in developed countries occurred also due to improved community sanitation and personal hygiene.[36]

The results of the study suggest that any major shift in age at paralysis may not occur in India in spite of high vaccine coverage with OPV unless there is marked improvement in sanitation and hygiene, which is unlikely in the near future. The study also indicates (1) that acute flaccid paralysis in children under 5 may provide a highly sensitive and specific case definition for acute poliomyelitis in developing countries such as India, and (2) that the programme managers should concentrate on the incidence of poliomyelitis to monitor the trend of disease rather than the age at paralysis and should not be discouraged if they do not find major age shift despite reduction in incidence.

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Have Improvements in Birth Weight and Gestational Age Over 20 Years Influenced Neonatal and Infant Survival?

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Low birth weight (LBW) and prematurity are important causes of neonatal and infant mortality. To improve neonatal and infant survival, one of the targets set by the government of India is reduction in the proportion of LBW to 10 per cent by the year 2000.[1] However, there are ethnic variations in fetal growth and duration of pregnancy.[2–4] Indian fetuses have a slower intrauterine growth pattern compared to European fetuses. Furthermore, Indian babies tend to be born at an earlier period of gestation than European babies.[4] Both these account for birth of a small Indian baby. Antonysamy *et al.* (1994)[5] have reported that the incidence of LBW decreased by 10 per cent over a period of 20 years in Vellore. The present study examines the reduction in infant mortality from 1969–1974 to 1989–1994, separately and after adjusting for the effect of birth weight (BWT) and gestational age (GA).

Data for the period 1969–1974 and 1989–1994 were obtained from studies reported by Rao and Inbaraj (1978)[6] and Amalraj and Rao (1994)[7] respectively. These studies were conducted in the same localities in rural and urban settings, that is, in K. V. Kuppam and Vellore block, respectively, in North Arcot Ambedkar District, Tamilnadu. Test for homogeneity was done to compare the distribution of BWT and mortality for time periods.[8] Logistic regression analysis was done, using LR module for table data in BMDP PC/90 software.[9]

Two cohorts, one of 4220 live births in 1969–1974

7-15 December 1998

International Course on Prevention and Control of Traffic Accidents & Injuries

13-14 December
Three parallel workshops:

Mobility and Safety for Bicyclists & Pedestrians
Motor Vehicles and Road Safety
Pre-hospital Care for Trauma Victims

Transportation Research and
Injury Prevention Programme
Indian Institute of Technology
New Delhi 110016, India

Introduction

The Transportation Research and Injury Prevention Programme (WHO Collaborating Centre for Research and Training in Safety Technology) at the Indian Institute of Technology, Delhi, and the French National Institute for Transport and Safety Research (INRETS), France, are organising an eight day "International Course on Prevention and Control of Traffic Accidents and Injuries". The course will be held in Delhi, India, 7-15 December 1996. The course will include three stand-alone two-day parallel workshops on: (1) *Mobility and Safety for Bicyclists and Pedestrians* , (2) *Motor Vehicles and Road Safety,* and (3) *Pre-hospital Care for Trauma Victims* . These two-day parallel workshops will be held on the 6th and 7th day of the course. Participants attending the full course will be able to opt for one of the workshops. Extra places will be available for participants to register for one of the two day workshops alone. The Transportation Research and Injury Prevention Programme (TRIPP) at the Indian Institute of Technology will be the host Institution.

The Course (7-15 December 1998)

This eight day Course will bring together professionals working in the area of road safety to acquaint them with the state-of-the-art information in the field. The Course is especially designed for an interdisciplinary audience of law enforcers, police officers, traffic and road engineers, behavioral scientists, medical professionals and biomedical engineers. The contents of the Course are especially focussed to give a global perspective to the road safety problem.

By the end of the Course the participants should:

1) know about the latest findings in methodologies for prevention of traffic accident and injuries
2) be aware of policies and methods which have been shown to be successful or have not worked in the past
3) be able to improve or start their own programmes in road safety

Parallel Workshops (13-14 December 1998)

(1) Mobility and Safety for Bicyclists and Pedestrians
This will be one of the pre-conference workshops held in Asia in preparation of Velo Mondiale 2000 to be held in Amsterdam in June 2000. The objective of the workshop will be to assess the issues concerning mobility and safety for bicyclists and pedestrians in the Asian region. Experts from Japan, China, Bangladesh and India will make presentations on special projects. Workshop findings and recommendations will be presented to the Velo-Mondiale programme committee to be discussed in Amsterdam.

(2) Motor Vehicles and Road Safety
Participants in this workshop will be exposed to the methodology involved in setting motor vehicle safety standards and their relationship to the accident patterns and

statistics of the population involved. The workshop will include discussion on the scientific and biomechanical rationale of such standards, vehicle crash modeling and latest developments in research on vehicle safety including motorcycles and scooters.

(3) Pre-hospital Care for Trauma Victims
This workshop will include discussions on the latest international research findings in design of effective emergency care systems for trauma victims. Issues concerning drug use, technologies for trauma care and operation of ambulance systems will be included.

Participating Institutions

The Transportation Research and Injury Prevention Programme at the Indian Institute of Technology has been involved in road safety research for the past fifteen years, with special emphasis on vulnerable road users. The Institute has been designated as a WHO Collaborating Centre for Research and Training in Safety Technology.

The members of TRIPP have expertise in epidemiological studies of road crashes, designs of safer vehicles and safety equipment, traffic flow modelling, transportation planning and care of injured persons.

Institut National de Recherche Sur Les Transports Et Leur Securite (INRETS) is the French National Institute for Transport and Safety Research, under the authority of both the Ministry of Transport and the Ministry of Research. INRETS has a staff of 400. A third of the work force is devoted to road safety research and injury prevention, with over 25 years experience. Research projects are conducted in cooperation with other European countries and a special programme on information transfer and research cooperation with less developed countries has been under way for fourteen years. INRETS was designated as a WHO Collaborating Centre for Accident Prevention in 1990.

Participants

The eight day Course:
The Course will have places for a limited number (30) of participants who must have knowledge of English. The participants will be selected on the basis of their involvement in road safety research, involvement in policy making and implementation of safety measures. An attempt will be made to have a balanced mix of engineers, law enforcers, social scientists and medical professionals. The participants will have to bring road accident statistics pertaining to their countries/states to the Course. The Course participants can opt for one of the three workshops on the sixth and seventh days.

Two-day parallel Workshops
Additional participants can register for the stand alone parallel Workshops. Each workshop is expected to have about 20 participants with backgrounds relevant to each topic.

Course outline

Day 1 International overview, systems approach, injury as a health problem
Day 2 Accident phenomenon, behaviour, risk analysis
Day 3 Traffic conflicts, safety diagnosis
Day 4 Diagnosis and evaluation, education
Day 5 Country presentations, field visit
Day 6 Regulation, blackspot treatment, human tolerance
Day 7 Parallel workshops
Day 8 Parallel workshops
Day 9 Road safety management, evaluation

Fees

Full Course:
- Indian industry and international participants : US$ 250 or INR 10,000
- Indian educational institutions & public sector organisations : US$ 125 or INR 5,000

Participants registering for one two-day Workshop *only*:
- Indian industry and international participants : US$ 125 or INR 5,000
- Indian educational institutions & public sector organisations : US$ 75 or INR 3,000

All fees include tuition, course material, tea/coffee during breaks and lunches

Accommodation

Lodging arrangements can be made on request in the Institute guest house at the rate of US $ 16.00 per day or in an inexpensive hotel for about US $ 50.00 per day. Both are based on *double occupancy* including breakfast and dinner.

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PREVENTION AND TREATMENT OF VITAMIN A DEFICIENCY

MINISTRY OF HEALTH & FAMILY WELFARE, GOVERNMENT OF INDIA

PREFACE

In recognition of the magnitude of the problem of vitamin A deficiency, India was the first country to launch a national programme for prevention of blindness in 1970. The overall strategy includes long and short term measures. The long term measures are designed to increase the availability and consumption of foods rich in vitamin A. The administration of concentrated dose supplement of vitamin A to children between the ages of 6 months to 5 years constitutes the short term measure to prevent blindness and offer relief from other debilitating effects of vitamin A deficiency.

The National Programme has been reviewed by the National Institute of Nutrition in 1978 and the findings indicate a need to improve the awareness of concerned health professionals and programme managers as well as elicit community participation in order to achieve the desired impact.

The Department of Health and Family Welfare set up a Task Force on Vitamin A in 1989 to formulate policies and streamline procedures for improving the implementation of the vitamin A programme. In this effort, a booklet for health professionals and programme managers has been developed to provide the requisite information and encourage interlinkages with other professionals particularly of the ICDS scheme. Professionals engaged in the National Prophylaxis Programme against Blindness due to Vitamin A Deficiency have an excellent opportunity and great responsibility in controlling easily preventable nutritional blindness.

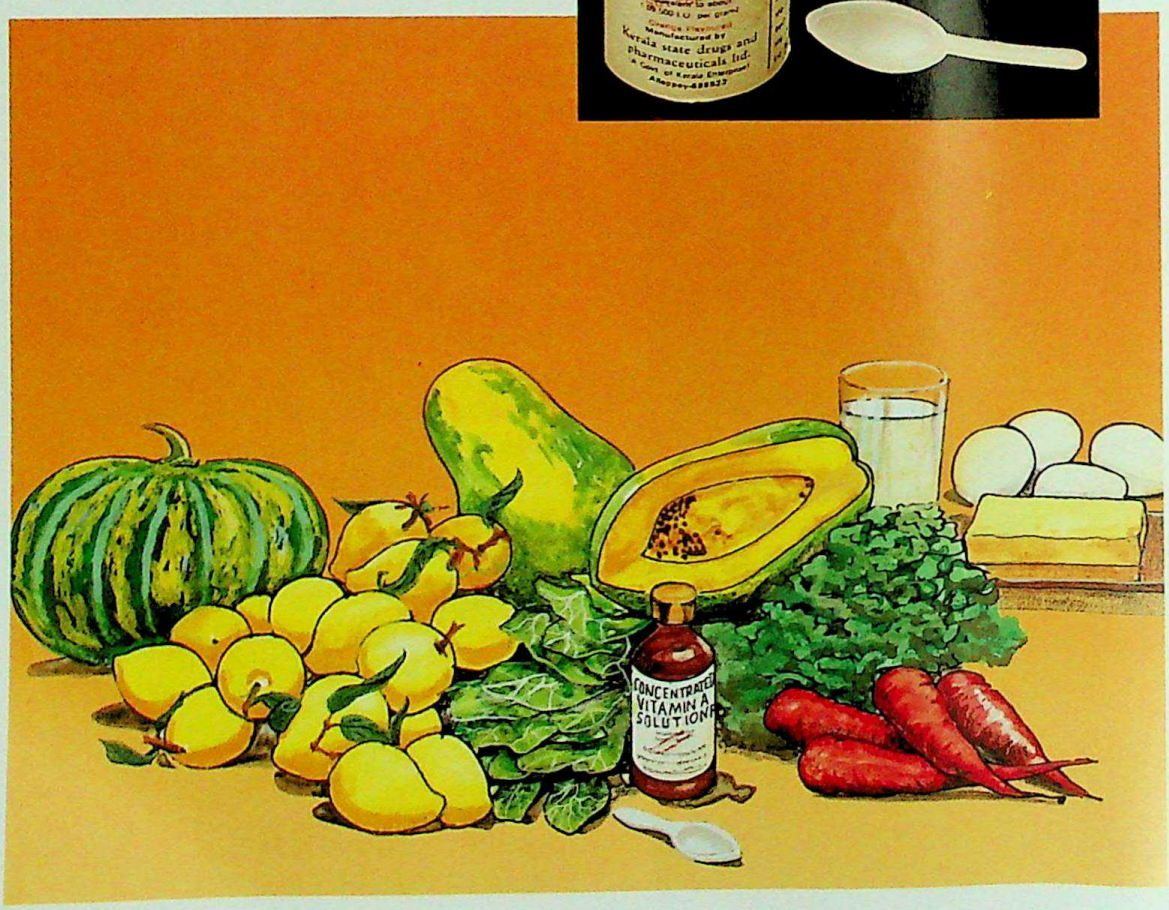
K Banerjee.

(K.B. Banerjee)
Deputy Commissioner (MCH)
Ministry of Health & Family Welfare

This booklet is addressed to:

- Medical Officers (PHC)
- Project Officers & Supervisors (ICDS)
- Health Administrators
- Programme Managers

engaged in the National Programme on "Prophylaxis against Blindness due to Vitamin A Deficiency".



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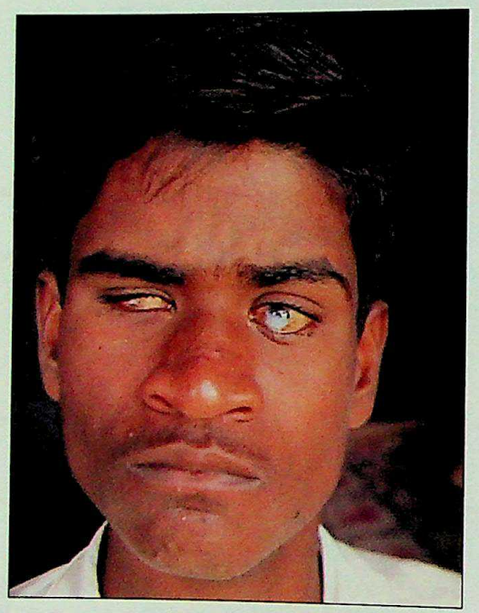


1 VITAMIN A DEFICIENCY IN INDIA

A. VITAMIN A DEFICIENCY PROBLEM

Vitamin A deficiency has been recognised as a major controllable nutritional problem in developing countries. The role of vitamin A in preventing nutritional blindness is well documented. According to rough estimates 30-40,000 children may lose their eye sight due to vitamin A deficiency in India (1). An adequate vitamin A status prevents nutritional blindness. Evidence is accumulating that it also contributes significantly to improved child health as well as reduced mortality. The mechanism by which it has this effect is unclear.

Vitamin A deficiency is a serious public health problem in India

Prevent Vitamin A deficiency. Prevent blindness, and reduce risk of disease and death.

In Indonesia, children with vitamin A deficiency have been shown to be two to three times more prone to develop diarrhoea and ARI (2). These two diseases account for nearly half the childhood deaths in India (3) (Figure 1).

Measles can severely deplete vitamin A stores, leaving a child more vulnerable to other diseases such as pneumonia, diarrhoea, and xerophthalmia. Every year, thousands of children die as a result of measles and its secondary complications. In addition, this can lead to a destruction of cornea and blindness.

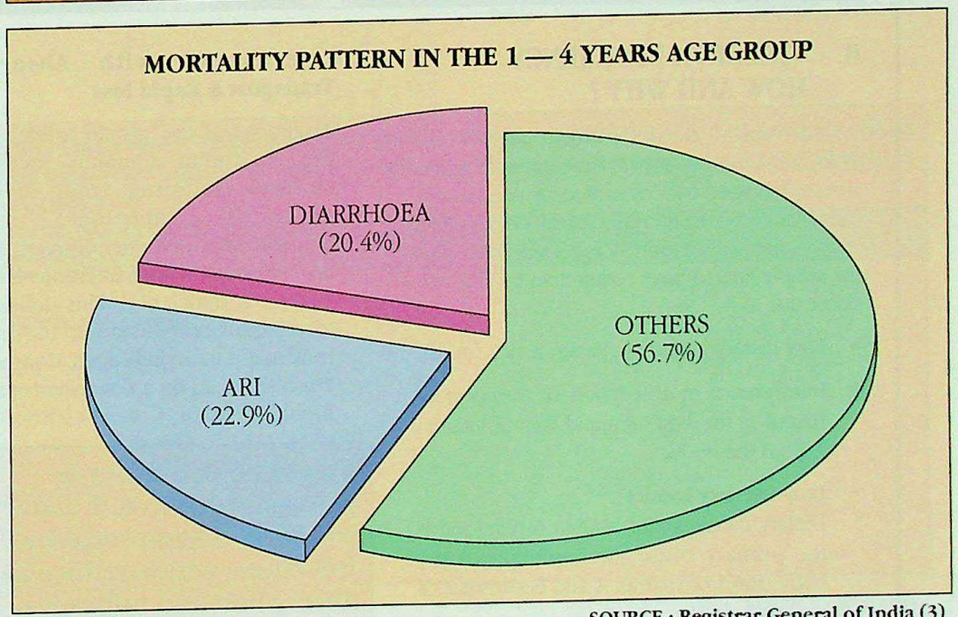
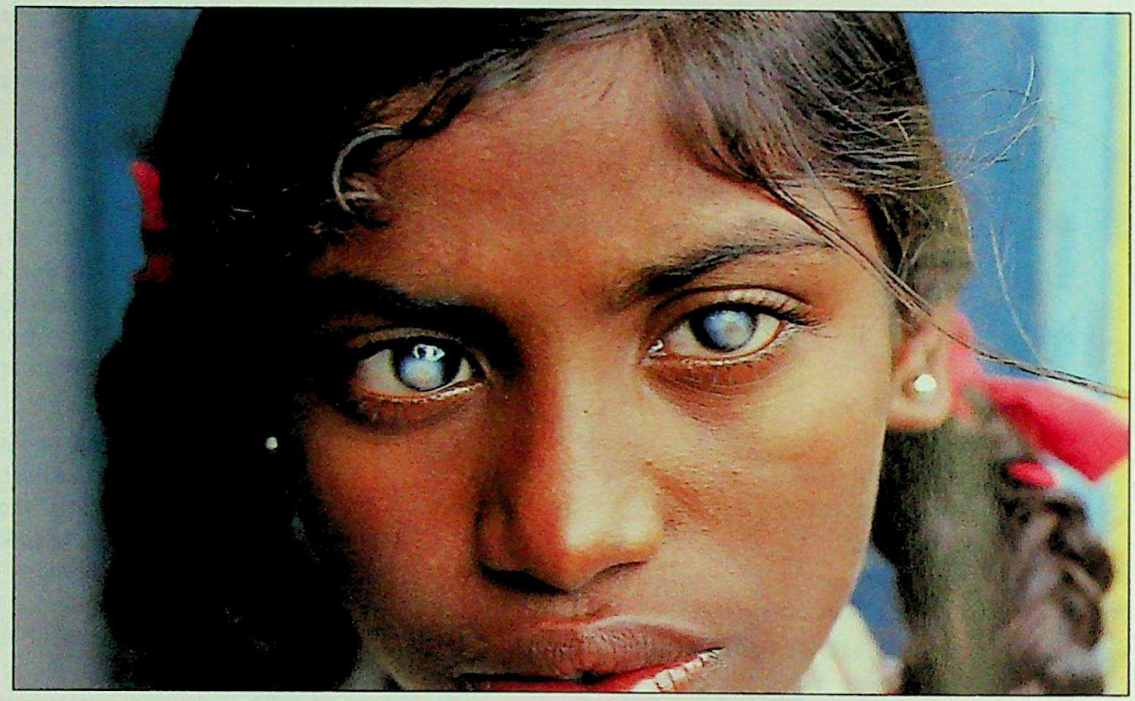


Figure - 1

SOURCE : Registrar General of India (3)



B. VITAMIN A DEFICIENCY HOW AND WHY ?

Vitamin A deficiency results in xerophthalmia i.e. drying of conjunctiva and cornea followed by destruction of cornea and blindness. Xerophthalmia occurs when the body stores of vitamin A are exhausted and the supply fails to meet requirements. This can occur due to :

- Low dietary intake of vitamin A rich foods
- Interference in absorption or transport of retinol in the body & rapid loss of vitamin A from the body
- **Low Dietary Intake**
  Dietary deficiency of vitamin A rich food is the primary cause for depletion of the body stores of vitamin A and occurrence of xerophthalmia.
- **Interference with Absorption, Transport & Rapid loss**

  Absorption of the vitamin is impaired and the metabolic demands increase in children suffering from diarrhoea, respiratory infections, measles, gastroenteritis and other diseases. This sets up a vicious cycle of infections leading to vitamin A deficiency. This deficiency in turn reduces resistance to infection resulting in more infections. Consequently, it sets the child on a downward spiral of ill health.

Vitamin A deficiency is primarily due to low dietary intake. It is aggravated by infections which reduce its absorption and utilization.

2 DIAGNOSTIC CRITERIA FOR VITAMIN A DEFICIENCY

A. OCULAR SIGNS AND SYMPTOMS

Although vitamin A deficiency affects many tissues in the body, it is most detrimental in its effects on the eye resulting in blindness. The surface of the cornea becomes dry and rough, and may ultimately break down partially or completely. This may lead to ulceration, which causes permanent scarring or irreversible damage and loss of sight.

The ocular signs are considered the most reliable criteria for identifying vitamin A deficiency . Severe forms of the disease can appear quickly without preceding milder signs and symptoms appearing in sequential progression.

A classification of the disease according to eye symptoms and prevalence levels above which it is considered a public health problem is shown in Table 1 & 2.

Classification of Xerophthalmia by eye (ocular) signs	
Night blindness	(XN)
Conjunctival xerosis	(X1A)
Bitot's spots	(X1B)
Corneal xerosis	(X2)
Corneal ulceration/keratomalacia < 1/3 corneal surface	(X3A)
Corneal ulceration/keratomalacia ≥ 1/3 corneal surface	(X3B)
Corneal scar	(XS)
Xerophthalmic fundus	(XF)

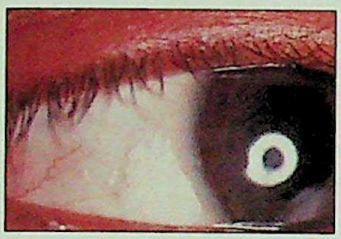
Table - 1 **SOURCE : WHO (4)**

Prevalence criteria (in percentage of preschool-age population, 6 months to 6 years old, at risk) for determining the public health significance of xerophthalmia and vitamin A deficiency.	
CRITERIA	
Night blindness (XN)	in > 1.0 %
Bitot's spots (X1B)	in > 0.5 %
Corneal xerosis / Corneal ulceration/keratomalacia (X2/X3A/X3B)	in > 0.01 %
Corneal scar (XS)	in > 0.05 %
Plasma vitamin A of <0.35 µmol/l (10µg/dl)	in > 5.0 %

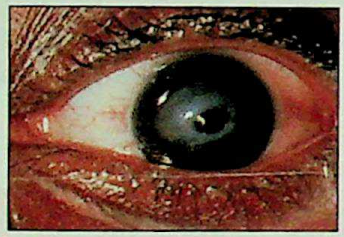
Table - 2 **SOURCE : WHO (4)**

B. RECOGNITION OF THE SIGNS AND SYMPTOMS OF XEROPHTHALMIA:

Night blindness –
The first symptom of xerophthalmia. A child cannot see to get around after dark or in a dark room.

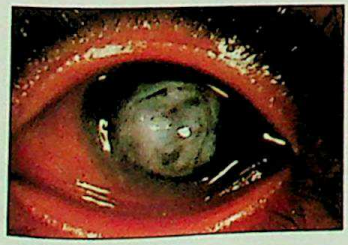


Bitot's spots –
Although Bitot's spots differ somewhat in size, location, and shape, they have a similar appearance. They are accumulations of foamy cheesy material on the conjunctiva, often in association with other signs of xerophthalmia such as night blindness.



Corneal xerosis/ulceration –
The cornea becomes dry (xerosis). If the disease is not treated, the xerosis can progress within hours to an ulcer of the cornea.

Keratomalacia –
If the disease is not treated, a corneal ulcer can lead to "melting" or "wasting" of the cornea (keratomalacia).



Corneal scar –
Keratomalacia can lead to perforation of the cornea. At this stage, a corneal scar will remain in the eye. The sooner the disease is treated, the smaller the ulcer and the smaller the scar which will remain forever. If treated early, corneal scars and blindness can be prevented.

3 THE NATIONAL PROGRAMME

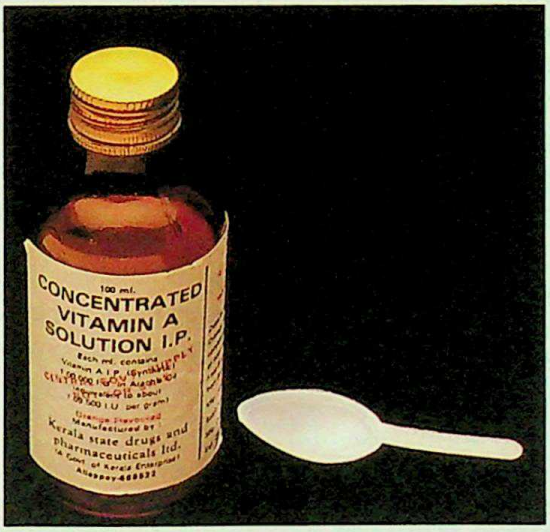
India was the first country in the world to implement a National level Programme for prevention of Vitamin A Deficiency in children under five years of age.

The "National Prophylaxis Programme against Blindness due to Vitamin A Deficiency" was started in 1970, initially in seven states where the incidence of the problem was higher. By 1975, it was extended to other regions of the country as a national programme.

The Programme today covers 30 million children under five years of age and utilizes two major strategies:

A. Short-term : Administration of megadose of vitamin A solution periodically to children under five years.

B. Long-term : Promotion of dietary intake of vitamin A.



Effective intervention can save the sight of millions of children in our country.

A. SHORT TERM STRATEGY

The short term strategy focuses on periodical provision of a massive dose of vitamin A solution to children under five years. This approach is based on the property of vitamin A that it can be stored in liver and utilised slowly over time.

Children under five years are at the highest risk of blindness due to vitamin A deficiency. In the past, pre-school children (1-5 years) were covered under the Programme. The National Programme is now being extended to cover children 6-11 months and a priority is being proposed for children 6 months to 3 years.

Vitamin A supplementation, administered periodically, is effective and safe. When vitamin A is administered in doses as recommended, there are rarely any adverse effects. Side effects such as headache and vomiting may occur sometimes, but these are rare, mild and transitory and do not require any treatment.

Prevention of blindness due to vitamin A deficiency

The recommended oral dose is :

Children 6 -11 months	one dose of 100 000 IU of vitamin A orally. (Measles immunisation is a good time to give a routine dose.)
Children 1-5 years	one dose of 200 000 IU of vitamin A orally every six months.

Treatment of vitamin A deficient cases

All children suffering from vitamin A deficiency must be administered a single oral dose of 200 000 IU of vitamin A immediately

on diagnosis. This should be followed by another dose of 200 000 IU of vitamin A one to four weeks later. Regular consumption of vitamin A rich foods such as dark green leafy vegetables and yellow vegetables and fruits (carrots, pumpkin, sweet potato, papaya, mango) must be encouraged to prevent recurrence.

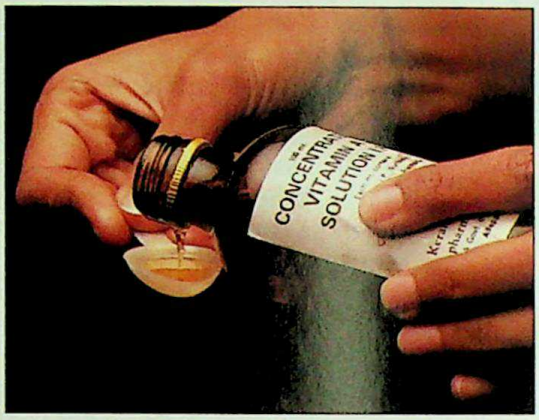
Children who suffer from diarrhoea, acute respiratory infections and measles should be monitored carefully and given usual prophylactic doses of Vitamin A.

Vitamin A Concentrate

Vitamin A solution for the National Programme is supplied as a flavoured syrup in 100 ml bottles with a concentration of 100 000 IU vitamin A/ml. A spoon measure of 2 ml solution is supplied with each bottle. A spoon of syrup to the marked level provides the recommended dose of 200 000 IU vitamin A. Vitamin A does not require any special storage measures such as a cold chain. However, it should be stored in a cool dark room protected from direct sunlight.

The bottle once opened must be utilised within 6-8 weeks. The shelf life of a sealed bottle of vitamin A solution stored in cool dark place is about one year

The price of the syrup is around Rs. 25.00 per bottle of 100 ml or 50 paise per dose of 200 000 IU vitamin A. The solution is provided free of cost to the community through primary health centres (PHCs).



A single spoon measure (2 ml) of 200 000 IU of Vitamin A.

Keep Vitamin A solution away from sunlight. A sealed bottle can be kept at room temperature for one year. However, a bottle once opened must be utilised within 6-8 weeks.

Additionally, fixed dose vitamin A capsules (100 000 IU), are also being supplied for infants. The content of the capsule should be squeezed gently and completely into the mouth of the infant after snipping an end of the capsule with a pair of scissors or a clean razor blade.

Oral Dose -Vitamin A

One dose of 100 000 IU to infants (6-11 months).
Six monthly doses of 200 000 IU to children 1-5 years of age.

Procurement and Distribution

The Ministry of Health and Family Welfare, supplies the vitamin A syrup. The indent of supplies should be made by the medical officer of the PHC on the basis of expected number of beneficiaries i.e. children 6 months to 5 years of age.

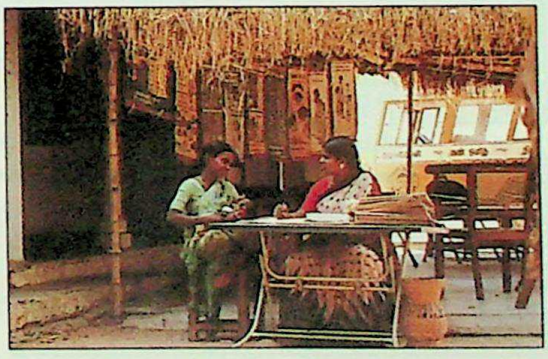
i) Department of Health and Family Welfare

The Maternal & Child Health (MCH) Division, Department of Health and Family Welfare is the nodal department responsible for the overall administration of the Programme. In rural areas the Programme is implemented through the Primary Health Centre (PHC) and its sub-centres, under the supervision of the Medical Officers. The medical officer of PHC is in-charge of indenting of supplies of vitamin A, coverage of beneficiaries and monitoring of the programme.

The Auxiliary Nurse Midwife and other paramedicals at PHCs are responsible for administering vitamin A solution to children.

The Universal Immunisation Programme offers opportunity to reach infants. The contact with children between the ages of 9-12 months for measles vaccination provides an excellent opportunity for administering the first dose of vitamin A solution, 100 000 IU for infants.

ii) Department of Women and Child Development



The Integrated Child Development Services (ICDS) Programme provides an opportunity to distribute vitamin A to children six months to five years. In places where ICDS is functioning, the Anganwadi workers should participate not only in the identification of children but also assist in the administration of vitamin A and in imparting nutrition education.

Monitoring

A child should receive a total of 9 oral doses of vitamin A by the fifth birthday.

The Mother -Infant Immunisation Cards should be used to record and monitor the vitamin A doses administered to children under two years.

All children under six years are being monitored for their growth under the ICDS Programme. The usage of Growth Monitoring Card is a practical method for recording and monitoring administration of vitamin A solution till the age of five years.

Under the MCH services and the ICDS Programme, a list of under-fives is maintained by the health and anganwadi workers. In addition to immunisation and growth cards, such records are recommended to be used for identifying as well as monitoring children for administration of all 9 doses of Vitamin A. A sample format for vitamin A monitoring is given in Table 3.

B. LONG TERM STRATEGY

1. Consideration of Causes

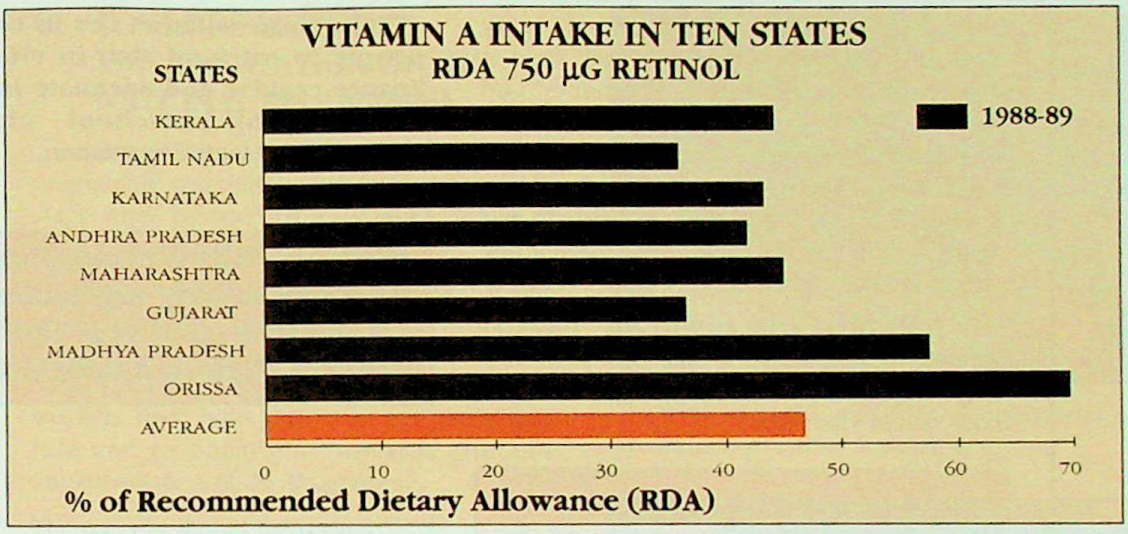
Dietary deficiency is the primary cause for depletion of body stores of vitamin A and occurrence of xerophthalmia.The dietary intake of vitamin A has been reported to be much below the recommended daily allowance (Figure-2) in all the ten states surveyed by NNMB (6).

The vitamin A and carotene (vitamin A precursor) deficient diet of the preschool children, who comprise the 'at risk group' is of particular concern. Their intake is reported to be well below half the recommended allowances of 400 µg of retinol or 1600 µg ß-carotene per day (7-9).

Absorption of the vitamin is reduced in children suffering from diarrhoea, respiratory infections, gastroenteritis, measles and other diseases. The prevention and control of infection & malnutrition by immunisation, proper nutrition, oral rehydration and timely medical care contribute towards the prevention of vitamin A deficiency.

Name of the child	Mother's name	Date of Birth	Sex	Address	Dates of Administration of vitamin A 1	2
1. *Sonu*	*Mangala*	*17.1.88*	*F*	*Katpara*	*17.10.88*	*17.4.89*
2. Pratap	*Krishna*	*29.1.88*	*M*	*Katpara*	*29.10.88*	*29.4.89*

Table - 3 **A sample format for Vitamin A monitoring**



SOURCE : NNMB (6).

2. Ultimate Solution

The ultimate solution for the prevention of vitamin A deficiency is regular consumption of vitamin A rich foods which are cheap and locally available. Vitamin A consumption must be ensured from birth itself. Breast feeding, including feeding of colostrum, will protect children from vitamin A deficiency during the first six months. From the age of four to six months, it is crucial to introduce complementary (semisolids) food containing rich sources of vitamin A such as dark green leafy vegetables and yellow vegetables and fruits (carrots, capsicum, yam, pumpkin, papaya, mango). Most leafy vegetables contain more than the recommended dietary allowance (RDA) of vitamin A for Indian children in about 100g edible portion. This quantity, on cooking, is reduced to two tablespoons. A child can easily consume this amount with other food items, thereby meeting its total daily requirement (10).

In addition to green leafy vegetables as far as possible, incorporation of other vitamin A rich foods such as dairy products (milk, cheese, curd , butter,)liver, eggs, oil and fat should be encouraged in the daily diet of preschool children who are prone to develop vitamin A deficiency. Such food practices will prevent vitamin A deficiency.

Breast feeding protects against vitamin A deficiency during first 6 months of infancy. Colostrum is rich in vitamin A.

Daily consumption of dark green leafy vegetables and other vitamin A rich food is also recommended during pregnancy and lactation. Mother's vitamin A status influences the vitamin A content of mothers milk and the health status of her children. Adequate intake of vitamin A rich food during pregnancy and lactation will prevent vitamin A deficiency both in mother and child.

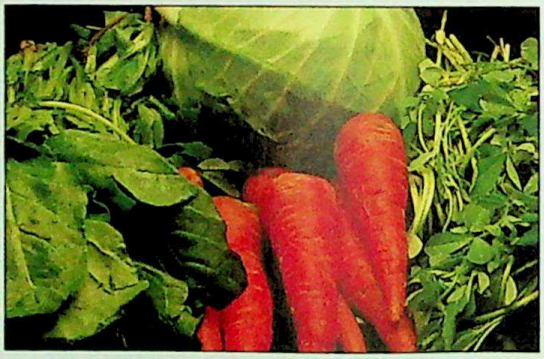
Nutrition education, an integral component of Primary Health Care and ICDS Programmes, must emphasise on promotion of foods rich in vitamin A.

Foods rich in Vitamin A

- **Green leafy vegetables**
- **Yellow fruits and vegetables (Papaya, Mango, Pumpkin, Chillies, Carrots)**
- **Milk , Cheese, Curd, Butter, Eggs**
- **Liver, fishes**
- **Red palm oil**

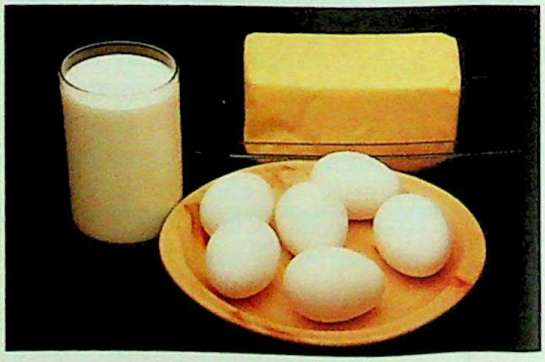
The ultimate solution lies in educating people to eat food rich in vitamin A. Ensure regular and adequate intake of vitamin A by preschool children, pregnant and lactating women.

Promote daily consumption of dark green leafy vegetables and yellow fruits and vegetables to prevent vitamin A deficiency.



Dark green leafy vegetables and Yellow/Orange vegetables

Yellow fruits



Dairy Products & Eggs

C. TOWARDS EFFECTIVE IMPLEMENTATION OF THE NATIONAL PROGRAMME

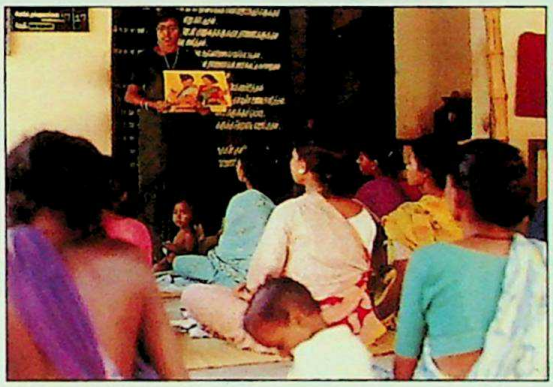
The effective implementation of the programme can only be achieved with trained staff and by creating a high level of community awareness.

The training should be imparted to the entire health delivery team and to functionaries of Integrated Child Development Services (ICDS) Programme. This will help the workers from both sectors in executing their role and responsibility towards effective implementation of the Programme.

The training should focus on :

- Effect of vitamin A deficiency.
- Recording signs and symptoms of vitamin A deficiency.
- Identifying beneficiaries.
- Prevention and Treatment with massive dose of vitamin A including indent of supplies.
- Prevention of vitamin A deficiency by promoting consumption of locally available vitamin A rich foods. Specific messages and effective methods of communication.
- Encouraging community participation.
- Recording system and reporting procedures.

Training of health and ICDS functionaries on prevention of vitamin A deficiency.



Community education on prevention of vitamin A deficiency.

4 SUMMARY

Vitamin A deficiency is a major public health problem among pre-school children in India. According to rough estimates, every year thirty to forty thousand children may become victims of preventable blindness. Vitamin A deficiency increases the susceptibility of children to infections and possibly influences morbidity and mortality.

The benefits of vitamin A supplementation makes the "National Prophylaxis Programme against Blindness due to Vitamin A Deficiency" extremely cost effective.

The health and ICDS administrators and programme managers can form an effective partnership in saving the sight and improving lives of millions of children.

The National Prophylaxis Programme against Blindness due to Vitamin A Deficiency today covers 30 million children under five years of age and utilities two major strategies — short term and long term strategy. The former entails mass distribution of a megadose of vitamin A to children between 6 months to 5 years of age. The latter emphasises the improvement of dietary intake of vitamin A through regular consumption of vitamin A rich foods such as dark green leafy vegetables, yellow vegetables & fruits, dairy products and the promotion of breast feeding. Educating the people and increasing regular consumption of vitamin A rich foods which are cheap and locally available is encouraged since this is the ultimate solution for the prevention of vitamin A deficiency.

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Ministry of Health & Family Welfare, Government of India.
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4

The Evolution and Mutual Adaptation of Insects, Microorganisms and Man

James R. Busvine*
Maidenhead, England

In the course of evolution a remarkable series of adaptations occurred, involving the alternation of parasitism between a large animal (or plant) and a small creature such as an arthropod (commonly an insect). This arrangement was found to be beneficial to organisms originally parasitic on the smaller host, as well as those on the larger ones. . . .

Only a relatively few exceptional [insects] are serious pests . . . a few thousand species of insects have become adapted to feeding on vertebrate blood.

* Emeritus Professor of Entomology, London School of Hygiene and Tropical Medicine.

I. Insect Pests and Disease Vectors

When insects are very troublesome, it is difficult to realize that only a relatively few exceptional forms are serious pests. Chapman (1973) gives a figure of 4500, which is about 0.6% of known species. Most of them damage plants or plant products, as shown by the fact that agriculture claims about 97% of insecticidal usage (Hamon, 1976). Among pests of public health importance, the most serious are the bloodsucking forms, which include important disease vectors.

The mouthparts of primitive insects were adapted for chewing and biting their food, so that substantial modifications were necessary to enable them to pierce tissues and suck blood. These changes evolved several times, in various groups, and in different ways. Probably the earliest adaptation was by the bugs, or Hemiptera, which very early specialized in sucking the sap of plants. They have become a large and successful group, which includes many plant pests. A small proportion turned their attention to attacking other insects and sucking their juices; and from these a few adapted to attacking vertebrates, and some of them will feed on human blood.

The ectoparasitic lice and fleas probably developed from scavenging forms, feeding on debris in burrows and nests, then on skin products among fur and feathers, and finally on blood. Somewhat more puzzling is the widespread habit of bloodsucking in the order Diptera, which includes mosquitoes, biting midges, sandflies and blackflies. In these groups, the males feed only on nectar; but the females, needing protein for egg production, take blood meals. It is not easy to guess how this came about.

The more advanced families of Diptera nearly all lick up liquid food, having lost the piercing stylets used by the more primitive types for bloodsucking. In some of them, however, the habit redeveloped in a different way, as follows. Many flies and blowflies associate with large mammals, such as cattle, and drink up their sweat, lachrymal fluid and the blood from small scratches. A few of these insects have developed rasps on their tongue-like mouthparts, to abrade scabs; and thence, the change to a spine-tipped spike was the next step. This is the mechanism used by both sexes of tsetse flies, stable flies and some others.

In these various ways, a few thousand species of insects have become adapted to feeding on vertebrate blood, mainly of warm-blooded mammals and birds. These emerged about 150 million years ago, but relatively late in the evolution of insects, which probably extend back 300 million years (see Fig. 1). Most of the blood sucking forms are fairly

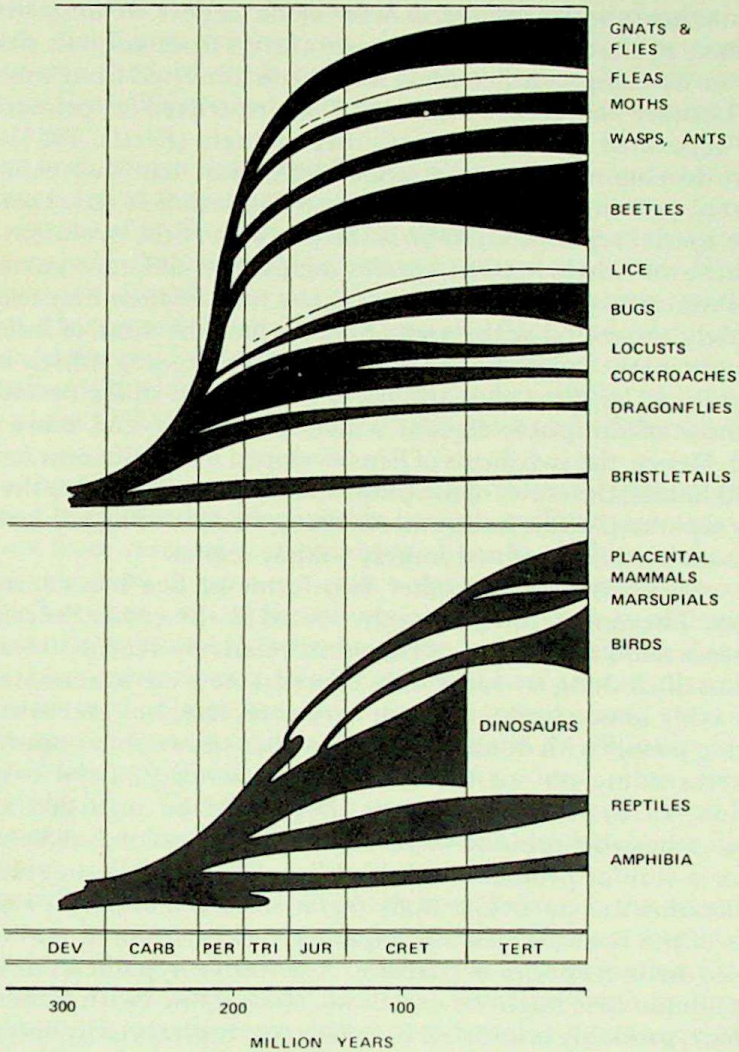

Fig. 1. Simplified evolutionary trees of the insects (above) and the vertebrates (below). Key to geological eras: Dev, Devonian; Carb, Carboniferous; Per, Permian; Tri, Triassic; Jur, Jurassic; Cret, Cretaceous; Tert, Tertiary.

catholic in taste, but some have become adapted to one or a few closely related host species. Man appeared on the scene so late that he has attracted the undivided attention of only a very few parasitic insects; in short, three kinds of lice, which infest his head, his underclothes or his pubic hair. Sucking lice tend to be more closely associated with a

particular host species than most other blood feeders. While some of the latter have probably been accidental transfers from animals sharing a shelter or dwelling, our lice must have been inherited from subhuman stock, because the genera concerned are restricted to primates and widely separated from the lice of other animals (Ferris, 1951). Their further development in association with man has interesting and important practical aspects. First, since speciation is now thought to require spatial separation, there is the problem of the evolution on the same host, of closely related species adapted to different parts of the body. Presumably when our ancestors lost most of their hair except for the widely separated scalp and pubic region, the sites of infestation were sufficiently isolated to prevent the interbreeding which inhibits speciation. It happens that the hairs of the head differ considerably from those of the pubic region, which are thicker and more widely spaced. Hence, the two forms of lice developed quite different morphology and habits, the claws of the pubic louse being adapted to the coarse widely separated pubic hairs and the insect itself settling to a sedentary life, which keeps it confined largely to that region.

The exact status of the other two forms of lice has caused controversy. They are certainly closely related in the genus *Pediculus*, and their separation must have occurred in relatively recent times, when adoption of clothing by early man offered a new environmental niche for the body louse. Isolation is not very complete; but recent evidence involving people with double infestations has shown that opportunities for interbreeding do not blur the distinct identity of the two forms (Busvine, 1978). For this reason, they could be regarded as good species. A possible solution to the separation question has been proposed for a similar problem with bird lice. Clay (1957) suggested that specialization to a particular body region occurred in widely separated groups of the hosts, which subsequently became united and infested with two distinct species of parasite. A tentative hypothesis to account for the human case might be as follows. *Homo sapiens*, with rather sparse body hair, probably originated in a warm or tropical environment. As a result of competition (? with other hominoids) he ventured into colder regions and began to use skins and other clothing. Eventually, his body hair almost vanished and his lice retreated to the scalp in one region and to the clothing in another. Subsequently, mixing of the two groups of men allowed infestation with two species of lice.

So much for the theoretical aspect. Of practical importance is the fact that, though biologically closely related, head lice and body lice present two very different public health problems. Body lice are only prevalent among people who do not change their underwear at all regularly, and

are consequently being eliminated from all but unhygienic communities. Head lice, however, are not necessarily killed by the mildly warm water used to wash hair, and they persist in most civilized countries. Crab lice are widely prevalent in a small proportion of most populations.

It has been shown that head lice, as well as body lice, can transmit relapsing fever in the laboratory; and both, as well as crab lice, could act as vectors of typhus (evidence summarized by Weyer, 1978). But body lice are by far the most important in actual epidemics. Therefore these diseases, though once prevalent in Europe, are now restricted to areas of widespread lousiness in Central Africa and parts of South America. After the First World War some 30 million cases occurred in Eastern Europe, with about 10% deaths (Zinsser, 1935); now the global total is about 15 000 cases annually, with a few hundred deaths (WHO, 1974).

Since these lice are restricted to man, they can only transmit human diseases. Other bloodsucking insects, more catholic in taste, may bring infections from reservoirs of pathogens in wild or domestic animals; alternatively, they too can transmit a strictly human disease, such as malaria. In either case, their willingness to bite man is critical; some will do so very reluctantly, others eagerly and those are the dangerous vectors. The factors responsible for choice of host are complex and not fully understood. Some are quite simple components, such as the warmth, humidity and carbon dioxide emitted by all warm-blooded animals. More specific factors are the body odour, size, shape and colour of the host. These are the criteria used by insects which feed in open country; i.e. many culicine mosquitoes, blackflies, tsetse flies and horseflies.

Apart from the characteristics of the host, however, the actual environment of the encounter may be important. Human dwellings, for example, are attractive to many insects with a predilection for caves and shelters. It is very likely that the common bedbug began its association with man during a cave-living period in human pre-history, since all of its relatives in the highly specialized bedbug family feed on cave-haunting creatures—either bats, or swallows and swifts. Unlike the lice, which have been with us since primordial times, the bedbug only became cosmopolitan during the historical era. The first record of this pest in England was in the sixteenth century (Mouffet, 1634). The other bloodsucking group of bugs, the South and Central American Triatominae, or conenose bugs, are less specialized and degenerate. Primarily adapted to living in the nests and lairs of birds and beasts, some species find ill-constructed rural hovels a convenient alternative.

Fleas constitute a small peculiar group of insects, of uncertain origin, possibly an offshoot from primitive Diptera. Even within the order, there are phylogenetic puzzles due to convergent evolution; that is, modifications to suit a similar habitat, rather than inherited from a common ancestor (Traub, 1971). The larvae are non-parasitic and live on debris in the nest or lair of the adult's host. For this reason, fleas do not parasitize nomadic animals, like ungulates or wandering monkeys (Hopkins, 1957). The so-called human flea, *Pulex irritans*, can infest a considerable number of wild and domestic animals. Farmers will confirm the liability of pigs to be heavily infested. Perhaps this is how the association with man began; as Holland (1969) has pointed out, the primitive human dwelling is not unlike a pigsty and man and pig share several other parasites. The most dangerous disease-transmitting species of mosquitoes are also those which associate with human dwellings. Thus of nearly 500 species of *Anopheles*, only a score or so are important malaria vectors, because of their willingness to bite man, usually indoors. The more numerous culicine species (perhaps 1500) include many tiresome biting pests which attack us in open country. However, two of the most dangerous disease vectors linger near dwellings: *Aedes aegypti*, vector of both urban yellow fever and dengue, and *Culex fatigans*, which transmits filariasis. Sometimes, too, human activities provide convenient breeding sites for particular mosquitoes. Species which naturally tend to breed in polluted water adapt easily to drains and cesspits (for example, *C. fatigans*). Other forms, which prefer small rock pools, find convenient sites in water-filled tins, gutters and miscellaneous debris around dwellings, as does *A. aegypti*.

Apart from bloodsucking insects, there are others which have become pests because human habitations and their contents supply their natural needs. Food stores, of course, are ideal for species of beetles and moths which breed in seeds and dry vegetable matter. Their depredations are serious; but, because of the sedentary habit of the food-destroying grubs, they present no danger to health. The more mobile houseflies and cockroaches, however, may be involved in disease transmission (Roth and Willis, 1957). Not only do they visit and soil human foodstuffs, but they are prone to visit drains and privies in search of water and (in the case of flies) breeding sites; from these, they can import disease germs. This method of pathogens passing to new hosts, however, is not very reliable and generally no more than an alternative to other means of transmission. The involvement of bloodsucking arthropods produced a more efficient system, and it may be of interest to speculate on how this came about.

II. Transmission of Parasitic Microorganism by Biting Insects

In discussing this subject, it is useful to consider the problems which parasitic microorganisms have to overcome (Busvine, 1975). The microorganisms concerned comprise viruses, rickettsiae, bacteria, protozoa and parasitic nematodes. All of them are very small, the largest of the single-celled forms being about 10 to 50 μm long, and the filarial nematodes about 300 μm long by 6 μm thick. Such tiny creatures are, to a large extent "in passive slavery to forces of the environment" (Huxley, 1941); flagella or cilia may transport them a metre or so in an aqueous medium, but generally they are dependent on chance forces of wind and water. This does not matter so much for the great majority, which are saprophytes in soil or some such extensive habitat; but the parasitic forms must sooner or later make the journey to a new host. This is certainly imperative for the pathogenic forms, whose host may sicken and die.

There are various solutions to this problem. If the host is an animal, parasitic microorganisms may colonize another one during social, familial or sexual contacts. Alternatively, they can invade the gut and be passed out with the faeces, with the chance of contaminating food or water of the host. Or they can be coughed or sneezed out in droplets, to be inhaled by another potential host.

In the course of evolution, however, a remarkable series of adaptations occurred, involving the alternation of parasitism between a large animal (or plant) and a small creature such as an arthropod (commonly, an insect). This arrangement was found to be beneficial to organisms originally parasitic on the smaller host, as well as those on the larger ones. For the former, a large animal like a vertebrate represented a big and long-lived reservoir, capable of infecting many of the small creatures over a long period. The parasites of vertebrates, on the other hand, found a new long-range method of transmission. Where human or animal diseases are concerned, the arthropod involved is usually described as the "vector" and the vertebrate as the "reservoir". In some circumstances, however, the roles may not be so obvious. Thus some arthropods, such as ticks, are quite long-lived and may also pass on the pathogen to their progeny (by transovarial transmission), so that they virtually constitute reservoirs. On the other hand, a man infected with malaria or yellow fever and whisked to another continent by air, could certainly spread the disease, though he would normally be called a "carrier". Dengue was recently introduced into the Pacific Islands in this way and imported malaria is increasing in several countries (see Chapter 9).

The arthropods, especially the insects, have proved to be important vectors, because many of them habitually associate with vertebrates; this is particularly true of the bloodsucking ones. The simplest transmission system consists merely in contamination of the mouthparts with blood pathogens. This mechanical method of infection, by various biting flies, is responsible for spreading surra among horses and camels. It may also account for some cases of human sleeping sickness (Buxton, 1955), though most are due to a complex cycle exclusive to tsetse flies, as mentioned below. The virulent rabbit disease myxomatosis is also dependent on mechanical inoculation by biting insects. In Australia the main vectors were mosquitoes (Fenner *et al.*, 1952), whereas in England rabbit fleas were principally responsible (Lockley, 1954).

The method of mechanical transmission, however, is not very efficient, since the very small traces of blood on the mouthparts may not contain enough pathogens to infect a new host. Also, they would soon dry up, which would harm many microorganisms. Therefore, mechanical transmission is only likely to succeed when an insect, disturbed during feeding, immediately resumes on another animal. In subsequent evolution there was more and more radical infection of the vector, and a progression from casual involvement of various bloodsuckers to association with a particular genus of vectors. Probably the first step was for the pathogen to proliferate in the foregut of the vector, thus increasing the chances of infection. This occurs in tsetse flies carrying nagana, a trypanosomal disease lethal to imported horses and cattle in Africa; and also in the human diseases kala-azar and oriental sore, carried by sandflies. Somewhat more efficient is plague transmission by fleas, in which the bacilli multiply to such an extent that they often block the gut, so that some of them are regurgitated when the hungry fleas try to feed again.

Pathogens which multiply in the gut of the vector can also be passed out with its faeces in an infective state. Examples are louse-borne typhus and Chagas' disease. The infective faeces may enter abrasions in the skin, following scratching by the host, or they can dry to a fine powder and become inhaled or enter a mucous membrane.

In a further stage of parasitizing the vector, the pathogens may penetrate the gut wall and invade its tissues, though this complicates the matter of escaping to enter the next host. A simple solution is for the arthropod to be eaten, which must often happen when an animal grooms itself. An example is the transmission of the dog tapeworm, *Dipylidium caninum*, one stage of which occurs in a flea and is released when the dog swallows it. A human disease transmitted in this way is louse-borne relapsing fever, the spirochaetes of which are confined in

the insect's body cavity. Infection can occur when primitive people "pop" lice between their teeth.

There are obvious limitations to a transmission system which involves the death of the vector, which can thus only infect one new host. A better arrangement is for infection to occur during subsequent bites of the vector. A simple example is provided by the forms of filariasis, in which the tiny worms make their way to the insect's proboscis and burst out during the next blood meal. This method of infection occurs with mosquitoes transmitting urban filariasis and the blackflies which carry onchocerciasis. In the final and most efficient system, the pathogens in the arthropod's body make their way to the salivary glands and are injected into one or more new hosts with the saliva. Several widespread and important diseases are spread like this, including malaria, yellow fever, dengue, sleeping sickness, mosquito-borne encephalitis and some forms of tick-borne relapsing fever (see also Chapter 9).

III. The Origin of Two-Host Pathogens

Presumably, microorganisms alternatively parasitic on arthropods and vertebrates began as parasites of one of them and extended their range to the other host. It is not always easy to guess which was the original host. Obviously there is no fossil evidence to trace their evolution, but something can be learnt from the biology of their existing relatives. Thus the Rickettsiae responsible for louse-borne and other forms of typhus belong to a group virtually all of which are parasitic on arthropods at some stage; so these were probably the original hosts. Other parasites which probably began as parasites of invertebrates are the trypanosomes, which cause sleeping sickness, transmitted by tsetse flies, and Chagas' disease, spread by trypanosomal bugs (Hoare, 1967). Other trypanosomes not involving man are carried by leeches to frogs or fishes; and in contrast to the variety of invertebrate hosts involved, there is only one species which passes directly between vertebrates (it causes a venereal infection of equines, almost certainly a secondary adaptation).

At one time it was considered that insects were the primary hosts of malarial parasites, since they are all spread by mosquitoes to a variety of tree-living animals (birds, bats, monkeys and man—who may have once been arborial) (Christophers, 1934). However, further consideration of related parasitic protozoa suggests that the remote ancestors were parasites of vertebrates, perhaps gut parasites. Later, invasion of

the bloodstream allowed the use of blood-suckers as intermediate hosts, and finally, only mosquitoes were involved (Mattingly, 1969).

The relapsing fevers, transmitted by lice and ticks, belong to spirochaetes of the genus *Borrelia*. Both types of arthropod vector are unaffected by their presence and the ticks can pass them on to their progeny; these facts point to a long association with the arthropods (Walton, 1973). If, however, these were the original hosts, *Borrelia* must be an anomalous kind of spirochaete, since other genera are associated with ulcers of the mouth or genitalia or lung abscesses. Furthermore, distant relatives are the treponemes, responsible for the tropical skin diseases yaws and pinta. Still others are free-living and are found in mud, sewage and polluted water, while some occur in oysters and other molluscs. In short, the affinities of *Borrelia* are obscure.

A similar uncertainty prevails in the origin of the plague bacillus, *Yersinia pestis*. Little can be deduced from the characteristics of related bacteria. Only one of these, *Francisella tularensis*, is involved in a disease transmitted by arthropods (tularaemia) and this species differs substantially in morphology and immunology.

IV. Infectivity and Virulence

Whatever the origin of a two-host parasite, the extension to a new host must have involved adaptations analogous to the initiation of the original parasitism. Apart from the mechanical arrangements for transfer, there would need to be development of new enzyme systems to assimilate nutrients from a different type of tissue and also adaptation to neutralize the protective immunological reactions of the new host. These developments must involve complex biochemical changes to meet the particular characteristics of the new animal. Such adaptations are often rather specific, so that many pathogens will only thrive in a limited range of vectors and hosts. Thus the species of *Plasmodium* responsible for human malaria will only develop in man and must be transmitted by anopheline mosquitoes. Sleeping sickness trypanosomes will only develop fully in tsetse flies, though other biting flies may transfer them mechanically.

After the major step of establishing a system of alternating hosts, the microorganisms may extend its range to yet more species. Though not as radical as the original change to an entirely different organism, extension to a new vector or host must involve some adjustments. In the early imperfect stages, this may result in harm to either host or vector; in other words, virulence. In the course of time, however, diseases tend

to evolve to a benign state, with improved chances of survival for both host and parasite; and this condition is generally regarded as evidence of a long-established association between a microorganism and its host.

Many human diseases transmitted by arthropods are almost certainly extensions from cycles in wild animals and their arthropod parasites, which have reached the stage of natural immunity. Examples are scrub typhus, Rocky Mountain spotted fever, Chagas' disease and Rhodesian sleeping sickness. There are, however, other vector-borne diseases carried over from wild animals, in which the latter suffer to various degrees. Thus jungle yellow fever kills many American forest monkeys and "sylvatic" plague periodically decimates wild rodents.

Extension of vector-borne infections of wild animals to man would depend on opportunities for frequent human feeds by the vectors concerned. Occasional infections would be unlikely to establish themselves in the alien tissues. If, however, a change in human habits or the environment allowed the vectors frequent opportunities to feed on man, there would be a chance for a mutant capable of developing in man to establish itself. Human defences would be unprepared and severe virulence could result. In some cases, a further environmental change (or dependence on a new vector associated with man) might isolate the pathogen, so that an exclusively human disease resulted; for example, malaria, typhus or louse-borne relapsing fever. Some of the changes mentioned could have occurred in relatively recent times, corresponding to the environmental changes wrought by emerging human urbanization. Not only would this involve vectors ready to take advantage of the shelter of human dwellings (as mentioned earlier) but also other mammalian reservoirs, in the form of domestic animals or commensal pests, like rats and mice. Examples are typhus and plague.

The pathogen of typhus, *Rickettsia prowazekii*, is closely related to another pathogen, *R. typhi*, which occurs in wild rodent populations and is transmitted among them by various ectoparasites, including fleas. Sometimes a rodent flea bites man and transfers the infection, which is ill-adapted and causes only a mild disease, murine typhus. It is possible, however, that a mutant form of the Rickettsia began to be spread through human populations by the body louse, causing epidemic typhus. It could be that this innovation occurred in relatively recent times, since the Rickettsiae are lethal to lice as well as being dangerous to man; perhaps in historical times, since there is no mention of a disease which can be identified as typhus earlier than the fifteenth century (Zinsser, 1935). Plague records extend back much further; but this may be because that disease is more easily recognized and described.

So far as plague is concerned, there seems to be no evidence of a change in the nature of the pathogen during the course of human history; but there have been profound changes in its distribution and importance (Hirst, 1953). As is generally known, plague is a disease of wild rodents, transmitted among them by their fleas. These do not readily bite man, so that sylvatic plague foci are not dangerous, except to fur trappers. Plague epidemics are begun by the transference of the infection from wild rodents to scavenging rodents on the periphery of human settlements; and from them, urban pest rats acquire the disease, to which they are very susceptible. The presence of numerous dead town rats is a dire warning of impending plague. In temperate climates, the common rat flea, *Nosopsyllus fasciatus*, is reluctant to bite man; but the tropical rat flea, *Xenopsylla cheopis*, will often feed on man, especially if desperately hungry after the death of its rat host, with the possible result of plague developing. Human cases are not infectious to rats, because the plague bacilli are concentrated in the buboes, or swellings in the armpit and groin. Sometimes, however, intense lung infections develop and these pneumonic cases can spread plague by droplet inhalation (see Chapter 14).

The original home of bubonic plague may have been Central Africa or perhaps Central Asia; certainly the latter part of the world was an early source of plague, which travelled with rats and fleas in caravans to cause the epidemics which were recorded throughout history. Camel trains, however, are slow and restricted; but with the great expansion of ocean trade in the nineteenth century, plague was spread all round the world, from a source in China. The first result was a series of urban epidemics in seaports; but from these, new foci were set up in the hinterlands of various continents. There they remain today, among wild rodents in California, South America and South Africa. The chances of their spreading into modern cities, however, is remote, except in some ill-kept tropical towns. This is because the likelihood of a rat flea attacking man depends on the location of the dying rat. The rat common in modern cities, *Rattus norwegicus*, lives underground in sewers, with little or no contact with humans. Whereas the dangerous plague rat *R. rattus*, is a climbing type, which readily invades wooden buildings and was common in medieval towns. This rat is now rare in modern cities except, to some extent, in warehouses.

The great changes in human populations over the past 10 000 years, with the growth of agriculture and later, urbanization, have affected the importance and distribution of many diseases (McNeill, 1977, and this volume) including those transmitted by vectors. Malaria, for example, is well known to have waxed and waned as past civilizations emerged and declined. To some extent, this could be due to the

increased opportunities for breeding of the mosquito vectors, when the destruction and disorganization of war interrupted careful agricultural drainage schemes. This would have brought extra disease to augment the ill-effects of the war and hasten the decline of a civilization.

In recent times, we have seen a marginally successful attempt to eradicate malaria from the world, by attacking the vectors with insecticides, especially DDT. Though effective in eliminating the disease from very large peripheral areas, the attempt failed in the hyperendemic tropical zone, largely because the mosquitoes developed resistance to the insecticide (Harrison, 1978) (see also Chapter 10).

Another mosquito-borne disease where the incidence is changing because of human development is urban filariasis. Nelson (1977) has pointed out that, with about 400 million people at risk, there are probably more infected cases now than 100 years ago, when Patrick Manson incriminated mosquitoes as the vectors. This is largely due to population growth in the tropics, combined with the enormous growth of unhygienic slums around tropical cities, which have provided a vast increase in breeding sites for the urban vector, in drains and cesspits. This insect, too, has become largely immune to chemical pesticides.

At one time, the new potent synthetic insecticides seemed to promise an easy solution to vector-borne disease. In the last 10 to 15 years, however, their use has been hampered by increasing concern about environmental pollution and by pest resistance. The first difficulty can be overcome by using less persistent and more specific insecticides (though they are more expensive); but resistance remains as a severe handicap. Nevertheless, insecticides are likely to play a valuable role for a decade or two, if carefully used in combination with other measures (Busvine, 1977). The optimum solution for many diseases is a general improvement in living standards. So far as vector-borne diseases are concerned, this should eliminate ectoparasites such as lice and fleas, as well as many house pests like bugs and triatomids. Better sanitation (especially water-borne sewage) should greatly reduce *Culex fatigans* and many flies and blowflies. Raising living standards, alas, is an enormous task. Moreover, there are some vectors which originate beyond the effects of personal or urban hygiene, such as blackflies, tsetse flies and many anopheline mosquitoes. These present a challenge to physicians, entomologists and chemists, to find suitable drugs and pesticides. Much, too, depends on the importance accorded to these diseases by politicians and, indeed, the people they represent. One sometimes wonders what progress could have been made if they had attracted the money and effort expended on atomic weapons or even on rockets to explore space.

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This document results from the meeting of the WHO Working Group on Parkinson's Disease held at WHO, Geneva, 27 & 28 May 1997. The following experts participated:

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Dr Leslie Findley, UK
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Dr Oscar Gershanik, Argentina
Ms Liz Graham, UK (Rapporteur)
Dr William C. Koller, USA
Dr Douglas MacMahon, UK
Dr Matthew Menken, USA (Chairman)
Mr Simon Morris, UK (Observer)
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PARKINSON'S DISEASE AND PUBLIC HEALTH

EDUCATIONAL AND MANAGEMENT IMPLICATIONS

Introduction

Movement disorders contribute substantially to the burden of illness globally. The prototypical movement disorder is Parkinson's disease (PD), first described by James Parkinson in 1817. However, many patients have difficulty with spatial and temporal patterns of movement, without paralysis, yet are often excluded from movement disorder categories because of the conventional disease classification categories commonly used. For example, patients with multiple lacunar strokes, including many individuals with diabetes mellitus and hypertension, have some degree of rigidity and bradykinesis that is poorly responsive to currently available pharmacotherapy. These people would be classified on morbidity and mortality tables as having cerebrovascular disorders, yet an important part of their disability reflects the impact of the movement disorder upon their quality of life. Likewise, patients with schizophrenia and other mental illnesses may have their disorders of thought reasonably well controlled with neuroleptic therapy, yet be unable to function effectively in the workplace or participate in the social life of the community because of a medication-related movement disorder that would not be reflected in national data banks of morbidity.

This document has been prepared by Dr M. Menken (World Federation of Neurology) and Dr A. Janca (World Health Organization).

Even if one limits data to PD as such, the annual incidence for this disorder is about 20 per 100,000 people, and the prevalence about 165 per 100,000 people. The average general practitioner (primary care physician) available to provide care for 2000 people will therefore encounter 2 new patients every 5 years. A neurologist available to provide care for 250,000 people would have 50 new patient encounters annually for PD, whereas a neurologist who is available to provide care for only 25,000 people will have 5 such encounters.

To delineate the public health aspects of PD, and to enhance care for all people with PD, a Working Group on Parkinson's Disease was established by the Division of Mental Health and Prevention of Substance Abuse of the World Health Organization (WHO). The Working Group consisted of 12 physicians including representatives of the World Federation of Neurology, WHO and several experts in Parkinson's Disease as well as 3 non-physicians, including the President and Vice-President of the European Parkinson's Disease Association. Of the 15 participants, 2 suffer from PD. The Group met in Geneva, Switzerland, during May 1997 and its discussions and recommendations were focused upon the public health aspects of PD including: Epidemiology; Organization of Services and Treatment; Education, Training and Information; and Direct and Indirect Costs of Care. This publication follows the consensus and recommendations of the Group and is specifically focused on educational and management aspects of PD. Issues related to the epidemiology of PD will be addressed in a separate document.

Educational Implications

Clearly, all medical students and other health care workers need to learn about PD. But what shall they be

taught? They certainly need to have some information from the basic sciences about the physiology, biochemistry, and pathology of the disease, and they need the opportunity to have sufficient clinical exposure to patients with PD under competent mentoring. In sum, they need to have the appropriate information, skills, and attitudes needed to diagnose and treat PD patients whom they are likely to encounter from time to time in most clinical practices. But is this enough? Should not medical education look beyond the care of individual patients and provide all future doctors and other health care providers with a population-based perspective about the disorders they study? Should not doctors be trained as educators, not only of their students and allied health personnel, but also as teachers of the public at large? Should not doctors acquire the attitudes that enable them to learn open-mindedly from their patients' experiences and insights, and from social support groups within society?

In support of WHO's Health for All programme, the Edinburgh Declaration (1988), sponsored by the World Federation for Medical education (WFME), outlined 12 principles for reform of medical education. Emphasis was given to national health priorities in curricular design, an examination system that focused upon competence and values (not mere retention and recall of facts), and a community setting for learning experiences, among others. In 1993, a Working Group for Neurology, including representatives of WHO and WFME, showed how these 12 principles for reform of medical education might be applied to a specialized clinical field such as neurology. This Group called for collaboration among teachers representing several disciplines in undergraduate medical education, to include primary care educators in the teaching of neurology to medical students, and recommended that doctors be generally educated in the cultural traditions of

the communities in which they provide care. More recently, the World Health Assembly urged WHO to undertake reform in the education of health care workers in further support of Health for All, including the concept that quality in medical education be measured in part by the relevance of educational programmes to the health needs of individuals and communities.

Recommendation 1

The WHO Working Group on Parkinson's Disease urges support for these global efforts to reform medical education in support of the WHO Health for All initiative. Steps to increase public awareness of PD as a priority health problem should be pursued vigorously, including the collection of salient information about all aspects of PD, (epidemiology, available treatment modalities, costs of care, consequences to patients and families resulting from a loss of independence, types of care givers and support services for PD sufferers, and so forth), along with widespread dissemination of this information to the public, health authorities, medical schools, and teaching hospitals. Experts in PD should acquire the attitudes necessary to participate in health care teams, especially in those situations where other groups of health workers or patients serve as team captains.

Organization of Services and Treatment

Since PD is treatable, but not yet curable, the duration of treatment grows ever longer as new medications become available. The aging of the population has increased the number of patients with PD who make demands upon the

health services of all countries. The way in which health services are organized and financed impacts critically upon the comprehensiveness of care for patients. Moreover, there is a need to study carefully the range of health care, social and other services received by these patients as a function of social class or socioeconomic status. The care received by patients with PD may be considered within three overlapping time frameworks, or tiers: first, diagnostic investigations and treatment options during the early years of PD, when response to medication is usually quite good; second, maintenance therapy as the disease progresses and becomes progressively more difficult to control sufficiently for patients to function effectively in the workplace, and to discharge activities of daily living without assistance; third, palliative care, when patient comfort and relief for care givers is all important, since no presently available pharmacologic agents control physical symptoms amply, and cognitive and psychological deterioration are likewise difficult to treat.

Clearly, neurologists alone cannot meet all of the care requirements of patients with PD. The World Health Organization in 1977 proclaimed that Health for All in each nation is contingent upon an effective primary care infrastructure for each health system. In developing countries, this will mean that primary health care is obtained in community health centers, and provided by primary care workers whose training is measured in months. In more affluent nations, nurses and general practitioners (primary care physicians) will provide the majority of services. It is part of the Health for All concept that patients usually obtain access to specialized services upon referral from primary care. In the USA, the transformation to integrated care networks that rely upon primary health care (so-called managed care), has demonstrated that even a rich nation cannot provide a full

range of services for its people at a cost that society can afford when health services are organized to deliver services on a specialty-by-specialty basis, without coordination of effort.

Recommendation 2

The WHO Working Group on Parkinson's Disease recommends that all health authorities worldwide support the WHO Health for All concept, and implement the programme in the case of PD consistent with the resources that are available at each stage of industrial development. To provide the greatest number of the most important services for the largest number of people, priority health problems must be identified in each society. For disorders such as PD, there should be a national steering group comprised of patients, care givers, and national organizations that advocate patients' interests, and should include health care providers of all types, including doctors who represent specialist, subspecialist, and general practitioner viewpoints and experiences.

Since current pharmacologic therapy is for some patients with PD a qualified success, and for other patients a failure, a dual population-based approach that promotes biomedical research to develop new therapies should be coupled with development of support services for people whose PD is now poorly responsive to available medications and other treatment modalities at disparate stages of the disease trajectory.

Recommendation 3

The WHO Working Group on Parkinson's Disease recommends that health authorities take steps to achieve coordination of effort by health workers within the three-tier model of service delivery presented in broad outline above, and to arrange care that is structured in accordance with results of cost-effectiveness studies of alternative provider arrangements and sites of care provision across the full spectrum of illness.

Direct and Indirect Costs of Care

All societies now recognize that it is impossible to provide all of the care that might possibly be of benefit to all of its citizens at an affordable cost. For patients with PD, there are often huge costs that quickly exhaust family resources unless there is a comprehensive social insurance scheme or national health service in place. A Delphi panel examining national needs for neurologists in the USA estimated that the average patient with PD would require 140 minutes of a neurologist's time during the first year of illness (50 minutes during the first visit and a total of 90 minutes during subsequent visits), and would require 60 minutes annually during the remaining 9 years of illness. The direct (accounting) cost to patients includes not only the cost of primary care and specialist services, medications, laboratory tests, and so forth, but also the many hidden costs that arise as a result of falls, incontinence, dementia, psychological changes including depression, and sometimes residential care.

Moreover, there are many indirect costs such as loss of earnings, inability to use public transportation, the burdens

that accrue to care givers, and the opportunity costs resulting from the time lost obtaining care and coping with disabilities and handicaps which could otherwise be spent in economically productive activity or at leisure. Finally, there are psychological costs that arise for sufferers from negative attitudes by the public about PD as a hopeless, end-stage illness for which little can be done other than temporary palliation.

Recommendation 4

Given total national costs and individual financial burdens that exceed private and governmental resources, the WHO Working Group on Parkinson's Disease recommends a partnership between neuroscientists and health workers to devise ways to improve access to needed care and treatment for all PD patients, and to foster practice guidelines to assist health care workers in the management of medication side effects, especially among the elderly.

Recommendation 5

The WHO Working Group on Parkinson's Disease likewise recommends a partnership between doctors and other health care workers with voluntary (non-governmental) organizations that represent patient interests to promote better public understanding of PD, to reach out to all ethnic and cultural groups of patients, and to overcome negative attitudes in society toward chronic neurologic and psychiatric illness.

Conclusions

The WHO Working Group on Parkinson's Disease urges support for global efforts to reform medical education in support of the WHO Health for All initiative. Steps to increase public awareness of PD as an important health problem should be pursued vigorously, including the collection of salient information about all aspects of PD, along with widespread dissemination of this information to the public, health authorities, medical schools, and teaching hospitals.

It is an integral part of the WHO Health for All concept that patients with PD will usually obtain access to specialized services upon referral from primary care. The Working Group recommends that all health authorities worldwide support the WHO Health for All concept, and implement the programme in the case of PD consistent with the resources that are available at each stage of industrial development. To provide the greatest number of the most important services for the largest number of people, priority health problems must be identified in each society. For disorders such as PD, there should be a national steering group comprised of patients, care givers, and national organizations that advocates patients' interests, and includes health care providers of all types.

Given total national health care costs and individual financial burdens that exceed private and governmental resources, the Working Group recommends a partnership between neuroscientists and health workers to develop more effective treatments, as well as a partnership between doctors and other health care workers with voluntary (non-governmental) organizations that represent patient interests.

Acknowledgement

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Mortality from neonatal tetanus in Indonesia: results of two surveys*

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Two, 30-cluster, retrospective surveys of deaths from neonatal tetanus in Indonesia were conducted during 1982. The first survey, in the city of Jakarta, identified 16 deaths from neonatal tetanus among 2310 live births, giving a mortality rate of 6.9 per 1000 live births. The second survey covered 19 of Indonesia's 27 provinces. Fifty-three neonatal tetanus deaths occurred among 4971 live births, giving a mortality rate of 10.7 per 1000 live births. Overall, 68.8% of mothers interviewed in the second survey received antenatal care on at least two occasions when tetanus toxoid was, in principle, available.

Cases of neonatal tetanus are frequently observed in hospitals in Indonesia. However, since most deaths from neonatal tetanus occur at home, hospital data provide little information about overall mortality from the disease. Two retrospective surveys were therefore carried out to gain more information about the extent of the problem (*1*, *2*). The surveys had two objectives:

—to estimate the incidence of neonatal tetanus in Indonesia;

—to establish a baseline against which the impact of immunization efforts could be measured in the future.

For this purpose, neonatal tetanus was defined using the following criteria recommended by WHO:[a]

—history in the neonate of normal suck and cry for the first 2 days of life;

—history of onset of illness between 3 and 28 days of life;

—history of inability to suck followed by stiffness and/or "convulsions";

—death within the first month of life.

The first survey, conducted in urban Jakarta, was implemented on 1-13 March 1982. The second survey was carried out on 14-30 June 1982 in 19 provinces on various islands of Indonesia. Eight of the 27 Indonesian provinces were excluded from the survey: five on Java that had previously been surveyed, and three where transportation would have been very difficult.

MATERIALS AND METHODS

A modified version of the 30-cluster sample method was used in both surveys (*3*).[b] By this means, 77 live births were identified in each cluster in Jakarta, giving a total of 2310 live births. In the provincial survey, 4971 live births were identified, i.e., approximately 165 live births per cluster. Both surveys were retrospective, the mothers recalling all births and deaths during the preceding 13 months, and both were conducted by 30 interview teams, each consisting of a local midwife or nurse and a local government guide. In the Jakarta survey, groups of three teams were supervised by a senior nurse, and in the provincial survey each team was supervised by a graduate student from the University of Indonesia School of Public Health. Interviews were conducted strictly on a house-to-house basis from a random starting-point. Data were recorded on two forms. One was used to register household visits, list all births and deaths of infants over the preceding 13 months, and record the type of antenatal and delivery

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[a] *Provisional guidelines for the diagnosis and classification of the EPI target diseases for primary health care surveillance and special studies*. Unpublished WHO document EPI/GEN/83.4.

[b] *WHO Regional Offices for the Eastern Mediterranean and South-East Asia. Prevention of neonatal tetanus*, Report of a Meeting, Lahore, 22-25 February 1982. EMRO Technical Publication No. 7, SEARO Technical Publication No. 3.

care received by the mothers. The second form was used to record clinical data on neonatal deaths.

RESULTS

In the Jakarta survey, 2310 live births were identified in 8811 households. Of the 40 neonatal deaths recorded, 16 resulted from tetanus. The overall neonatal mortality rate was 17.3 per 1000 live births, while the mortality rate for neonatal tetanus was 6.9 per 1000 live births. Table 1 summarizes the antenatal history of the 16 mothers whose infants died from neonatal tetanus and their contact with health services where tetatus toxoid was potentially available; 13 (81%) of them received antenatal care in a hospital or community health centre or from a trained midwife. Also shown is the delivery care history by source and type of attendant. Eight of the 11 mothers whose babies were born at home and were delivered by a traditional birth attendant (*dukun*) received antenatal care at a hospital or community health centre or from a trained midwife.

The provincial survey, which comprised five urban and 25 rural cluster sites, covered 18 633 households. In the survey year, 4971 live births were recalled. During the same period, 104 neonatal deaths, 53 of which were from tetanus, were identified. This gives an overall neonatal mortality rate of 20.9 per 1000 live births and a mortality rate from neonatal tetanus of 10.7 per 1000 live births.

It is noteworthy that 68.8% of mothers in the provincial survey received antenatal care at health services where tetanus toxoid was potentially available. Of the 4971 women who delivered children in the 13 months preceding the survey, 3420 had contact at least twice with a health facility (Table 2). Of these women, 728 (21%) had received two doses of tetanus toxoid, 328 (10%) one dose, while 2364 (69%) either had not been inoculated or did not know their immunization status. In 33 (62%) of the deaths from neonatal tetanus there was no contact with antenatal health facilities.

Table 1. Antenatal and delivery care history involving 16 deaths from neonatal tetanus, Jakarta, Indonesia, 1981–82

Source of antenatal care	No. of mothers	Source of delivery care	No. of deaths
Hospital	3	Hospital + *dukun*[a]	1
Community health centre	4	Hospital + midwife	1
Trained midwife	6	Community health centre + midwife	1
Private physician	—	Home + midwife	2
Dukun[a]	—	Home + *dukun*	11
None	3		

[a] *Dukun*: traditional birth attendant.

Table 2. Number of antenatal contacts at health facilities by mothers surveyed in the 19 Indonesian provinces, June 1982

No. of contacts with health facilities	No. of births	No. of neonatal tetanus deaths
None	1465	33
One	86	5
Two:		
with 2 doses tetanus toxoid	728	1
with 1 dose tetanus toxoid	328	3
with no dose or did not know	2364	11
Total	4971	53

DISCUSSION

In both studies described here the number of deaths from neonatal tetanus were identified by retrospective survey, and the results obtained may therefore underestimate the true situation. Retrospective surveys depend on the interviewee's ability to retrieve information and the interviewer's persistence. For cultural reasons many Indonesians are reluctant to mention a death involving a child younger than 1 year of age. The results obtained therefore represent minimum estimates of the mortality from neonatal tetanus.

Based on our results, we estimate that the minimum mortality from neonatal tetanus is 1850 per annum in the city of Jakarta and 20 000 per annum in the 19 provinces surveyed. A recent prospective survey in the province of Jawa Barat, an area not covered in our survey, indicated that the annual mortality rate from neonatal tetanus was 14.7 per 1000 live births (*4*). For Indonesia as a whole we estimate the annual number of deaths from this cause to be approximately 71 000 (Table 3). This is based on a conservative estimate of the crude birth rate (35 per 1000 population), a population of 7.5 million for metropolitan Jakarta, a population of 52 million for the 19 provinces in the survey, and a total Indonesian population of 155 million. Since the survey did not

Table 3. Estimated mortality from neonatal tetanus in Indonesia

Area	Estimated mortality rate for neonatal tetanus (per 1000 live births)	Population (million)	Estimated deaths from neonatal tetanus[d]
Jakarta[a]	7	7.5	1 850
19 Provinces[a]	11	52	20 000
Irian Jaya, Maluku, and Timor Timur[b]	11	5.3	2 050
Java[c]	15	90	47 250
Total		154.8	71 150

[a] Retrospective survey, 1982.

[b] Estimated from the rate determined in survey of 19 provinces.

[c] Prospective survey 1983 (*4*).

[d] Crude birth rate estimated as 35 per 1000 population.

Table 4. Attack rate as a function of the number of doses of tetanus toxoid reported by mothers in the survey of 19 Indonesian provinces, June 1982

No. of doses of tetanus toxoid	No. of deaths from neonatal tetanus	No. of infants at risk	Attack rate (%)
2	1	728	0.14
1	3	328	0.91
None	49	3915	1.25
Total	53	4971	1.07

cover the provinces of Timor Timur, Irian Jaya, and Maluku, we have used for the purpose of Table 3 the mortality rate found for the 19 provinces in our survey.

Two doses of tetanus toxoid given to previously non-immunized women during pregnancy provide enough transferable immunity to protect a newborn infant.[c] A study in India has indicated that protection from two doses is 73–91% effective, depending on the interval (4–16 weeks) between doses (*5*).

Table 4 summarizes data on deaths from neonatal tetanus according to the vaccination status of the mother. Vaccine efficacy, i.e., (attack rate, unvaccinated – attack rate, vaccinated)/(attack rate, unvaccinated), was calculated to be 88.8% (*6*).

UNICEF has sponsored schemes to train or retrain traditional birth attendants throughout Indonesia. Of the estimated 102 000 attendants, 61 000 participated in such a training scheme between 1979 and 1983 and were issued kits for cutting umbilical cords. Before 1979, about 13 000 attendants were trained, and the balance of approximately 28 000 have not yet completed training (*7*).

In Jakarta, second dose tetanus-toxoid immunization of pregnant women increased from 0.8% to 9.3% in the two-year, post-survey period (H. M. Sjatar Malik, unpublished observations, 1984).

When they receive antenatal care, the majority of women come into contact with potential sources of tetanus toxoid. There is therefore a need to increase the commitment of medical staff to immunizing these women. Extension of the use of tetanus toxoid to inoculate every woman of reproductive age who visits a community health centre or hospital clinic is being considered. In future, progress made in reducing neonatal tetanus will be monitored in three ways: surveillance at urban hospitals, surveys of the extent of antenatal inoculation with tetanus toxoid, and follow-up retrospective studies of neonatal tetanus mortality.

One priority of the fourth 5-year plan in Indonesia is to substantially reduce the infant mortality rate. The finding of the two surveys reported here that 40–50% of neonatal deaths in Indonesia are due to tetanus has heightened awareness that this infection is responsible for 15–20% of the total infant mortality rate in Indonesia.

[c] Galazka, A. *Immunization of pregnant women against tetanus.* Unpublished WHO document EPI/GEN/83.51.

RÉSUMÉ

MORTALITÉ PAR TÉTANOS NÉONATAL EN INDONÉSIE: RÉSULTATS DE DEUX ENQUÊTES

Deux enquêtes rétrospectives utilisant une version modifiée de la méthode des trente grappes, et portant sur le nombre de décès par tétanos néonatal, ont été réalisées en Indonésie en 1982. Lors de la première enquête, en ville de Djakarta, on a recensé 2310 naissances vivantes et 16 décès par tétanos néonatal dans 8811 foyers, ce qui donne un taux

de mortalité par tétanos néonatal de 6,9 pour 1000 naissances vivantes. Il est intéressant de noter que sur les 16 mères dont l'enfant est mort de tétanos néonatal, 13 avaient reçu des soins anténatals dans un hôpital ou un centre de santé communautaire ou avaient été vues à domicile par une sage-femme qualifiée.

La deuxième enquête a couvert 19 des 27 provinces de l'Indonésie. Sur les 30 grappes, 5 étaient choisies dans des sites urbains et 25 dans des sites ruraux. Pendant la période d'enquête, on a recensé 4971 naissances vivantes dans 18 633 foyers. Le nombre de décès par tétanos néonatal s'élevait à 53, ce qui donne un taux de mortalité de 10,7 pour 1000 naissances vivantes. Parmi les mères interrogées lors de cette deuxième enquête, 68,8% (3420 sur 4971) s'étaient rendues au moins deux fois pour une visite anténatale dans des centres où, en principe, on disposait d'anatoxine tétanique; toutefois, 21% (728) seulement de ces mères avaient reçu deux doses d'anatoxine.

En regroupant les données de mortalité par tétanos néonatal obtenues lors de nos deux enquêtes (1800 décès par an en ville de Djakarta et 20 000 décès par an dans les provinces) avec les résultats récemment publiés d'une enquête réalisée à Java, nous avons calculé que plus de 70 000 nouveau-nés meurent chaque année de tétanos néonatal en Indonésie.

Le quatrième plan quinquennal indonésien compte parmi ses priorités la réduction du taux de mortalité infantile et a désigné comme meilleur moyen d'atteindre cet objectif la lutte contre le tétanos néonatal au moyen de la vaccination.

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Walter W. Holland & Ellie Breeze

Good life-styles for good health

Chronic noncommunicable diseases, long responsible for death and disability on a large scale in developed countries, are now becoming highly significant in the developing world. Much can be done towards their prevention by encouraging people to adopt healthier life-styles. Ways of going about this are considered below.

In many developed countries, infectious diseases have been overtaken as the major cause of mortality by accidents and noncommunicable diseases, particularly circulatory disorders, stroke, malignancies, and chronic bronchitis. Other chronic disorders, including musculoskeletal diseases, psychosocial conditions, congenital abnormalities, diabetes, and dental caries contribute to the burden of morbidity and disability.

As developing nations overcome the ravages of endemic infectious diseases and become more industrialized and affluent, they too can expect an upsurge in noncommunicable diseases. This is indeed already occurring in some rapidly expanding urban and industrial centres *(1)*. The control of noncommunicable diseases should therefore be considered as a global problem, not one restricted to the developed nations.

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Community-based prevention

Despite enormous advances in medical knowledge and technology, clinical developments have reduced the incidence of chronic diseases to only a modest degree. As a consequence there has been growing interest in and support for preventive measures. This trend has been encouraged by evidence from some countries of a decline in cardiovascular disease mortality *(2)*, not apparently reflecting advances in medical technology but possibly resulting from changed attitudes in society towards diet, smoking and physical fitness.

Health care costs, especially those for acute care, have continued to escalate, and this has attracted political interest in prevention. Whether preventive measures can reduce expenditure is, however, a matter for debate, since they are not necessarily cheaper than conventional treatment. Furthermore, a longer productive life and an improvement in its quality must be seen in the context of the requirements of an increasing population of old people for welfare services.

There are already good grounds for implementing a wide range of preventive

On the positive side, the fact that the campaign was national brought ready cooperation and a feeling of being part of a larger enterprise. Health education staff, a tiny minority in the National Health Service, found that the Great British Fun Run brought their work from the shadows into the light, and led to an increased appreciation of health education and health promotion.

At the evaluation conference there was considerable debate on whether health festivals were cost-effective. The general view was that any benefits were likely to be long-term and difficult to measure. However, there was considerable support for the concept of a nationwide health promotion week with clearly defined aims, based on a similar concept of cooperation between the national and local levels.

Looking back at the Great British Fun Run, it is clear that the benefits far outweighed the problems. The attendance at health festivals, the weight and quality of mass media coverage, the stimulus to local action and cooperation, the prominence given to the ideals of health education and health promotion, and the enthusiasm engendered can all be seen as gains.

Our feeling is that the running element, exciting though it was and useful as it was to stimulate the organization of health festivals, need not be repeated. We believe that in a future national campaign the health festivals will be able to stand on their own.

In general, though, I hold firmly to the belief that sporting events provide an admirable means of stimulating action to promote health, whether at the international or the national level. Runs, bicycle races and football matches can all serve as a focus for the messages of good health.

The running element need not be repeated. We believe that in a future national campaign the health festivals will be able to stand on their own.

There is every reason to recommend the strategy and concepts outlined here to any nation wishing to adopt a fresh approach to health promotion. It is important to remember, however, that sufficient time should be allowed for planning, sufficient funds should be allocated to ensure a significant impact, as many people as possible should be involved, full evaluation should be carried out, and close coordination should be ensured. Finally, it is important to realize that any such exercise will be very complex and will involve much hard work. □

measures, even though much research is still needed in this field. The prospect of preventing chronic diseases has attracted considerable attention, but individual programmes have not proved as successful as was hoped. Antismoking campaigns, for example, have given disappointing results. This highlights the urgent need to intensify and coordinate efforts to devise practical, acceptable and effective measures of prevention.

Sanitary reforms introduced in the 19th century were broad-based public health measures that raised the overall level of health and controlled a number of infectious diseases. Specialization in clinical medicine has led to a tendency for the prevention of each noncommunicable disease to be considered in isolation. Measures against individual risk factors have been introduced piecemeal into health care systems. The potential for implementing an integrated, community-based approach to the prevention of noncommunicable diseases is suggested by epidemiological, logistical and organizational considerations *(3)*. This type of prevention involves a range of activities directed against indigenous chronic diseases. It makes optimum use of available communication channels and is coordinated by the primary health care team in cooperation with the community.

Risk factors

Many chronic diseases are multifactorial in origin, and various risk factors are shared by several diseases: cigarette smoking is the most often reported, other important ones being poor diet, excess weight, alcohol and drug abuse, and psychosocial stress. Health-promoting life-styles reduce not only the incidence of heart disease but also overall mortality *(4, 5)*. One risk factor may influence another, e.g., the risk of developing lung cancer in asbestos workers who smoke is greater than the sum of the risks of the independent exposures. Furthermore, a substance may be beneficial in one particular disease yet risk-producing at the same time, e.g., clofibrate prevents coronary heart disease but increases the incidence of hepatic disorders *(6)*. Risk factors and related conditions should not be considered in isolation. The integrated approach aims to avoid overlap, repetition and contradiction, and to ensure that one risk factor is not changed at the expense of increasing others.

Life-styles

The prevention of chronic diseases evidently requires, to a greater or lesser extent, a change in life-styles. The promotion of health and the reduction of overall population risk are common objectives.

Preventive actions have been characterized as: governmental and legislative; societal; and individual. Some focus on changing the overall risk to the population, others on identifying and treating high-risk individuals. However, such compartmentalization is artificial, as all of these measures interact *(7)*. A community-based programme marshals activities at all these levels in order to change public opinion and life-styles and to screen and treat people at high risk. This

Despite enormous advances in medical knowledge and technology, clinical developments have reduced the incidence of chronic diseases to only a modest degree.

appears the most promising vehicle for health promotion and long-term change in population risk. The North Karelia programme in Finland is an example of community-based action aimed at reducing the major risk factors for coronary heart disease *(8)*. The results have proved encouraging in this highly motivated community. Logistically it makes sense to encompass other chronic diseases in such programmes, including chronic bronchitis, various cancers, diabetes, high blood pressure, and dental caries. In this way the maximum benefit is gained from the expertise, communication channels, and community commitments that have been built up.

Specialization in clinical medicine has led to a tendency for the prevention of each noncommunicable disease to be considered in isolation.

It is highly desirable to have an organizational structure with a comprehensive system for data collection and evaluation so that the impact of a preventive programme can be monitored. Such a system allows new developments in chronic disease prevention to be introduced more quickly and makes it possible to avoid repeating mistakes.

Populations and individuals

Prevention may consist of reducing population risk or identifying and treating high-risk cases. The reduction of risk and the promotion of health in an entire population are more difficult to achieve than the identification and treatment of high-risk individuals. However, only a small proportion of a population is at high risk, and deaths from chronic disease are not confined to this category. The ultimate objective should be primary prevention through the reduction of population risk, but the finding of high-risk cases, and secondary and tertiary prevention, will all have their roles for the foreseeable future. Indeed, greater compliance with advice and treatment regimens may be achieved among high-risk individuals where the whole community accepts the need to aim for a healthier life-style. In this situation, high-risk individuals will not be singled out as different.

Health promotion

All potential outlets to the community, including schools, workplaces, shops, public buildings, clinics, hospitals, the mass media, and voluntary and community organizations, should be utilized to provide information, generate debate and increase awareness about the scope that ordinary people have for eliminating or reducing health risks. Illustrative material should be made available and there is a need for commitment and participation by influential members of communities.

Emphasis should be placed on the duty of families and the community to create an environment in which children can acquire health-giving habits. In large measure, children follow examples set by their relations, friends and teachers, and by popular figures such as pop stars and sports personalities.

Finally, the use of health services should be encouraged, e.g., screening or case-finding services, antenatal care, and well-baby clinics.

Primary health care

Primary health care should form the nucleus for locally organized programmes of disease prevention. No matter how primary health care is organized, it provides the first contact between the person and the health services and provides continuity of care.

Advice should be available to individuals on how to reduce risk factors for chronic diseases and there should be universally accessible screening and case-finding facilities. Specific services should include the following.

1. *Antenatal care.* The objective should be to provide antenatal care as early as possible during the first trimester of pregnancy. This contact should be used to promote a healthy life-style; for example, the danger of smoking, not only to the mother but also to her children, should be indicated, and advice should be given on parentcraft. Comprehensive screening should be offered for conditions harmful to the mother and her unborn child *(9)*, with the aims of ensuring that children are born healthy and reducing disability.

2. *Well-baby clinics and home visits.* Screening for congenital defects of vision, speech, hearing and development at key stages after birth is an important first step towards reducing later handicap and helping children to reach their full potential. How services are arranged to do this will vary between communities but there is likely to be a co-ordinated range of activities performed at home, in clinics and at school.

3. *Screening/case-finding for high-risk cases.* People usually visit a doctor at least once every five years. This occasion could be used to obtain information, through simple tests or questions, on blood pressure, obesity, cigarette smoking, serum cholesterol, alcohol intake, blood sugar, and, in women, cancer of the cervix and breast. With appropriate age/sex registers and record linkage, repeat screening at suitable intervals could be organized.

Community settings

A community-based programme requires the reorganization of primary health care to take on broader responsibility for prevention and to forge closer links with community leaders and the public in general. The development of an integrated programme for the prevention of noncommunicable diseases should be part of a process of modelling primary health care to meet the needs of the community, in cooperation with it. Reorganization should not be confined to the area of primary health care: more specialized hospital-based care should be modified so as to complement primary health care and community-based programmes for disease prevention.

All potential outlets should be utilized to provide information, generate debate and increase awareness about the scope that ordinary people have for eliminating or reducing health risks.

The value of a community-based programme is that it gives health professionals flexibility for identifying the best opportunities for health promotion activities. In general, however, the following sorts of setting and activity may be considered.

1. Schools should provide health education as part of the normal curriculum. There are many examples of this having been done on an experimental basis *(10)*. Further work is needed to identify the best methods, given

that different age groups will be responsive to different types of health message. Teachers should be persuaded to set a good example, and parents and popular, respected figures should be encouraged to involve themselves in health promotion in the school environment.

Schools also offer a setting for screening and the identification of high-risk children

Emphasis should be placed on the duty of families and the community to create an environment in which children can acquire health-giving habits.

through regular health checks, and also for counselling on stress, drug abuse, sex, psychological problems, and other matters.

2. Workplaces should be utilized for publicizing health issues. Managers, in cooperation with workers, should ensure that workplaces are pleasant and safe. If meals are provided on the premises they should be consistent with the attainment of a healthy, balanced diet. If sports facilities are available they should be publicized as part of a campaign to encourage the taking of exercise on a regular basis. If occupational health services are available they should contribute to health promotion and the giving of advice on the control of risk factors. Careful planning and organization are needed here to ensure that preventive services are not suspected of being used as a means of deciding on future employment.

3. By forging links with local traders and supermarket chains and gaining the cooperation of food manufacturers in the labelling of products and the provision of, for example, unsalted as well as salted tinned foods, it should be possible to reach a large part of the population. Information on diet and other health matters might be distributed through local stores.

4. Posters and leaflets should be placed in public buildings, including health care facilities, and on public transport. Public canteens should be encouraged to provide a varied, nutritionally sound menu. Smoking should be discouraged on public transport and in public buildings. Sports installations should provide a range of facilities to cater for different needs, including organized classes.

5. The mass media strongly influence people's ideas, opinions and life-styles. Contact should be made with newspapers, television and other media to encourage the discussion of health issues and to heighten the public's awareness and understanding of them. Advertising space should be used to present facts about preventable diseases and health promotion.

6. Community involvement should be reinforced by distributing leaflets to houses and organizing public meetings and events, e.g., fun runs. Sports clubs, youth clubs, women's institutes, and other bodies should be encouraged to take part.

Governmental and legislative measures

Governments should ensure that the necessary facilities are available. It is useless to advise regular exercise if sports facilities, organized classes, and so on are not available or accessible. Government support is also needed if food manufacturers are to be brought into campaigns aimed at modifying and diversifying what people eat. Finally, national and local governments are responsible for seat-belt legislation, road safety, pollution control, and other preventive measures. Dialogue between

government and community should be encouraged so as to improve cooperation and the awareness of responsibilities.

National strategy on prevention

While decentralization is a major component of the population-based approach to prevention, central government has an important role. In addition to providing political, logistical and financial support, establishing a national strategy is important. This provides a framework for the interchange of experiences so that the maximum benefit can be obtained from developments and experience. Such initiatives have been taken in recent years in Canada *(11)*, the United Kingdom *(12)*, and the USA *(13)*. On the international level, targets for the year 2000 have been set by the World Health Organization *(14-16)*. Governments should have objectives for the prevention of chronic disorders within the context of a strategy that can be adapted to different communities.

Evaluation and research

Evaluation is essential to justify a programme's continuation or provide for its modification and development; it is also necessary if the experiences in one area are to be applied elsewhere. It requires the collection of a minimum set of data. Some may be obtained routinely on a national basis, such as demographic and mortality data. Other information, e.g., that relating to risk factor levels, behaviour, and beliefs, has to be collected through *ad hoc* surveys. At the planning stage the types of data needed must be defined, and procedures for collecting the information must be included in the design of the programme. Where possible, national and local trends should be compared. However, it may also be desirable to compare local changes with trends in similar communities. To gain any indication of what is happening within a community, baseline data must be collected before initiating the programme.

Evaluation is an important research tool. There is strong etiological evidence to support the introduction of action to prevent noncommunicable diseases. However, there is still much to learn about how to change the incidence of diseases, reduce risk factor levels in communities, and promote healthy life-styles. Although the harmful effects of cigarette smoking are acknowledged, young people still take up the habit. Those women most at risk of cervical cancer and birth problems, i.e., the economically worst off, are still the least likely to take advantage of screening services.

We need to discover how to package preventive measures and health promotion in ways attractive to target populations. We need to determine how best to marshal all the settings and communication networks in communities and how to avoid conflict between the prevention of noncommunicable disease on the one hand and economic and social concerns on the other.

For many important noncommunicable diseases, however, there is a need for further basic research. In some cases, screening or preventive measures have not been devised, in others there is still insufficient knowledge of causative factors. This is true of various forms of cancer, certain congenital anomalies, neurological diseases, multiple sclerosis, and psychiatric disorders such as schizophrenia.

Training and manpower

The implementation of a community-based programme for the prevention of

noncommunicable diseases requires that new skills and knowledge be acquired by primary care workers, other health professionals, and community representatives.

Primary care workers should be trained to understand the health problems of their communities and the approaches to the early detection, prevention and treatment of disease. They should also acquire interpersonal and organizational skills so that they can motivate, encourage and respond to the communities. Moreover, primary health care workers should train community representatives, including elected leaders, teachers, and trade unionists, so that they can help to contact and motivate people.

The training of more specialized health professionals is important, since primary health care workers rely on them for guidance and training. Far from undermining the role of workers in acute care and hospital services, a community-based programme for the control of chronic disease gives them broader responsibilities.

* * *

The prevention of noncommunicable diseases is an international issue since they are of growing concern throughout the world. Much can be learnt by the exchange of ideas and experiences between countries and communities. The World Health Organization, particularly by supporting an integrated programme for the prevention and control of noncommunicable diseases, is making a major contribution to the development of strategies, the evaluation of their feasibility, and the promotion of the principle of prevention worldwide. However, it must be stressed that success will rest on political as well as societal commitment. □

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DIS-1.

coordinating the response to chemical emergencies.[2]

The meeting amply demonstrated that preparing professionals to manage emergencies caused by technological hazards is a great challenge, and one that faces health ministries which are already overburdened. Yet, the experiences of Bhopal and Mexico show that there is no time to lose, and several countries have already taken steps to meet the challenge. As a consequence of the symposium, a number of national seminars[3] have been organized in the Region for professionals from health and other sectors who will be called upon to cooperate in times of emergencies caused by chemical accidents.

[2] *Proceedings: Symposium on Chemical Emergency Preparedness*, 1984, 427 pp., issued by the Pan American Center for Human Ecology and Health. Available in English and Spanish from the Center, Apartado Postal 249, 50000 Toluca, Estado de México, Mexico.

[3] For further information on these seminars, write to the Pan American Center for Human Ecology and Health (for address, see footnote 2).

PAHO fellowships in the Caribbean not causing brain drain

Although there has been much speculation and anecdotal data about the migration of health professionals who have had an opportunity to study outside of their home countries, our survey shows that the great majority (82.6%) of the PAHO fellows . . . did return to their countries of origin. Moreover, an unspecified number of those found to be overseas at the time of the survey were still studying under a PAHO fellowship or had received another type of fellowship for additional study.

The survey findings thus strongly indicate that PAHO fellowships awarded in the Caribbean area have not encouraged or facilitated emigration. This low emigration rate can be partly attributed to the PAHO policy of awarding very few fellowships in medical specialties. It should also be noted that the high rate of return observed among former PAHO fellows in the Caribbean cannot necessarily be extrapolated to other fellowship programs or to other subregions of the Americas. Conceivably, this phenomenon could be characteristic only of the PAHO program or the Caribbean subregion. However, the finding that an overwhelming majority of the PAHO fellows surveyed did in fact return to their home countries and have been working in their fields of study should be regarded as very significant for future policy development in the Caribbean area.

From: LOCKETT, B. A. & TRUMAN, B. I. A retrospective study of the PAHO fellowships program in the Caribbean, 1970–1979. *Bulletin of the Pan American Health Organization*, **18** (3): 281–287 (1984).

WHO Chronicle, 39 (3): 98–103 (1985)

AIDS: where do we go from here?

Our understanding of the origins and course of the acquired immune deficiency syndrome, AIDS, changes almost as fast as the information can be communicated, and this year it was considered time for a thorough review of the knowledge gained to date. An International Conference on AIDS was therefore organized and held in Atlanta, Georgia, USA, in April. This article discusses some of the most important findings presented at the conference, and their implications for WHO.

Although evidence suggests that the acquired immune deficiency syndrome (AIDS) has been occurring in the USA and other parts of the world such as central Africa and the Caribbean since at least the 1970s, it was not until 1981, when the disease assumed epidemic proportions in the USA, that it was identified as a clinical entity and given the name AIDS.

The epidemic in the USA affected predominantly homosexual and bisexual males between the ages of 20 and 50, and manifested itself in a wide range of infections that frequently proved fatal to the AIDS patient because of his limited immune response and consequent inability to contain the infection. Of these "opportunistic infections" the two most commonly associated with AIDS were *Pneumocystis carinii* pneumonia, and a form of cancer, Kaposi's sarcoma.

However, though an epidemiological pattern could be detected early on, scientists were not able to establish the origin of the disease, which meant that for some time there was an unavoidably confused and piecemeal response from the medical profession. WHO recognized the need for coordination of the research and surveillance that was going on independently in many centres, and for making widely available the data that were being gathered. Much of the coordination work was taken on by the Regional Office for Europe in Copenhagen, which designated the Institut de Médecine et d'Epidémiologie africaine et tropicale of the Claude Bernard Hospital in Paris as a WHO Collaborating Centre for AIDS.

In October 1983 the Regional Office called a meeting to publish and disseminate all available information on the syndrome to date. A consultative meeting at WHO headquarters in Geneva, held the following month, concluded that, though the etiology of the disease remained a mystery, enough was now known about its characteristics to enable specific guidelines to be drawn up for prevention and control measures. These focused mainly on sexual behaviour, on the habits of drug abusers, and on the handling of blood and other body fluids, through which AIDS was believed to be transmitted.

Early 1984 saw a breakthrough, with two separate research groups announcing the isolation of the putative pathogen from patients suffering from AIDS. One virus, found by a team from the National Cancer Institute in the USA, was called human T-lymphotrophic retrovirus type III (HTLV-III) and the other, isolated by researchers from the Pasteur Institute in Paris, was given the name lymphadenopathy-associated virus (LAV).

Both are now considered to be variants of one and the same virus, which has since been firmly established as the etiological agent of AIDS. This virus is being called LAV/HTLV-III until the International Committee on the Taxonomy of Viruses approves a definitive name in accordance with the rules governing the nomenclature of viruses.

The Atlanta conference

From 15 to 17 April 1985 an International Conference on Acquired Immune Deficiency Syndrome (AIDS), sponsored jointly by the US Department of Health and Human Services and by WHO, was held in Atlanta, Georgia, USA. It aroused enor-

Intravenous drug abusers run an extremely high risk of infection with AIDS virus, LAV/HTLV-III. The sharing of unsterilized syringes is known to be a source of contamination.

mous interest, attracting more than 2000 participants from 50 countries and permitting a thorough review of all the information so far gathered on the etiology and characteristics of the disease and on its epidemiology, management, and control.

Etiology and characteristics

LAV/HTLV-III has been detected in blood, semen, and saliva. Though the average incubation period is 29 months in adults and 12 months in children, the virus can lie dormant in an individual for as long as five years before producing symptoms of AIDS or AIDS-related complex (ARC) —a milder form of AIDS in which opportunistic infections do not become life-threatening. However, not everyone found to be infected with the virus goes on to develop symptoms: studies show that within a period of two to five years of observation, 2–15% of infected individuals develop AIDS, 23–26% develop ARC, and 60–70% remain free of symptoms, though it is believed that symptom-free individuals can remain carriers of the virus indefinitely.

Clinically, AIDS often takes the form of opportunistic infections by pathogens that are common in the environment and not normally life-threatening but that prove fatal to at least 41% of AIDS patients because they have so little ability to fight disease. The clinical manifestations of AIDS fall into several common categories.

* Pulmonary problems. The most prevalent pulmonary infection in North America and Europe is *Pneumocystis carinii* pneumonia, though *Legionella pneumophila* and cytomegalovirus, also opportunistic infections, produce a similar pulmonary picture.
* Central nervous system disorders, which are found in approximately 30% of AIDS cases in North America and Europe. Patients present most frequently with cerebral lymphomas, vascular complications, and cryptococcus infections.
* Chronic diarrhoea, weight loss, and fever of unknown origin. These symptoms are most

common in central Africa and Haiti. In addition, severe thrush is also a common presenting symptom in Haiti.

* Malignancies, most frequently a particular form of Kaposi's sarcoma. Whereas traditionally Kaposi's sarcoma tends to affect the extremities, in AIDS patients it tends to be generalized, to involve lymph nodes, mucous membranes and viscera, and to be more aggressive.

Though children with AIDS show the same range of clinical manifestations as do adults, the most common clinical findings are chronic diarrhoea, anaemia, and thrush, and a consequent failure to thrive.

Epidemiology

Since AIDS was first recognized in 1981 nearly 11 000 cases have been reported, more than 80% of them in the USA. But the disease is also a major public health problem in Haiti, in central Africa, and in some parts of Europe—most notably in Belgium, Denmark, the Federal Republic of Germany, France, the Netherlands, Switzerland, and the United Kingdom. Cases have been reported from every continent, though relatively few from Asia and the Western Pacific countries other than Australia, where it is considered the most serious public health problem today.

In North America, Europe and Australia, homosexual men account for at least 70% of cases. The other groups for whom the risk of infection is high include intravenous drug abusers, persons with haemophilia, and recipients of blood transfusions. The heterosexual partners (including prostitutes) and children of high-risk individuals and of those already infected with AIDS virus are also especially vulnerable to infection, and the virus can be transmitted by a mother to her unborn child.

In Africa, where the picture is very different, it has been difficult to identify any group at particular risk. AIDS in Africa occurs almost as frequently among females as males. Neither homosexuality nor intravenous drug abuse, nor even haemophilia, is a particular characteristic of sufferers, though a multiplicity of sexual partners does appear to be one factor. In the Caribbean, a more or less similar picture is seen.

Comparative tests on AIDS patients have shown that the virus is very prone to change, and variations of up to 25% in its genetic structure have been recorded. Thus far, researchers have been unable to find any correlation between variations in the virus and the manifestations or outcome of disease. However, this is an intriguing and important line of inquiry since the genetic differences might help to explain the marked differences observed between the continents in the epidemiology of AIDS.

The realization that AIDS can be and has been passed on through infected blood products used widely by the medical profession in the normal course of events has caused great concern and led to demands that individuals from high-risk groups refrain from becoming blood donors. Where blood donation is voluntary and unpaid, there is greater chance of cooperation in this demand—a fact recognized by WHO, which recommended in 1975 that blood donation be totally voluntary everywhere. However, it is possible today to screen blood products for antibody to the virus, and to reduce the chances of infection of patients with

Several cases of AIDS were diagnosed in patients who showed none of the common risk factors, before it was understood that the syndrome could be transmitted via blood transfusion. The risk of contamination from this source can be virtually eliminated by screening out blood with antibody to LAV-HTLV-III and by the heat treatment of blood products.

haemophilia by applying heat treatment to the blood products they require, namely factors VIII and IX.

A further worry was that hepatitis B vaccine, which for the moment is prepared from blood plasma,[1] might be susceptible to contamination, especially since the plasma used frequently comes from a pool that may have been obtained from thousands of donors. But this fear has been dispelled by the discovery that certain steps in the preparation of the hepatitis B vaccine effectively inactivate the AIDS virus.

There have been case reports of donors of sperm for artificial insemination who have subsequently been identified as carriers of LAV/HTLV-III, and artificial insemination is now considered to be a possible source of infection, although the precise degree of risk has not been calculated. Serological testing of donors of sperm, organs, and other human material should be performed before these materials are used.

Careful surveillance in the USA and in Europe has shown that the number of cases doubled every six months up to 1983 and is now doubling every year. Studies also suggest that, as the observation period of positively identified cases lengthens, the mortality rate may well prove to be far in excess of the 41% generally quoted.

Education and information

There is no cure for AIDS at present, and though much is known about the structure and transmission of the virus, scientists are not optimistic about the chances of developing a vaccine in the near future. Care of patients at present therefore consists of symptomatic treatment for infection and cancer, and emotional support.

Under such circumstances the role of health education and information is crucially important in fighting the disease, as well as the fear it has aroused generally. Education is particularly important for the homosexual community. It is also indispensable for health care workers who are assigned to care for patients with a disease they do not fully understand and about which they may have an ambivalent attitude. Hostile attitudes are not uncommon in health personnel caring for patients with sexually transmitted diseases in general, and in the case of AIDS patients these may be compounded by intolerance of homosexuality.

Different educational methods have been adopted in different countries, but the central messages are the same.

- Sexual transmission is the commonest but not the only mode of transmission.
- AIDS in homosexual males is most commonly transmitted through anal intercourse. Though oral-anal contact, oral-genital contact, and open-mouth kissing have not been proved to be especially harmful, the risk of infection from such practices certainly cannot be ruled out since they can involve the exchange of semen or blood.
- The use of condoms decreases the risk since it limits exposure to semen, but this is seen as a precautionary measure only and not as full protection.
- Any activity that could involve the exchange of blood between people who might be infected with AIDS virus should be avoided, for example, sharing of toothbrushes, razors, and other personal equipment.
- Women suffering from AIDS or at high risk of infection should avoid becoming pregnant since a newborn baby is very vulnerable to infection from the mother, and the infection can even be transmitted *in utero*.

The group at next highest risk in the USA and Europe, after homosexual men, is that of intravenous drug abusers. According to some reports, these individuals are typically less receptive to health education than are other groups. Moreover, since drug abusers do not have their own advocacy organizations, they are difficult to reach as a target group. However, the main points to be put across to people in this category are that the sharing of needles and the use of equipment whose sterility is suspect are especially dangerous practices.

As for health personnel, the risks of laboratory-acquired infection need to be assessed more accurately, and it was therefore recommended at the Atlanta conference that a representative sample of laboratory workers be screened at the time of employment and regularly thereafter for the presence of LAV/HTLV-III antibody in their blood, the blood samples being collected and stored. To date only one case of transmission of AIDS to a health care worker in the course of duty has been reported, and the risk is considered small under conditions of good hygiene. However, in places where hygienic standards are low the risk is considerable, and the use of syringes, needles, and instruments without sterilization certainly plays a role in transmission of the disease in some countries.

Because of uncertainty about the risks involved there is a great deal of anxiety among health care workers, even where hygiene is good and worry needless. A WHO consultation held in November

[1] Alternative sources of antigen are now being explored, as reported in *WHO Chronicle*, **38** (6): 260–261 (1984).

The oldest profession in the world. Prostitutes are now considered to be particularly vulnerable to infection with AIDS virus. AIDS victims in Africa are predominantly heterosexual, and the incidence in the heterosexual population is increasing in North America, Europe and Australia, where heterosexual promiscuity is thought to provide a "bridge" of transmission between high-risk groups and the general population.

1983 reviewed and accepted the guidelines developed by the Centers for Disease Control (CDC) in Atlanta, Georgia, USA, for the precautions to be taken with AIDS patients and materials.[2] Some of these guidelines are summarized in the box overleaf. Basically, it is recommended that health workers observe the same level of precautions when caring for AIDS patients and handling their body fluids as they would with hepatitis B patients, since the mode of transmission and case distribution of the two diseases are similar. For work that involves the production and purification of LAV/HTLV-III, however, even more stringent biosafety standards are necessary.

The range of education and information activities reported to the Atlanta conference was very wide, and included training courses for health care workers, counselling for AIDS sufferers and their families, presentation of health education materials for the public and the media, and special cooperation between local homosexual organizations and health authorities. Task forces and special committees have been set up, and there has even been commercial promotion of the "Safe Sex" message in the USA through such products as T-shirts, handkerchiefs, and calendars. Much research has also been done on attitudes to the disease among special risk groups, and the most appropriate ways of putting across the health education messages. The task can be a delicate one: in some cases homosexuals have seen the focus of attention on their sexual practices as a sign of intolerance of their life-style. Clearly, confidentiality of information about the results of blood tests and the identity of AIDS patients is very important. Moreover, informed consent should be obtained before serological testing is undertaken.

A point stressed at the conference was that health education and information designed to protect those at risk should not overshadow the need to help those already suffering from AIDS and AIDS-related complex. These people should be helped to understand how to avoid infecting others and how to protect themselves from opportunistic infections, as well as how to cope with the psychological effects and possible social rejection. Not surprisingly, depression is a common characteristic of people suffering from this often fatal disease.

WHO reviews the conference

Immediately following the Atlanta conference, a WHO consultation was convened to review the information presented and to propose further action by the Organization and its Member countries.[3] The group recommended that WHO should:

[2] A memorandum from the meeting, published in *Bulletin of the World Health Organization*, **62** (3): 419–432 (1984), includes the full set of CDC guidelines for clinical staff, laboratory workers, dental care personnel, persons performing necropsies or providing mortician services, and persons handling experimental animals inoculated with material from patients.

[3] The full report of this consultation will be published in the *Bulletin of the World Health Organization*, **63**, (4): 667–672 (1985); a summary of the conclusions and recommendations appeared in *Weekly epidemiological record*, **60** (17): 129–130 (1985). The consultation was followed by a brief meeting convened by the Regional Office for Europe, at which regional guidelines were drafted.

Precautions recommended for health workers

1. Great care must be taken to avoid accidental wounds from contaminated sharp instruments, and to avoid contact of open skin lesions with material from AIDS patients.
2. Gloves and gowns should be worn when handling or exposed to soiling by blood specimens, body fluids, excretions, and secretions, as well as objects contaminated by them.
3. Hands should be washed before leaving the room of known or suspected AIDS patients. Hands should also be washed thoroughly and immediately if they become contaminated with blood.
4. Specimens should be labelled prominently with warnings, such as "Blood Precautions" or "AIDS Precautions". If the outside of the specimen container is visibly contaminated with blood, it should be cleaned with a disinfectant (such as a 1 : 10 dilution of 5.25% sodium hypochlorite household bleach with water). All blood specimens should be placed in a second container, such as an impervious bag, for transport. The container or bag should be examined carefully for leaks or cracks.
5. Blood spills should be cleaned up promptly with a disinfectant solution, such as sodium hypochlorite (see above).
6. Soiled articles should be placed in an impervious bag prominently labelled "Blood Precautions" or "AIDS Precautions" before being sent for reprocessing or disposal. Alternatively, such contaminated items may be placed in plastic bags of a particular colour designated solely for disposal of infectious wastes by the hospital. Disposable items should be incinerated or disposed of in accordance with the hospital's policies for disposal of infectious wastes. Reusable items should be reprocessed in accordance with hospital policies for hepatitis B virus contaminated items. Lensed instruments should be sterilized after use on AIDS patients.
7. Needles should not be bent after use, but should be placed promptly in a puncture-resistant container used solely for such disposal. Needles should not be reinserted into their original sheaths before being discarded into the container, since this is a common cause of needle injury.
8. Disposable syringes and needles are safest. Only needle-locking syringes or one-piece needle-syringe units should be used to aspirate fluids from AIDS patients, so that collected fluid can be safely discharged through the needle, if desired. If reusable syringes are used, they should be decontaminated before reprocessing.
9. AIDS patients who are too ill to use good hygiene, or who have altered behaviour secondary to central nervous system infection, should be given a private room.
10. Dental care personnel should wear gloves, masks and protective eyewear when performing dental or oral surgical work on AIDS patients.
11. Instruments used in the mouths of AIDS tients should be sterilized after use.

—establish a network of collaborating centres to be responsible for staff training and specialist advice as well as for epidemiological research;

—encourage and coordinate global surveillance of AIDS using uniform reporting methods, and disseminate information as widely and speedily as possible;

—assist in the development of a vaccine;

—take an active part in the health education of the public, as well as that of health care workers;

—assess the risk posed by AIDS country by country.

The subject of AIDS received a further airing at the Thirty-eighth World Health Assembly held in Geneva in May, at which delegates expressed the view that in spite of all that has been discovered about the syndrome, the world has seen only a small part of the problem. As the United States delegate pointed out, it seems ironic that barely eight years since we celebrated the end of smallpox, another deadly virus has appeared on the scene to frighten and perplex us all.

WHO Chronicle, 39 (3): 104–106 (1985)

Tobacco—its role in the economy and the health of African countries

Smoking is on the increase in many developing countries, and positive steps are needed by governments and health authorities to counter this threat to health. Africa, where tobacco is an important cash crop, faces its own distinctive problems in curbing the trend, and in the kind of health hazards it poses. These special problems were highlighted recently when English-speaking countries of the WHO African Region met in Lusaka to discuss the issue and decide on measures that should be taken.

The issue of smoking and health in Africa is complicated by the fact that in many countries tobacco is grown commercially and relied upon to bring in foreign exchange through export, or revenue for the government if sold on the home market. Thus, in some countries, the ministries of health and of agriculture are working at cross purposes.

This contradiction is recognized in the report issued recently of a WHO seminar on smoking and health organized for English-speaking Member States of the WHO African Region, and held in Zambia. The Prime Minister of Zambia, Mr N. Mundia, opening the seminar, stated that governments had an obligation to educate people on the risks involved in the use of tobacco, but he acknowledged too that this could pose a moral dilemma where tobacco production made an apparently significant contribution to the economy. He also warned that developing countries are considered valuable markets by tobacco companies, and stressed that if the promotion of tobacco products by such companies represented a threat "to the health of our people, we cannot let it happen".

The point was endorsed by Mr W. C. Mwambazi, the National WHO Programme Coordinator in the host country, who stated that smoking was on the increase in many developing countries as a result of unscrupulous marketing practices by cigarette manufacturers, and that smoking was a major threat to the achievement of health for all by the year 2000.

Health hazards: an African perspective

Aspects of smoking and health that have special relevance for Africa are emphasized in the report. Though there is a dearth of reliable information, the few studies carried out in Africa tend to confirm findings from the developed world that smoking increases the risk of cancer and coronary heart disease.

Most accounts of the health hazards of smoking describe the consequences of smoking the commercial products of the big tobacco companies, but this is only one aspect of the problem. The use of non-commercially produced tobacco is widespread. Not only is tobacco smoked in Africa, but it is chewed and taken as snuff, and these uses also entail a risk to health. Elsewhere, for instance in India, smoking of home-made cigarettes and tobacco chewing have been found to promote faster blood clotting, while the stillbirth rate for female tobacco chewers was found to be 50 per 1000 births, as against 17.1 per 1000 for non-chewers.

There are at least 65 varieties of *Nicotiana tabacum*, many of them grown in Africa, and they are not all equally harmful to health. Besides their intrinsic genetic differences, the methods of cultivating, curing and flavouring the tobacco have implica-

This article is based on *Smoking and health issues in selected English-speaking African countries. Report of a HQ/AFRO Regional Seminar on Smoking and Health, Lusaka, 26–28 June 1984* (unpublished document WHO/SMO/84.5).

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Management of Kwashiorkor in its Milieu—A Follow-up for Fifteen Months

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All under-fives in 20 villages were examined and the 32 suffering from kwashiorkor were studied in detail and followed-up for 15 months. The per capita income of these 32 families was Rs. 0·78 per day. These children on the average, were taking 79·1 calories and 2·3 g. of proteins per kg. actual weight per day before they were submitted to the domiciliary nutritional rehabilitation. Their mothers were given nutrition education and the children were provided with nutrition supplements from locally available foods. It took an average of 8·1 weeks for the oedema to subside. There were no deaths, but two children had recurrence. Details on clinical progress, dietetic intake, operational methodology of nutrition rehabilitation, impact on the nutrition awareness of mothers and cost/benefit have been discussed.

Introduction

For the last 35 years the clinical, pathological and biochemical aspects of kwashiorkor have been studied extensively, but mainly in hospitals. In India, the diet of children suffering from kwashiorkor has been found to be deficient, mainly in calories, due to small intake of food (Rao and Ramnathan, 1953; Rao *et al.*, 1961; Venkatachalam and Gopalan, 1960; Shah *et al.*, 1971). In African children, protein deficiency has been reported to be operative in producing kwashiorkor (Waterlow, 1948; Trowell *et al.*, 1954). The amount of protein advocated for the treatment of kwashiorkor has varied from 1 g./kg./day (Waterlow and Wills, 1960) to 6 g./kg./day (Dean, 1955). In India, satisfactory results were reported by Srikantia (1969) with 4 g./kg./day of protein and 200 cal./kg./day. With a mixture of Bengal gram (*Cicer arietinum*) and groundnut (*Arachis hypogea*) the children suffering from kwashiorkor recovered; however, the recovery was slow (Achar, 1963).

In a hospital, mortality due to kwashiorkor varied from 9 per cent to 50 per cent (Lawless *et al.*, 1966; Cook, 1971). Ramnathan *et al.* (1955) reported a hospital mortality rate of 20 per cent and a relapse rate of 30 per cent after discharge. It was observed that children returned to their previous diets as their mothers were not able to carry out the advice of doctors for economic and socio-cultural reasons (Suckling and Campbell, 1957; Shah, 1973).

For a hospital stay of three weeks, the cost of treatment of a case of Kwashiorkor to the Government was reported to be Rs. 367 while the cost to the parents in a free hospital in Bombay by way of loss of wages, travelling expenses and their own maintenance was Rs. 136.50 (Shah *et al.*, 1971; Shah, 1973).

From studies carried out in a nutrition rehabilitation centre or mother-craft centre in Haiti, it was reported that after eight weeks none of the children suffering from kwashiorkor had oedema and the mean gain in weight was from 68·9 per cent to 73 per cent of the reference weight and the cost of treatment for four months was about £7·16 (King *et al.*, 1968). About 69·5 per cent of the children treated in a residential type of nutrition rehabilitation centre in south India were found on follow-up to be improving, 5·8 per cent did poorly and 8·8 per cent died (Cutting and Cutting, 1972). With the domiciliary and clinic type of management at a rural health unit near Bombay, it took 5·2 weeks for the oedema to subside (Shah *et al.*, 1971). The cost to the Government (sponsors) was Rs. 9.90 per child and to the parents was Rs. 26. With this type of management, the diet of other younger children in the family and in neighbourhood changed for the better (Shah, 1973).

While tackling the problems of malnutrition, factors such as aetio-ecology and the constraints of money, materials and man power which may vary from country to country, and within such a vast country as India, should be considered carefully so that we may adopt integrated health and nutrition delivery systems most suited to our conditions.

Material and Methods

The present 15-months longitudinal study was undertaken at the WHO-aided project on the "Domiciliary Treatment of Protein-Calorie Malnutrition", Rural Health Unit, Palghar of the Grant Medical College, Bombay, to observe the effect of the domiciliary-cum-clinic type of management of severe forms of malnutrition on all 32 under-fives suffering from kwashiorkor out of the total of 1799 in these villages.

The area covered by two nurse midwives (NMs) of the Rural Health Unit, encompassing 20 villages, was taken up for the project. A domiciliary-cum-clinic type of Health care was created, using the infra-structure of the Primary Health Centre. The NMs assisted by five part-time social workers (PTSWs) formed the core of the service while the doctor's job was that of a manager (Shah *et al.*, 1974*a*). PTSWs made home visits and weighed the children regularly at monthly intervals or less either at home or clinics. The weighing scales were checked periodically against sand bags of known weights and also against a standard scale. The weights were recorded on a mother-retained weight chart and a duplicate weight chart was retained by the PTSWs. The PTSW established the grade of malnutrition with the help of a plastic overlay stencil. The weights and their recordings were checked periodically by the Nurse, Doctor or the Research Assistant. The PTSW gave health and nutrition education to mothers and held monthly nutrition demonstrations. Cases of protein-calorie malnutrition

with growth failure, oedema and apathy, but without any obvious cardiac, renal or hepatic cause to account for it, were diagnosed as kwashiorkor. The child having a height less than the third percentile for the age (Stuart and Stevenson, 1964) and associated weight loss or low weight (third percentile or less) was considered as having growth failure. All the children in this study had their heights and weights much below the third percentile for the age.

A record of the history of present symptoms, past illnesses, diet and gross milestones of growth and development was made. The diet consumed by the child on the previous day was considered for analysis, using the questionnaire method. If the child had an acute illness on the previous day, the diet of the child before the acute illness was ascertained. A complete physical examination of these children was carried out. Haemoglobin estimation was done using Sahli's method. The stool of the child was examined and a tuberculin test was done. Blood was also collected in heparinised micro-tubes for estimation of serum proteins. The nature of the illness was explained to the parents. The importance of frequency of feeding and of a high calorie and protein diet, obtainable from local sources, was stressed. The nutritional status of other children in the family was noted.

Nutrition supplements, which were started after the initial diet survey, were given to all these children up to the time their weights reached 65 per cent of the reference weight levels and the oedema disappeared. In 16 villages, 35 g. of roasted ground-nuts and 40 g. of roasted Bengal gram were supplied, giving about 17 g. of protein and 335 calories per day. In a coastal village, 150 g. of fried fish (Bombay duck), corresponding to 60 g. of edible portion, was given daily. In those villages, during the monsoon, 30 g. of dry Mangalore fish (100 per cent edible) was given. The cost of these supplements was Rs. 0.25 per day per child. In three villages 100 g. of *Sukhada* consisting of 60 g. of corn, soya and skimmed milk powder (CSM), 30 g. of jaggery and 10 g. of salad oil giving 12·1 g. of proteins and 430 calories was supplied.

Follow-up : The PTSWs followed up the kwashiorkor children every week, weighed them and gave nutrition education to the mothers. The investigators visited the children every month or more frequently, if necessary, and recorded the weight, height and diet, and made a clinical examination of each child. Medicines were prescribed whenever necessary and were dispensed by the nurse. Advice was given on the need to provide a more nutritious diet for the child from items available locally during particular seasons which the parents could afford, *viz* : boiled and salted potato, sweet potato, banana for an evening snack and *pithale* prepared from Bengal gram flour, *usal* a preparation made of sprouted pulses (legumes) for meals. Increasing the frequency of feeding to four times a day was stressed. The time taken for oedema to subside was noted and diet analysis and estimation of serum proteins were repeated at this stage.

The cost of the treatment to the parents by way of medicines, travel and loss of wages and also the cost to the sponsors of the project for medicines and staff were recorded. At the end of the study, the impact of the management was assessed, including the mother's knowledge about the illness.

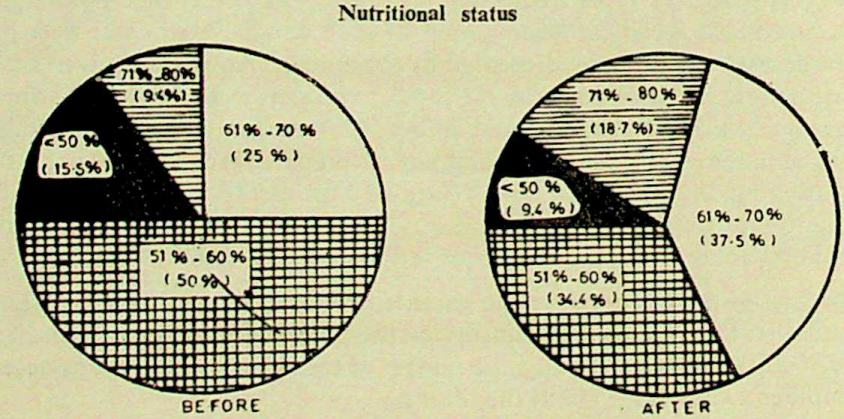
Results

Initially, about 65·6 per cent of the children suffering from kwashiorkor weighed less than 60 per cent of the reference weight (Stuart and Stevenson, 1964) and were having marasmic kwashiorkor and 9·4 per cent weighed between 71-80 per cent. The remaining children weighed 61 to 70 per cent of the reference. After 15-months' follow-up all the children gained weight. However, 43·8 per cent weighed less than 69 per cent and 18·7 per cent weighed between 71-80 per cent of the reference weight (Graph 1). Graph 2*a* and *b* depict the dietetic intake of the children in various age groups before and on subsidence of oedema. They show that while calorie intake was less than the requirement even at the end of the study, protein intake was more than adequate in most cases. The intake of calories of children belonging to the one to two and three to four years age groups was lower than that of children of other age groups.

About 78 per cent of the children had helminthiasis, 50·2 per cent had signs of Vitamin A deficiency and 43·7 per cent had infections of some kind. Out of the one-fourth of children who were passing blood in stools, 37·5 per cent had trichuriasis. Two children (7·4 per cent) had tuberculosis. All the children were anaemic and mean haemoglobin was 8·3 g. per cent. In 21·8 per cent of the cases, serum proteins were repeated on subsidence of oedema. The mean values of total proteins initially and after the subsidence of oedema were 5·1 g. per cent and 3·4 g. per cent and of serum albumin were 2·6 g. per cent and 2·9 g. per cent respectively, the changes in valves were not statistically significant.

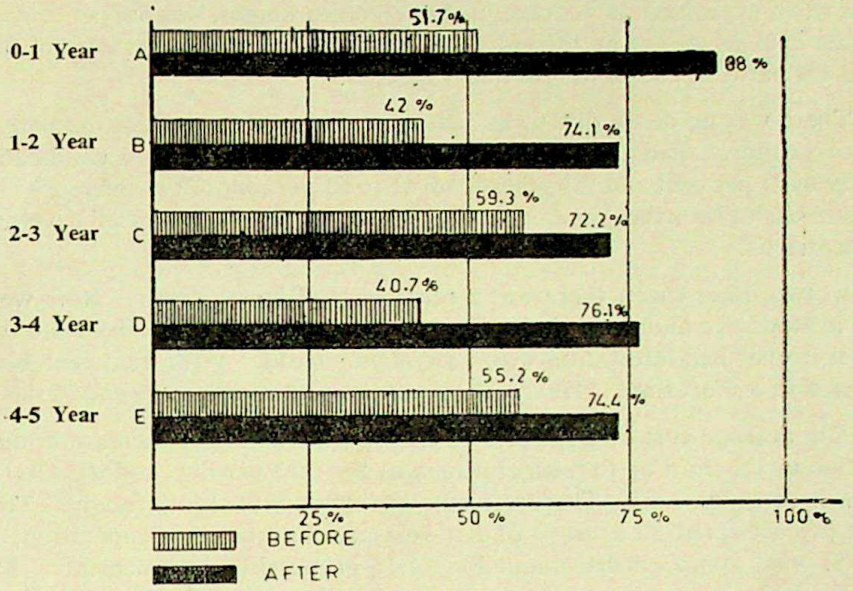
Graph 1

Nutritional status of the cases before and after rehabilitation.



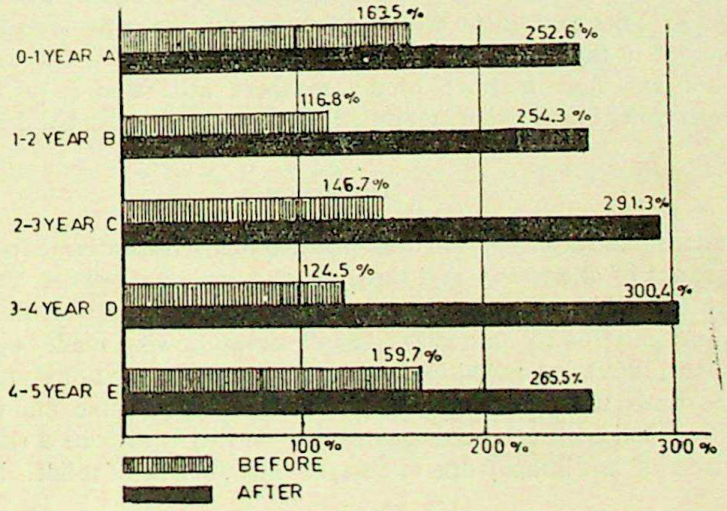
Graph 2a

Calorie intake as per cent of the requirement before and after the rehabilitation.



Graph 2b

Protein intake as per cent of the requirement before and after the rehabilitation.



It took an average of 8·1 weeks for the oedema to subside. None of the children died on account of kwashiorkor. At the end of the follow-up, it was observed that 75 per cent of the children had improved their nutritional status, their mean gain in weight when expressed as per cent of the reference weight, was 6·3 per cent above the expected gain in weight at the end of 15 months. About 25 per cent of the children gained 6·4 per cent less in weight than expected.

There was no death due to kwashiorkor during the recuperation period. However, two children died about 10 months later. The weight of one child had improved from 59 to 71 per cent and the other from 61 to 63 per cent of the reference. One died of post-measles broncho-pneumonia; the other within 48 hours of developing acute gastroenteritis.

In two other cases, there was a recurrence of kwashiorkor. Both were passing blood in stools for about a month before the recurrence. One of them had trichuriasis, while the other had infestation with *Fasciolopsis buski*. With treatment, both of them recovered in a short time. Hence, there was no recurrence at the end of the study.

The average cost of treatment to the family due to the increase in dietetic consumption by the child up to recuperation was Rs. 0·13 per day, and the total cost of recuperation was Rs. 7.40. There was no other expenditure to the family. The sponsors of the project spent an average of Rs. 16/- per child up to recuperation, of which Rs. 1·85 were spent on drugs and Rs. 14·15 on nutrition supplements. During this time, Rs. 570/- were spent on the remuneration of the five PTSWs who looked after the health and nutrition care of 1799 under-fives in the 20 villages. The staff structure of the Health Unit was otherwise unaltered and their salary is not included in these calculations.

At the end of the study, when asked about the cause of kwashiorkor, 62·9 per cent of the children's mothers attributed it to infection, infestation or anorexia, and only 11·1 per cent to a deficient diet. About 22·2 per cent could not say what caused the illness. About 48·2 per cent of the mothers thought that an improved diet coupled with medicines helped in the recovery of their children, 22·2 per cent atrributed recovery to diet alone and an equal number of the mothers attributed it to medicines only. About 29·6 per cent of the mothers modified the diet of their other children.

Discussion

The nutritional status and the diets of the under-fives suffering from kwashiorkor in the age groups of one to two and three to four years were poorer than that of the other age groups. The children whose improvement was poor were initially in the age group of nine months to one year, an observation also made by Gopalan and Venkatachalam (1969). A possible reason for the poorer nutritional status and dietetic intake of one to two year olds could be that during this period the infants did not take semi-solids and solids in adequate quantities. It might also be a reflection, in part, of malnutrition of late infancy due to delayed introduction of solids. Moreover, these

children were dependent for their food on their young brothers and sisters serving as mother substitutes while their mothers went out for work. In this age group diarrhoea was also an additional factor. Only 55 per cent of the two to three years age group and about 70 per cent of the children belonging to age group of three to four years had younger siblings in their families. It is possible, therefore, that the cause of the poorer nutritional status of some children in the three to four years age group, compared to those in the two to three years age group was the birth of another child. Early introduction of semi-solids during the second trimester of infancy is of utmost importance in the prevention of severe malnutrition. Spacing between children has an equally important role.

In view of the high prevalence of ascariasis among these children, periodic deworming of severely malnourished children living in areas of moderate-to-severe ascarial infestation may give rewarding results. Similarly, severely malnourished children may be given iron and vitamin supplements periodically.

The year of study was a year of scarcity in Maharashtra State. People were jobless for long periods; the food supply was not continuous and although the prices of all items had gone up, the wages of labourers in rural areas to whom these children belonged did not increase. The rate of improvement, and the overall improvement in nutritional status of all the children in these 20 villages was slow. The poorer weight gain than expected was apparently on account of inadequate calorie intake which could have been improved upon by further increasing the frequency of feeds at home and providing more calories in the nutrition supplements. Cereals or jaggery provide more calories as compared to proteins but those are to be eaten in a big quantity. Oil seeds, particularly ground-nuts, could be added in more quantity in the nutrition supplements to increase the calorie intake. By increasing calories, the gain in weight would have been better.

For long-term solutions to the problem of malnutrition, job security for parents is essential. Establishment of a minimum wage, implementation of food-for-work schemes or well organised cottage industries during jobless periods of the parents even in the most remote villages are vital steps that may be effective.

In the present study, there was no death due to kwashiorkor. Mortality of domiciliary and other non-hospital lines of management of kwashiorkor is considerably low. In a small series of 16 children reported by Shah *et al.* (1971), where the kwashiorkor cases were managed at home, not a single child died.

The low recurrence rate was probably due to the continuation of the modified diet of the children by their mothers even after subsidence of oedema. Regular weighing and mother-retained weight charts further helped in early detection of deteriorating nutritional status and subsequent modifications of the diets. Moreover, the domiciliary management was economically and socio-culturally acceptable to the families.

In this study, the parents did not have to pay for treatment nor to lose their daily wages. Their only expenditure was on the modified diet of the child. In other words,

the amount spent by the parents on treatment of one kwashiorkor case in a hospital with a 20 per cent chance of the child dying and a 30 per cent chance of a recurrence after the discharge (Ramnathan *et al.*, 1965) could be 12 times the cost of home management.

What the majority of mothers referred to as the cause of the disease was actually the illness which caused a sudden deterioration in the health of the child and precipitated oedema. Longstanding, inadequate, infrequent diet was not given as a major cause of illness by mothers. However, when asked about recuperation, 70 per cent of the mothers gave diet as one of its causes and 22·2 per cent gave diet as the only cause of recovery, indicating that although deficient intake of food was not given as a major cause of the illness by mothers, most of whom were illiterate, the role of diet in the genesis of the illness was understood well by a majority of them.

In the residential type of nutrition rehabilitation treatment, the stay of mothers at centres was an inhibitory factor in the management because it caused a loss of wages and great inconvenience to family life. In nutrition clinics, or centres of the non-residential type, it was observed that often the benefit of the services was derived only by those who resided near the clinics or centres, that attendance was irregular and there was a high drop out rate. Considering these facts, the domiciliary out-reach for nutritional rehabilitation proved effective. This approach was economical and did provide long-term beneficial impact (Shah *et al.*, 1974*b*). The traditional approaches to the problems of malnutrition need to be modified. Every community member needs to be covered, vulnerable children identified and beneficiaries selected for nutritional supplements on the basis of medically established criteria with the help of village assistants from the local community (Shah *et al.*, 1974).

Acknowledgment

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CYSTITIS

How you can help control it

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CYSTITIS

How you can help control it

Understanding cystitis— cornerstone to control

Cystitis, or infection of the urinary bladder, is one of the most common afflictions occurring in adult women. In fact, surveys indicate that 2 to 6% of adult women may have a urinary infection at any time. And estimates are that *one* out of every *four* women will probably have a urinary infection at least once during her lifetime. This

being so, can you protect yourself against cystitis? Are there rules that you can follow to minimize the danger of urinary tract infection? . . . to help in its treatment? . . . to prevent its recurrence?

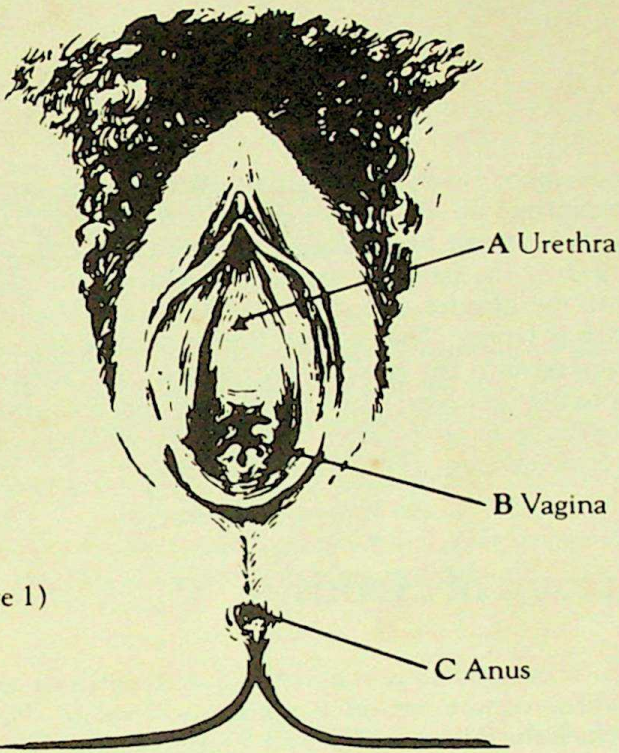
The answer to all of these questions for the majority of women is YES. If you understand the reasons why cystitis occurs, and the factors that foster its development, you can, with the help of your physician, counteract and control these factors.

This little booklet will try to give you the information you need to know to better understand and thus help prevent or control this troublesome, discomforting, and potentially dangerous bladder infection.

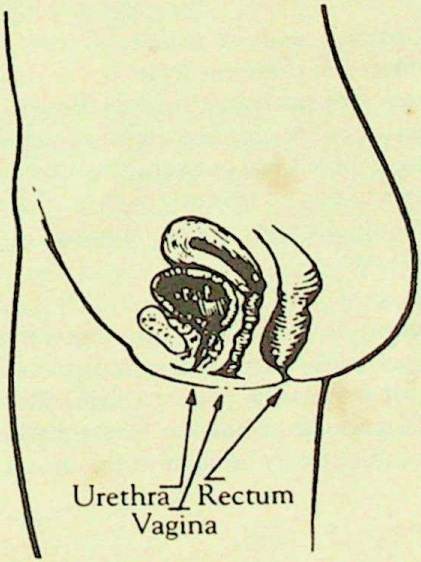
The role of anatomy

Although there are many reasons for the widespread occurrence of urinary tract infection in women, the most important is probably anatomy—that is, the nearness to each other of the female excretory and genital organs—the urethra (A) and vagina (B)—and excretory organ, the anus (C) as shown in Figures 1 and 2.

The urine should always be bacteria-free. However, bacteria are always present in the gastrointestinal tract and in the bowel movement. Sometimes even with the most careful hygienic practices some of these bacteria in the bowel



(Figure 1)



(Figure 2)

movement reach the vaginal area. This is because the vagina and the anus are so close to each other. Once bacteria get into the vaginal area, their entry into the bladder is aided by the fact that the female urethra, the passageway into the bladder, is straight and very short—only about 1 inch in length. Thus it offers little resistance to the entry of bacteria into the bladder. In contrast, it is interesting to note that in males, where cystitis is comparatively rare, the urethra is about 9 inches long, and its opening is at a considerable distance from the anal opening.

Symptoms of cystitis

When bacteria do gain a foothold in the bladder and cause cystitis, certain typical symptoms will result. These will include the following:

- a constant desire to urinate even within seconds or minutes after having done so
- a feeling of straining towards the end of urinating
- a feeling of incomplete emptying of the bladder
- considerable pain or burning on urination
- a need to get up several times at night to urinate
- a feeling of a poor urinary stream

Not every patient will have all of these symptoms, nor will the symptoms in each patient exist to the same degree or intensity. It is possible, however, for all of these symptoms to exist, and the condition may progress to the point where blood can actually be seen in the urine.

Counteracting factors that contribute to cystitis

Because of the close anatomical relationship of the anus, vagina, and urethra, 90% of all urinary tract infections in women are caused by bacteria that come from their own gastrointestinal tract. What can you do to prevent these bacteria from reaching the vaginal area in the first place? And, if bacteria enter the vaginal area, are there steps you can take to prevent the bacteria from entering the urethra and infecting the bladder, causing cystitis?

Instituting a good program of personal hygiene

Any successful long-term program of management for the prevention of urinary tract infections has to be built upon the foundation of a good program of local hygiene. Probably the most important factor in promoting bacterial invasion of the urinary tract is improper hygiene following urination and bowel movements. Without realizing it, many women will tend to wipe from back to front–from the anus towards the vagina–thus moving bacteria-laden stool directly into the vaginal area. The correct way to wipe, then, is from front to back–from the vagina towards the anus. This way you eliminate a major contributing factor to cystitis.

Another method often suggested as an aid to decrease the number of bacteria available for contaminating the urinary tract is to take showers instead of baths. While some medical authorities feel that the chances of contamination are lower when you shower, many other physicians feel that taking tub baths is fairly safe and will not result in urinary tract contamination.

A possible contributing factor can be certain types of wearing apparel, such as bodysuits, pantyhose, or tight slacks worn for long periods of time. They promote a warm, moist, dark environment, thus increasing the opportunity for bacterial growth. Prolonged bicycling, horseback riding, motorcycling and similar activities may also aggravate bladder infection but will not cause cystitis by themselves. The infection must first be there. The irritation caused by these activities acts to promote the infection.

Sitting in a wet bathing suit, on the other hand, does not lead to cystitis since the physical activity involved in swimming allows air and cold water to reach the urogenital area, thus avoiding a still, warm, dark, moist environment for long time periods.

Drink plenty of liquids

The bladder has an excellent defense mechanism to rid itself of the bacteria that might enter. It is the washout of bacteria that occurs with each urination. The amount of liquids you drink determines the amount of urine that is formed. Therefore, the more you drink, the more rapidly urine is formed, and the more rapidly any bacteria that may

have been introduced into the bladder are diluted by the sterile urine coming down from the kidneys. So when you drink water, fruit juices or other liquids frequently during the day you are helping your bladder exercise its own defense system against cystitis.

Don't hold back—urinate frequently

Generally, most women tend to urinate less frequently than is optimal. Often they will wait to finish what they are doing before going to the bathroom, delaying urination sometimes for an hour or more. This practice tends to increase chances for infection, because delaying urination gives the bacteria more time to multiply and take root. When bacteria are present in the urinary tract, the more frequently you void, the more rapidly the bacteria will be removed, thus reducing the chances for infection. In fact, it has been shown experimentally that even when pure cultures of bacteria are introduced into the bladder, they can often be eliminated by drinking enough fluids and urinating more frequently. A reasonable schedule for urination for most women would be to urinate at least once every 3 to 4 hours. This not only allows for a normal social existence but greatly decreases the chances of getting cystitis.

Sexual intercourse—another factor in cystitis

For some women, the act of sexual intercourse can cause cystitis. Through the act of intercourse, bacteria that may

be present in the vaginal area can be introduced into the urethra because of the close anatomical relationship of the urethra and vagina. The motion associated with intercourse may also introduce bacteria into the urethra. Nevertheless, cystitis and urinary tract infection do not follow every episode of sexual activity. Why some women are especially prone to cystitis due to sexual intercourse is not completely understood. Cystitis caused as a result of sexual activity is often referred to as "honeymoon cystitis" since its most frequent occurrence, and often its first occurrence, is among young women who have only recently become sexually active.

However, there are several actions which most women can take to decrease any bacterial contamination secondary to sexual activity. One of the most important is to urinate after intercourse. Bacteria which may have been introduced into the urethra will be promptly washed out before they have a chance to multiply and invade the bladder. Other factors which may help prevent "honeymoon cystitis" are showering before sexual activity, good lubrication, and the avoidance of positions that are painful as well as circumstances that increase the likelihood of contamination of the urinary tract with bacteria such as anal stimulation and anal intercourse when they are followed by vaginal intercourse.

Treatment of cystitis

Despite measures taken to prevent urinary tract infections, when enough contributing factors are present, bacteria do enter the urinary tract and can cause infection. The task of the physician then is to eradicate the bacteria as rapidly as possible and allow the urinary tract to return to its normal bacteria-free state.

Identifying the bacterial "germ"

The vast majority of urinary infections are caused by only a few bacterial types. These include organisms with names such as *E. coli*, *Proteus*, enterococcus, and *Klebsiella*. Because of the small number of organisms which cause urinary tract infections, the physician can usually choose a suitable antibacterial agent without identifying the bacteria. However, if the symptoms fail to subside, then it is necessary to grow the bacteria, identify the type, and test it against a variety of antibacterials to see which ones actually kill the particular infecting "germ." Physicians call this a urine culture and sensitivity test.

Collecting a sterile urine sample

The urine sample on which such a test is performed is called a "clean catch midstream" urine sample. To obtain this sample, you should cleanse the vaginal area by washing with soap and water, towelettes or any appropriate cleansing agent. Start to urinate and collect the sample carefully during the middle of the stream, after you have already passed some urine. The urine, thus collected, is then placed in an incubator where the bacteria are grown. The bacteria and the antibacterials which kill them are identified. This then serves as a guide to therapy, and those few infections which did not respond initially will usually be cleared up by the specific antibacterial agent chosen as a result of the sensitivity test.

At the same time, the doctor will usually tell you to drink a lot of fluids since this will dilute the bacteria with

fresh uninfected urine from the kidneys. So not only can drinking lots of fluids help prevent urinary tract infections, but it may also be an important part of therapy when a urinary tract infection does occur.

When cystitis persists

In some cases, symptoms in women treated with a specific antibacterial agent chosen as a result of a urine culture and sensitivity test will not disappear. Moreover, a quick recurrence of symptoms may result after their apparent disappearance.

In these patients, further investigation may be necessary in order to identify such possible factors as bladder stones, kidney stones, bladder tumors, etc. which may make it more difficult or impossible to eradicate the urinary tract infection with antibacterial therapy. These diagnostic studies will often include kidney x-rays or intravenous pyelograms (IVP), bladder x-rays or cystograms, or looking into the bladder with an instrument called a cystoscope (cystoscopy). Often these studies will identify some complicating factor which has either made it more likely for urinary infection to occur or has made it more difficult to eliminate.

Inadequate treatment— a common cause for recurrence

One of the most common causes for constant recurrence of cystitis is inadequate treatment with antibacterials. Often

the antibacterials will relieve the symptoms within 2 to 3 hours. *The patient then feels that she is well and discontinues taking medication. It is well known, however, that while the symptoms may be relieved, the infection is not completely eradicated and with incomplete treatment the infection is likely to recur in 1 to 3 weeks.* Accordingly, most physicians feel that adequate treatment of acute cystitis consists of a regimen of antibacterials for a minimum of 5 to 7 days, but preferably for 10 to 14 days.

In those circumstances in which the infection proves difficult to eradicate, often, treatment is continued for much longer periods of time—sometimes for many months with the amount of antibacterial being lowered gradually. *To decrease the likelihood of an acute infection becoming a chronic infection, it is important for you to complete the full course of antibacterial therapy as prescribed by your physician.* Naturally, if the infection is chronic, strict observance of your physician's medical regimen becomes even more important.

Preventing cystitis— often it could be up to you

Generally then, in the vast majority of women, urinary tract infections can be cleared up. With some, the disappearance of the infection will be quite rapid while others may require some of the more complicated and time-consuming measures discussed above. Since the chances for urinary tract infection in women are quite high, it is proba-

bly most important for all women to adopt a lifestyle which reduces the likelihood that cystitis will occur. The measures which have been outlined above, drinking a lot of liquids, urinating at frequent intervals, and the adoption of simple hygienic measures relating to bowel movements and sexual intercourse, will greatly decrease the probability of urinary tract infection.

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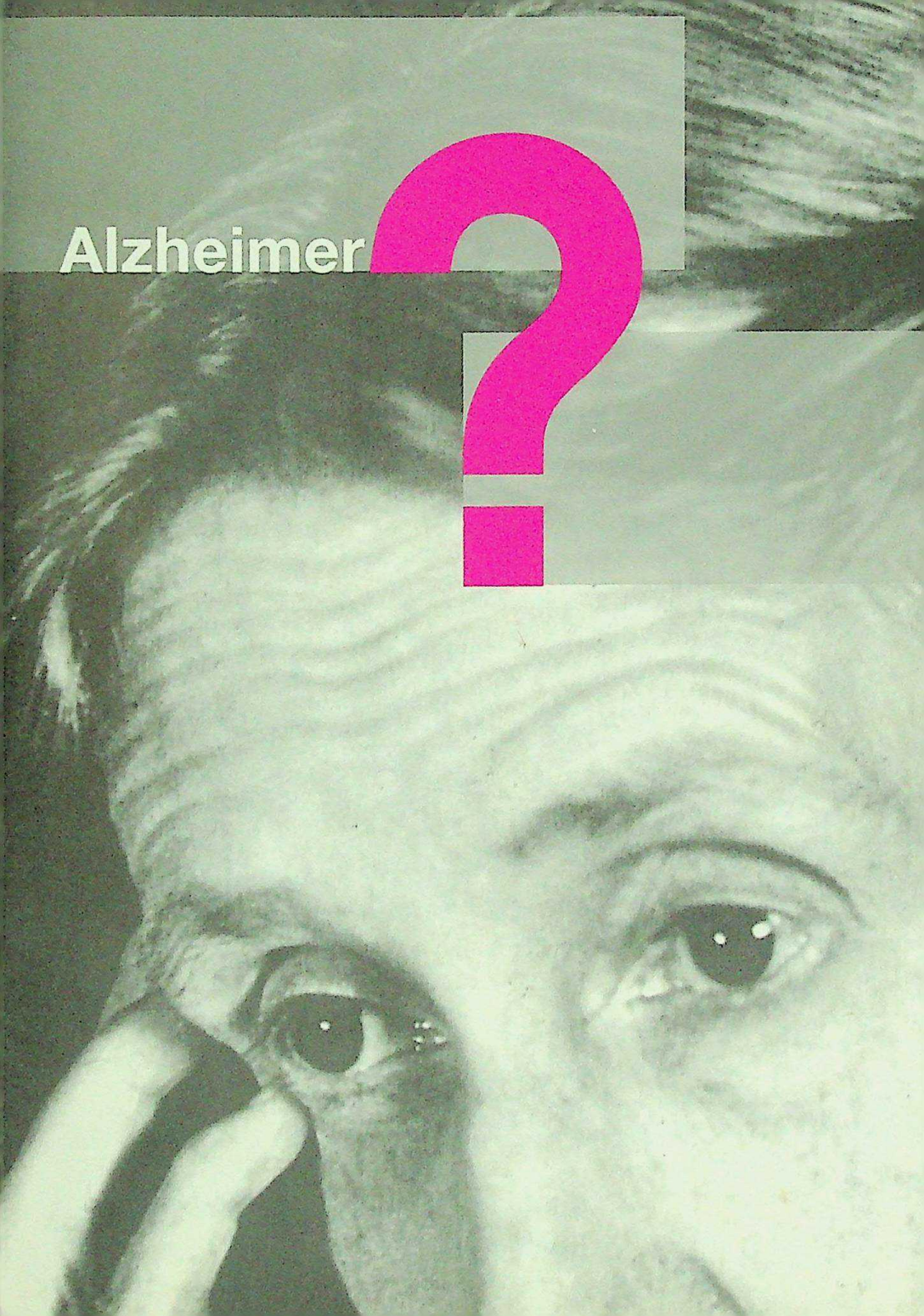
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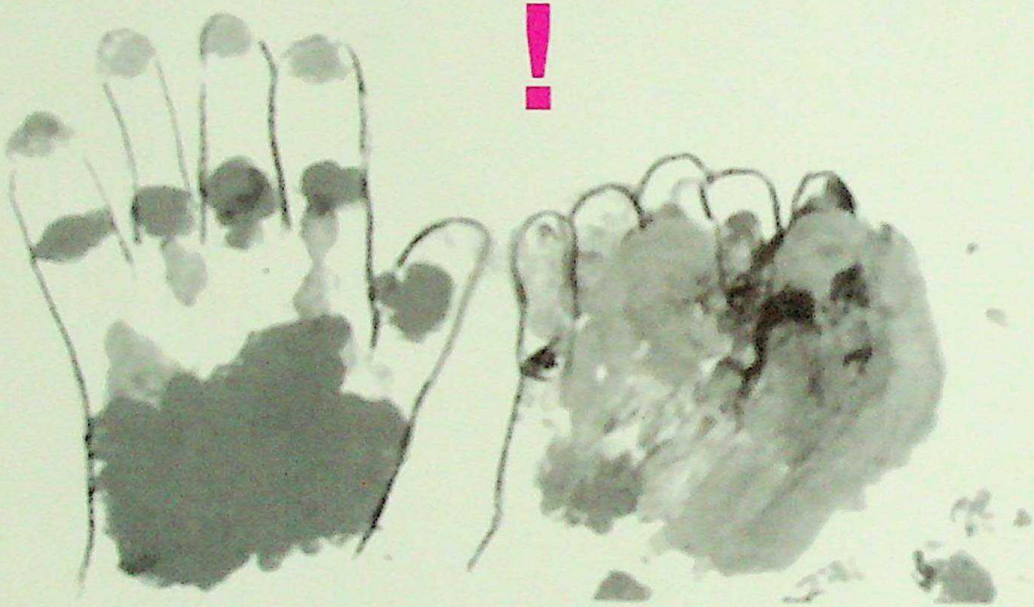

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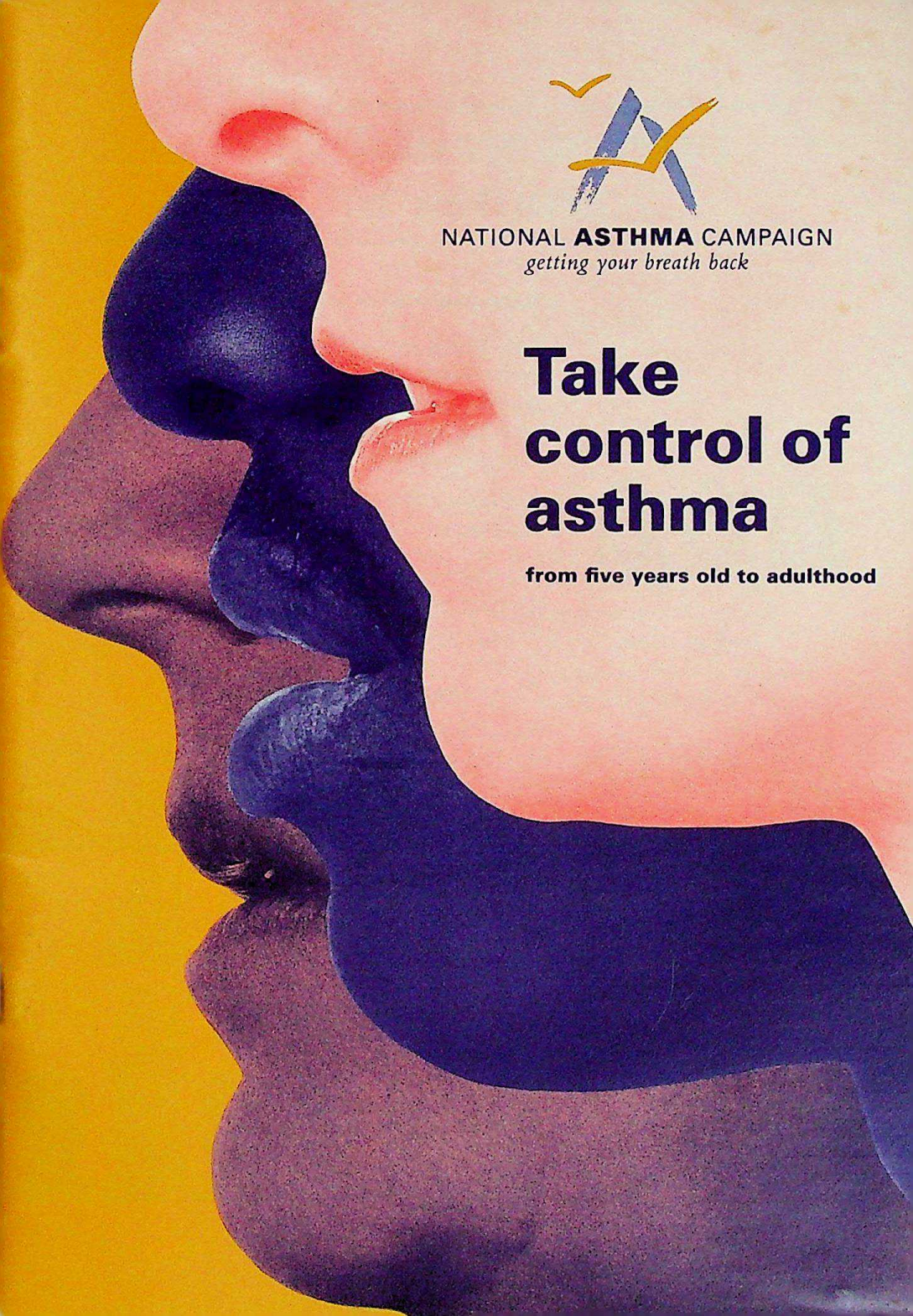


NATIONAL **ASTHMA** CAMPAIGN
getting your breath back

Take control of asthma

from five years old to adulthood

Contents

Taking control of asthma

"Before I took my asthma seriously it knocked me from pillar to post"

This booklet has been written by people with asthma and is based upon the latest medical opinion. It covers a range of subjects that should interest both those with asthma and those who care for people with asthma.

Asthma is very common indeed. Around 3 million people in Britain have it (5 per cent of the adult population). It is the most common chronic illness to affect children, causing them more time off school than any other condition.

Asthma can be distressing and it may become a serious problem if it is not properly controlled. Because it is not generally recognised as being an illness that can do serious harm, there is a tendency not to act on the information available or to reject carefully prescribed treatment.

This booklet will show how life with asthma can be mostly trouble-free and enjoyable. The majority of us can control asthma.

If after reading this booklet you have more questions ask your doctor or nurse. You can also call the Asthma Helpline on 0345 01 02 03 between 1 and 9pm, Monday to Friday (for the price of a local call).

Describing asthma

"It's like having a heavy weight placed suddenly on my chest"

No one knows everything there is to know about asthma. Understanding about asthma grows as doctors' knowledge increases and more experiences (symptoms) are now being described as asthma.

Lungs have hundreds of tiny corridors, called airways, that carry the air we breathe in and out. Those of us with asthma have airways that are almost always red and sore (inflamed). Because they are inflamed our airways are quick to respond to anything that triggers (irritates) them.

Although they vary from person to person, triggers such as flu, cigarette smoke and cold air can make our airways narrower by tightening the surrounding muscles. As our airways narrow, the inflamed and swollen lining of the airways produces a sticky mucus. This often, although not always, makes a wheezing noise when we breathe and seems to stay there even when we cough.

Common causes of attacks the triggers

Allergies Some airways may be 'angered' into narrowing because they are allergic to something that is touching them. Allergies can include furry or feathery animals, dust, certain foods and pollen. There are many others and you may have any number of them. (see page 8)

Air temperature Changing weather conditions often affect people with asthma. This is particularly the case when breathing in large amounts of cold, dry air during exercise or laughter.

Environment Pollutants such as car fumes, cigarette smoke and certain chemicals can trigger an attack.

Infections Flu and other viral infections can start an attack.

Stress Some people find that being under stress brings on an attack.

Medicines Those who first developed asthma as adults may be triggered by aspirin, non-steroidal anti inflammatory tablets (eg Brufen), some blood pressure tablets, and some eyedrops for glaucoma. Paracetamol is safe.

Hormones Some women find their asthma varies in response to changing hormone levels and before periods.

You may be affected by any one or more of these triggers at different times.

Once our airways have narrowed the result is usually one or more of the following symptoms (they are often worse at night and in the early morning or after exercise):

Coughing A cough is often a sign of asthma. Sometimes the cough produces mucus which can be white, yellow or green.

Tight chest This is a popular description among people with asthma. It feels as if a large elastic band has been placed around your chest.

Wheezing You can hear a whistling noise and often feel as though something (mucus) is catching inside the lungs.

Shortness of breath You cannot finish each breath before you need another.

Who gets asthma?

Asthma, like its related conditions eczema and hay fever, often runs in the family and may be inherited. Smoking during pregnancy and around a small child may increase the risk for that child.

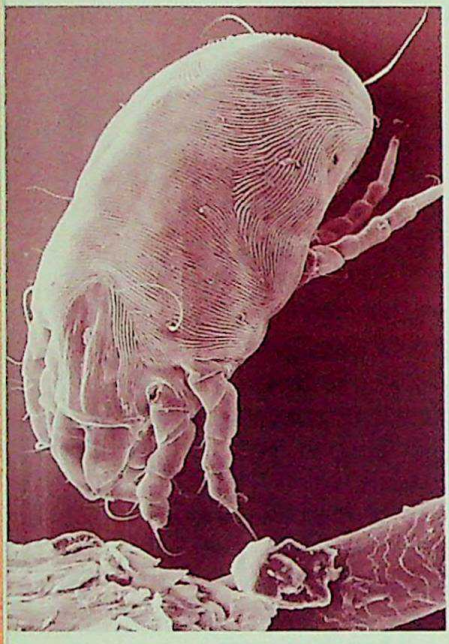
Stress and pollution have not been proven to cause asthma although certain chemicals in the workplace can.

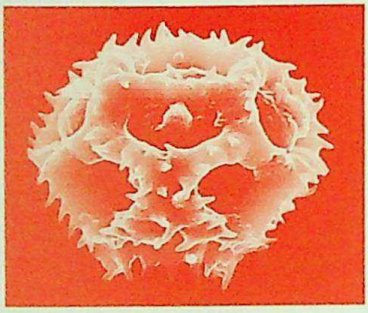
Predicting asthma

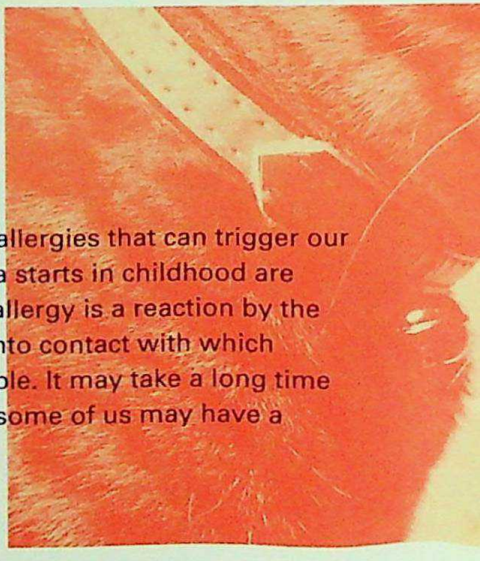
Asthma is an unpredictable illness. It can change as we get older and be triggered unexpectedly; symptoms come and go: they can last for a few moments or keep going for days.

There are some times of the day that are predictably low points. Breathing may become more difficult late at night or early in the morning. This is called nocturnal asthma. In this state we are frequently awoken up by our coughing or breathlessness. There are also many people who only get the symptoms of asthma during the day. In general we are at our best by late afternoon.

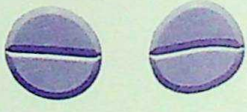
Allergies







Many of us have allergies that can trigger our asthma; people whose asthma starts in childhood are more likely to be allergic. An allergy is a reaction by the body to something it comes into contact with which doesn't affect most other people. It may take a long time for an allergy to develop and some of us may have a number of allergies.



Allergies that trigger asthma attacks

Dust mites We can become allergic to the droppings of house dust mites that live in our beds and soft furnishings.

Pollen Grass pollen grains (mainly found from mid May through to late July), tree pollens in the spring and weed pollens in the spring and autumn sometimes trigger asthma.

Animals Close contact with furry or feathery animals can sometimes trigger asthma.

Mould spores These are tiny seed-like particles that are released from mould which grows in almost any warm and damp areas. Mostly released in summer or on damp days, they remain active until late autumn and are common in damp housing.

Drugs Aspirin triggers asthma in up to 1 in 30 people. Other pain relievers and medicines are known to trigger asthma (Paracetamol is safe).

Chemicals The number of agents used in industry which are now recognised as causing asthma is over 200.

Food Food allergy is not very common and is difficult to diagnose. Dairy products, alcohol, sea food, yeast and peanuts are some of the offenders.

Controlling asthma

"I had no idea that I could do so much to improve my asthma"

At most times the symptoms of asthma can be controlled, it is not easy to avoid the triggers but we usually respond well to modern medicines.

Practical ways of avoiding trouble

Without doubt the most important part of controlling our asthma is the carefully planned use of modern treatments. However, it seems wise to look at the ways of avoiding asthma before we look at how the medicines can help to control it.

Given that there are many different things that cause asthma attacks (triggers) it is unlikely that we can avoid them all. We should consider the way we live our lives carefully and, before we visit the doctor to discuss them, draw up a list of things that make our asthma worse. The doctor will help us to avoid only the triggers that affect us. For instance there is no point in avoiding cats if you are not allergic to them. Most steps that you can take to avoid the triggers are commonsense.

In general

- Avoid cigarette smoke. Do not smoke yourself.
- Place a scarf around your face on cold days.
- Exercise outside on warmer days (unless pollution levels are high) and inside on cold ones. Although exercise can cause breathing problems modern treatments mean people with asthma can still participate.
- Ease yourself gently into vigorous exercise.
- Avoid rushing around; keep stress to a minimum.
- Always tell pharmacists that you have asthma.
- Keep healthy and avoid people who have a chest virus if possible.

Allergic triggers

- Do not keep warm-blooded pets (it can take months before a house is safe again).
- Use special non-fabric mattress covers to provide a barrier against dust mite droppings.
- Check to see if food labels include ingredients that you are allergic to.
- On hot, dry days avoid spending too much time outdoors.
- In summer avoid long grass and keep car windows closed.
- Use synthetic pillows and duvets. Wash bedding every week at 60°C.
- Use short pile synthetic carpets or vinyl.
- Soft toys should be washable, if they are not place them in the freezer regularly.
- Let others vacuum using a cleaner that retains most of the dust (see Which? June 1992).
- Have moulds removed quickly, avoid damp houses and do not hoard old clothes.

At work

- Keep away from industrial fumes, consult the local Health and Safety Executive if you are concerned.
- Ask not to work near people who smoke in the workplace.
- Balance your workload so that you are not under too much stress.

A note about treatment that reduces our allergic reactions (desensitisation). This was used in the treatment of hay fever but it is no longer thought to be safe or effective for the vast majority of people with asthma.

Modern treatments

Asthma needs to be regularly treated so that we can get on with our lives.

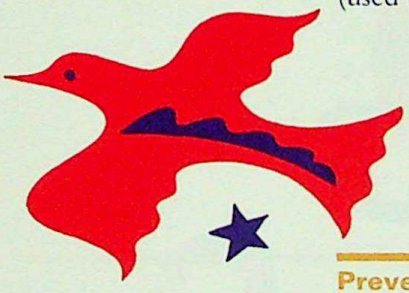
There are two types of treatment:

Preventers are those that guard against asthma happening at all.

Relievers are those that rescue us from breathing difficulty as it happens.

There is no connection between the effects of reliever drugs and preventer drugs. They do quite separate things.

Please note that the names of drugs beginning with a capital letter are their trade names (the ones you will see in shops) and ones beginning with a small letter refer to their chemical name (used by chemists and doctors).



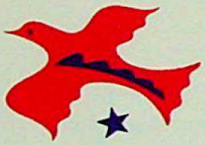
Preventers

As there is no cure for asthma itself, if we want to stop having attacks and being restricted in what we can do, then preventing the symptoms is the best answer.

Treatments, called preventers, build up a protective shield in the lining of our airways that makes them less likely to narrow when triggered. They do not bring any instant relief from symptoms of asthma and will not work unless they are used regularly. Preventers save lives and may restore the quality of our day-to-day living.

Intal (cromoglycate) is a non-steroid preventer, it protects against two of the five main triggers: exercise and allergy induced asthma. However, these days most preventers are based on steroids and Intal is now usually only the first line of treatment for children with allergic asthma.

Low dose inhaled steroid treatments are the modern answer for adults, they may also be used by children who are not helped by Intal. Examples are beclomethasone (Becotide or Becloforte) or budesonide (Pulmicort). They work by acting slowly on the airways to reduce the inflammation and mucus.



We will discuss any risks of taking steroids on page 18 in the section covering medicines.

Preventers must be used regularly and may take up to 14 days to build up the preventive shield.

Relievers

Reliever treatments or bronchodilators are the ones that rescue us from breathing difficulties as they happen. They are designed to relax the muscles surrounding our airways by opening them and allowing us to breathe again. Relievers do not reduce the inflammation in our airways.

The reliever should only be used when the symptoms of asthma appear. The only exceptions to this rule are the long acting reliever drugs that need to be taken regularly.

Short acting rescue relievers

The most commonly used relievers are based on adrenaline, the chemical we produce naturally when taking exercise. It opens the airways and makes our heart beat faster so that the body is prepared for action. Doctors call this class of drugs adrenergic bronchodilators. The newer adrenergics have their main effect on the lungs and much less effect on the heart. Salbutamol (Ventolin) and terbutaline (Bricanyl) are two examples and they are effective for around four hours.

Long acting relievers

The long acting reliever drugs include the inhaled oxitropium (Oxivent), the newer salmeterol (Serevent) and the xanthine tablets (Slo-Phyllin and Uniphyllin are two examples). Serevent and Oxivent go on working for a longer time than the short acting relievers but they do not deal with the inflammation in our airways. Because they are long acting doctors may use them on a regular basis to keep control of asthma that lingers despite regular preventer medicine and short acting relievers. The xanthine tablets last for around eight hours.

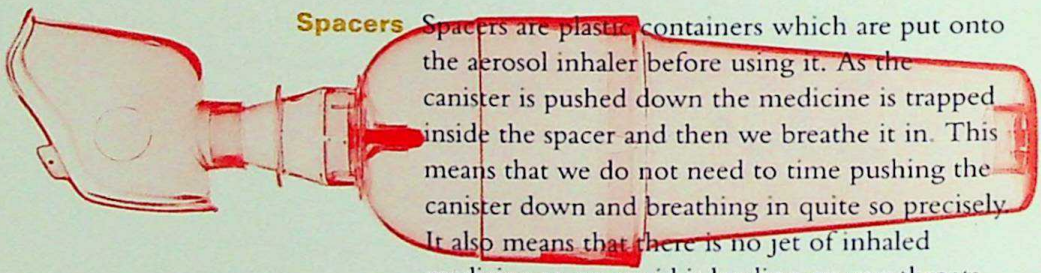
Relievers show us how well our asthma is being controlled. If we are using them frequently it could mean that our asthma is not under control or is getting worse. Don't be embarrassed if you feel that you are using too much of the reliever – but do talk to your doctor about it.

Because asthma starts in the lungs most of the drugs we use to control it are designed to be breathed in (inhaled). This means that they reach the airways quickly and the doses needed are small. There are also medicines which can be swallowed (taken orally) or injected. We will look closely at the most common type, inhaled medicines. There are many variations of inhaler, so if one does not work very well doctors can recommend another.

Aerosol inhalers puffers

These are the most popular devices for both reliever and preventer treatments. They work in the same way as other sprays: the medicine is mixed into a liquid and forced under pressure into a small canister. When used the liquid quickly evaporates leaving the active medicine as a fine dust which is breathed in. Each time the canister is pushed down a measured (metered) amount of the medicine is released and takes half a minute to recharge ready for the next dose.

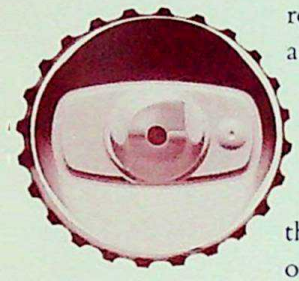
Autohaler The autohaler is another type of aerosol inhaler. Rather than waiting until the canister is pushed down, the autohaler works automatically as we breathe in.



Spacers Spacers are plastic containers which are put onto the aerosol inhaler before using it. As the canister is pushed down the medicine is trapped inside the spacer and then we breathe it in. This means that we do not need to time pushing the canister down and breathing in quite so precisely. It also means that there is no jet of inhaled medicine so we avoid it landing on our throats and being swallowed. They are often recommended to reduce the side effects from the higher doses of inhaled steroids and for children who cannot co-ordinate using their inhalers.

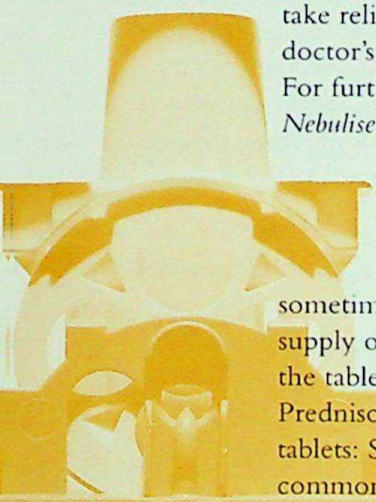
Powder inhalers

These inhalers use a dry powder version of the medicine. This may come inside a small container (capsule). The capsule is broken open inside the inhaler device and we breathe in the fine powder. In other devices the powder comes inside a disk or compartment inside the inhaler. Powder inhalers are used for both preventer and reliever medicines. Examples are the Rotahaler, Diskhaler and Turbohaler.



Nebulisers

Nebulisers work by forcing air or oxygen through the medicine to make a fine mist. They are occasionally used so that very young children can take their preventers, such as Intal. More often nebulisers are used to give us reliever medicines when we have severe asthma. Here the nebuliser is useful because it allows bigger doses of medicine to be taken, but there are other ways to have bigger doses, for instance by using a spacer. If we use a nebuliser to take reliever medicines it is very important to follow our doctor's instructions carefully and not to take extra doses. For further information about nebuliser's please read *Nebulisers for Asthma*.



Tablets

When our asthma is severe steroid tablets are sometimes taken for a short while. We can be given a supply of tablets to use when the asthma slips out of control; the tablets generally used are called Prednisilone or Prednisone. Doctors also occasionally prescribe relievers as tablets: Salbutamol, Terbutaline and Theophylline are common examples.

Side effects

Medicines may have unwanted side effects. The treatments we use for asthma do not generally have any serious side effects. Some relievers can produce distressing effects such as 'the shakes' or a feeling of nausea; these effects are more common when taking high doses.

Steroids

The word 'steroid' covers many groups of chemical substances produced naturally by the body. Only one group, the corticosteroids, is used to treat asthma. Corticosteroids are simply steroids manufactured by pharmaceutical companies to match the body's own steroids. They are not the same as anabolic steroids used by athletes to improve their performance.

Steroids are mostly inhaled in small doses. They carry a small risk of mouth infection or voice 'huskiness'. If we need very high doses of inhaled steroid (more than 1,000 micrograms per day of budesonide or beclomethasone) to control our asthma it is possible that we may absorb some steroid. Even if this happens the dose is likely to be smaller than the smallest steroid tablet. We can reduce the risk by using a spacer device and by rinsing our mouths after using the inhaler.

The main serious risk comes from steroid tablets taken over a long period of time. Because there is a risk of side effects (weight gain, thinning of bones and increased blood pressure) the doctor will agree a decision with us first and then decide whether the benefits outweigh the risks. Short term steroid tablets (for 7 to 14 days) should not cause any long term side effects.

Complementary treatments

Many people have suggested that complementary treatments have improved their asthma. Because complementary treatments have not undergone the same strict trials that medicines have, doctors will rarely recommend them with the same certainty that they can with preventers and relievers.

Some trials have been made and there are some positive results. The evidence that acupuncture can relax wheezing, for instance, is quite good and it can be shown to protect against exercise-induced asthma. Acupuncture does not, however, seem to be of use in the long term treatment of asthma.

Hypnosis and relaxation techniques such as yoga may relieve stress in an individual and this can help their asthma. However, some popular products such as ionisers have not been proven to be effective in reducing symptoms.

The choice to take complementary treatment is ours, although it is worth remembering that asthma varies from person to person and just because someone else improves it is not proof that it will help others.

Complementary treatments are often wrongly called 'alternative' treatments: there are no alternatives to modern medicines. If you decide to consult someone other than your doctor remember to continue using your preventer and reliever medicines.

Keeping asthma controlled

Working as a team

We have seen what the two types of treatment on offer do for us. Now it is worth looking at how they will be used to control our asthma.

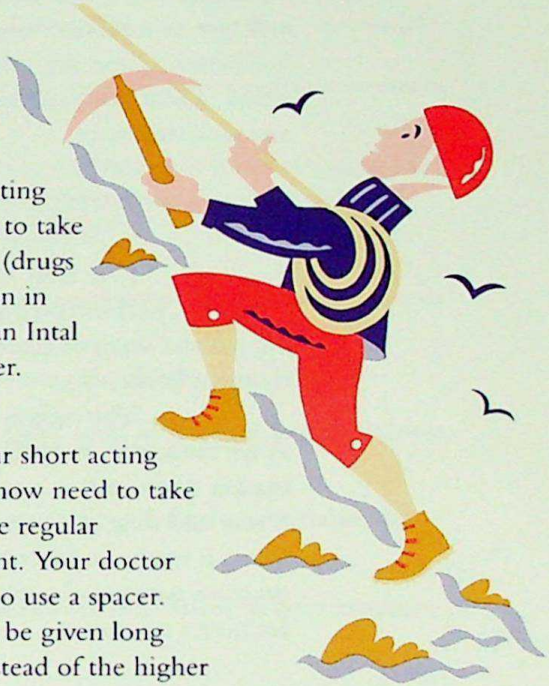
Asthma is an illness that gives us, the patients, a lot of responsibility to manage things for ourselves. Generally speaking we are good judges of our own day-to-day health and we can decide whether to increase or decrease the treatment we are taking.

Doctors have made what they call the 'stepwise treatment plan' (illustrated opposite). Each step shows us what treatment is needed to control our asthma. If the treatment on one step is not controlling our asthma then our doctor will recommend moving up to the next step. If our asthma is well controlled then our doctor may recommend moving down to the next step.

The success of stepwise treatment relies on our reporting back to the doctor or asthma clinic and telling them about any difficulties and how we are doing. Our doctor must know that we have taken the treatment on that step, otherwise she may make the wrong decision and place us on more or less of the medication.

Step 1

You can occasionally use a short acting reliever. If you are using it more than once a day you should go onto the next step.



Step 2

In addition to your short acting reliever, you will now need to take regular preventer treatment (drugs that reduce the inflammation in your airways). This can be an Intal type drug or a steroid inhaler.

Step 3

In addition to your short acting reliever, you will now need to take higher doses of the regular preventer treatment. Your doctor may also ask you to use a spacer. Some people may be given long acting relievers instead of the higher dose preventer.

Step 4

In addition to your short acting reliever and high dose preventer you may also need one or both of the following treatments:

- Long acting reliever
- Different type of short acting reliever

Step 5

In addition to your short acting reliever, high dose preventer using a spacer, one or more of the long acting relievers, you may be given regular steroid tablets in single daily doses.

Measuring our performance

One way in which we can help ourselves and our doctor is to measure how our lungs are performing. This will give us a picture of when our asthma is worse and when it is better. Using this information we can better predict an attack, adjust our treatment accurately and see the benefits of any new treatment.

Peak flow meters

Peak flow meters are the best way of measuring our lungs' performance and this can help to diagnose asthma and warn of trouble. We can be prescribed one to use at home.

The meters have a marker which slides up a scale as we blow into it. At the point when we blow hardest the marker stops next to a number. Each time we use the meter (every morning and evening) the best result from three blows is marked onto a chart. By comparing it with our previous pattern of results we can look for gradual changes for better or worse.

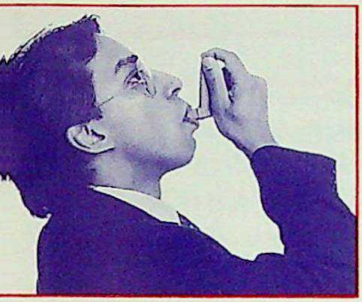
Signs that our asthma is slipping from control

As well as checking our lungs with peak flow meters there are other signs that show us whether we are in control of our asthma or not. If we have any of the following signs, then we should see our doctor so that he/she can change our treatment to bring our asthma back under control:

- Needing our reliever more often, or if it has a less good effect which doesn't last for long.
- Waking at night with coughing, wheezing, shortness of breath or chest tightness.
- Cannot keep up with our usual level of activity or exercise.

Asthma attacks

As a part of our asthma management we should discuss with our doctor what to do in the event of an asthma attack.



What is an attack?

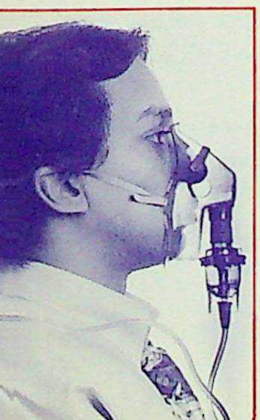
- When the reliever treatment is not rescuing you from breathing difficulties after the first 5–10 minutes of taking it.

What to do

- Take a reliever again after 5–10 minutes.
- Try to stay calm; relax as much as your breathing will let you.
- Sit in a position that you find comfortable, do not lie down.
- Rest your hands on your knees to help support your back.
- Try to slow your breathing down as this will make you less exhausted.

Whom to ask for help

Each situation is different and we should discuss with our GP whom to contact in the event of an asthma attack. In rural areas, particularly, it may be the case that the GP is quicker at getting to the scene and can help us avoid being admitted to hospital. If you are having difficulties contacting your GP during an attack or are away from home call an ambulance.



Do not be afraid of causing a fuss. Doctors prefer to be called early so that they can easily alter our medication to make us well again.

What next?

We know about asthma in the present – but what about the future?

Growing out of asthma

It is impossible to decide whether any one person is likely to 'grow out of' asthma. Many children do grow out of asthma – but if we have asthma at the age of 14 there is an 80 per cent chance of it continuing into adult life.

If you are an adult with asthma the chances are that it will not go away, although the symptoms should be controlled by treatment.

Will our asthma get worse as we get older?

Not necessarily; in fact our asthma can get better. Being realistic we should accept that asthma is a long term condition, but it is not necessarily a deteriorating one. If we take our preventer treatments regularly this will improve our chances of controlling asthma.

A cure for asthma?

As this booklet is being written there is no cure for asthma. Thanks to research this may change in the future. One thing is for certain: the outlook for asthma research has never looked so good.

Researchers are tackling asthma from many different directions: pollution, allergies, infant asthma, cell biology and chemical structures are just a few. Not only are they looking for a cure but they are also looking at ways to improve asthma care and the quality of our lives today.

National Asthma Campaign

The National Asthma Campaign is the only charity dealing exclusively with asthma and related allergies. It funds research into asthma and provides information to a host of people including doctors, nurses, people with asthma and their friends, teachers and employers.

The Campaign has a network of local branches around the United Kingdom. Each branch offers support to the local people and raises money to help support the charity.

The National Asthma Campaign works at home and abroad to promote the interests of people with asthma. It raises the public's awareness of asthma and does everything in its power to ensure that no person with asthma suffers needlessly.

Getting more information

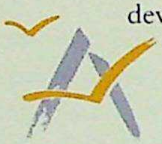
Use your local health services; they are free and with regular visits and teamwork you have the best chance of controlling your asthma. If you would like additional information you may like to do the following:

- To speak to an asthma nurse call the Asthma Helpline on 0345 01 02 03 between 1 and 9pm Monday to Friday, calls are charged at local rates.
- Subscribe to Asthma News, the National Asthma Campaign's quarterly newspaper.
- Contact your National Asthma Campaign local branch.
- If you are 12 or under you may like to join the Junior Asthma Club.

- Write to the National Asthma Campaign, enclosing a self-addressed envelope at least 10 inches by 7 inches, for an information leaflet on any of the following subjects:

1 Asthma in the under 5s
2 Nebulisers for asthma
3 Exercise and asthma
4 Asthma at school
5 The asthmatic on holiday
6 Asthma and pregnancy
7 Peak flow measurement
8 Asthma at work:
are you eligible for compensation?
9 Hay fever
10 Steroid Treatment for asthma
11 Asthma and the environment

We also have Fact Sheets covering the latest developments and ongoing issues



NATIONAL **ASTHMA** CAMPAIGN
getting your breath back

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Providence Place
London N1 0NT

If you want to help...

Your donation towards asthma research will help us to find new ways of treating this devastating disease. We rely almost entirely on voluntary contributions and last year spent nearly £2,000,000 on vital research.

I would like to make a donation of:

☐ £50 ☐ £25 ☐ £12 ☐ £5

☐ Other _____

If you need support...

If you or your child has asthma you can obtain regular information and help by becoming a member. Please tick one box

☐ Annual membership minimum £5

☐ Overseas membership (Europe) minimum £7

☐ Overseas membership (outside Europe) minimum £11

£_____ Total donation

£_____ Total membership

£_____ Grand total

Method of payment

☐ I enclose a cheque payable to the National Asthma Campaign

☐ Please debit my Access/Visa/NAC Mastercard (delete as necessary). My card number is:

_____

Expiry date _____

☐ I wish to pay by Banker's Order, please send me a form.

If you pay tax...

You can increase the value of your regular gift by one third at no extra cost to yourself by taking out a Deed of Covenant. A covenanted donation of £60 per year is worth £80 per year to the National Asthma Campaign. Please tick this box for further details on taking out

☐ a Deed of Covenant.

Signed _____

Date _____

Name _____

Address _____

_____

Postcode _____

Please return this form to the
National Asthma Campaign
Providence House
Providence Place
London N1 0HT
Registered charity number 802364

Asthma...

- is the most **common chronic disease** in the developed world
- is the only **treatable** condition in the western world which is **increasing in prevalence**
- affects around **3 million people** in the UK including more than **750,000 school children**
- causes **more time off** school than any other condition
- prevents people working on over **7 million days** each year
- attacks cause **100,000 people** to be admitted to **hospital** each year
- causes around **2,000 deaths** each year: 80 per cent could be avoided

please help to control asthma

GLOBAL DATABASE ON BLOOD SAFETY

Summary Report

1998–1999

World Health Organization
Blood Transfusion Safety
Department of Blood Safety and Clinical Technology
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Tel: +41 22 791 4385 Fax: +41 22 791 4836
http://www.who.int/HTP/BCT/BTS
E-mail: bloodsafety@who.int

Background

Millions of lives are saved each year through blood transfusions. In most developing countries, however, people still die due to an inadequate supply of blood and blood products. This has a particular impact on women (as a consequence of pregnancy-related complications), children (malnutrition, malaria and severe life-threatening anaemia), trauma victims and, especially, the poor and disadvantaged. It is estimated that up to 150 000 pregnancy-related deaths each year could be avoided with adequate transfusion therapy.

The emergence of HIV in the 1980s highlighted the importance of ensuring the safety, as well as the adequacy, of national blood supplies. In many countries, even where blood is available, many recipients remain at risk of transfusion-transmissible infections (TTIs) as a result of poor blood donor recruitment and selection practices and the use of untested units of blood.

WHO strategy for blood safety

The World Health Organization (WHO) has identified blood safety as a health issue requiring high priority and launched the Global Collaboration for Blood Safety (GCBS) as a worldwide effort to improve blood safety by building on knowledge, utilizing existing expertise, promoting dialogue and suggesting realistic, effective and practical mechanisms.

WHO has developed the following strategy for global blood safety, which is described more fully in the WHO *Aide-Mémoire: Blood Safety.*

Organization and management

The establishment of well-organized, nationally-coordinated blood transfusion services with quality systems in all areas.

Blood donors

The collection of blood only from voluntary non-remunerated donors from low-risk populations.

Blood screening

The screening of all donated blood for transfusion-transmissible infections, including HIV, hepatitis viruses and syphilis; blood grouping; compatibility testing; blood processing.

The clinical use of blood

A reduction in unnecessary transfusions through the appropriate clinical use of blood.

WHO Global Database on Blood Safety

Following the launch of the Global Collaboration for Blood Safety, it became apparent that baseline information was required about blood transfusion services in Member States to identify the exact nature of problems and develop appropriate strategies.

The WHO Global Database on Blood Safety (GDBS) was therefore established to obtain data on blood transfusion services in all Member States of the World Health Organization, with the following objectives:

- To assess the global situation on blood safety
- To obtain best available information on blood transfusion services in each Member State
- To identify problems and needs in order to provide appropriate technical support
- To identify countries for priority assistance
- To monitor progress and trends in blood safety.

A questionnaire, based on the *Aide-Mémoire*, was developed in 1997 as a tool for the standardized collection of data from Member States and was sent to national heath authorities for completion. The status of blood transfusion services in selected countries was also assessed during field visits by WHO consultants, whose observations assisted in the analysis of the data.

Data analysis

Data was obtained from 175 of the 191 Member States and was analysed on a regional and global basis. Since significant differences were revealed between some countries in the same regions, a common factor was sought to enable meaningful analysis. The Human Development Index (HDI), devised by the United Nations Development Programme *(Human Development Report,* UNDP, 1999), satisfied this requirement.

The Human Development Index classifies countries as having a low, medium or high HDI, based on the following criteria:

- Life expectancy
- Educational attainment
- Adjusted income.

In the majority of developing countries (low and medium HDI), there is little systematic collection of data at national level due to a lack of coordination of blood transfusion services. The data obtained from these countries was therefore limited to information from the main centres, usually based in cities.

Key observations

Global blood supply

Globally, more than 75 million units of blood are donated each year. Although the majority of the world's population live in low or medium HDI countries, around 60% of the global blood supply is donated in countries with a high HDI, as shown in Table 1.

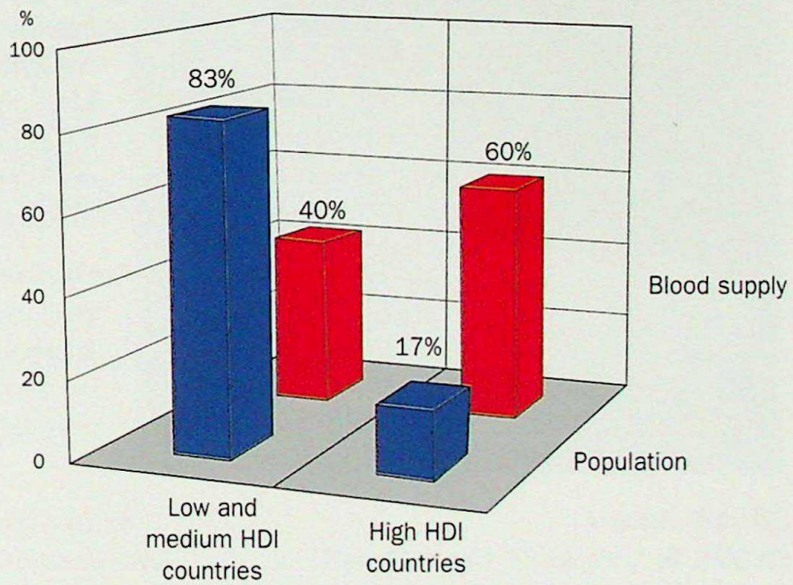
Table 1: Global annual blood donations, analysed according to HDI criteria, 1998–1999

	Low HDI countries (n=41)		Medium HDI countries (n=89)		High HDI countries (n=45)	
Blood supply, in millions of units and by percentage	1.3m	1.7%	28.9m	38.5%	44.9m	59.8%
Estimated blood donation rates per 1000 population	Average Range	2 0.3–5.3	Average Range	10 1.7–50.3	Average Range	40 10.4–74.0

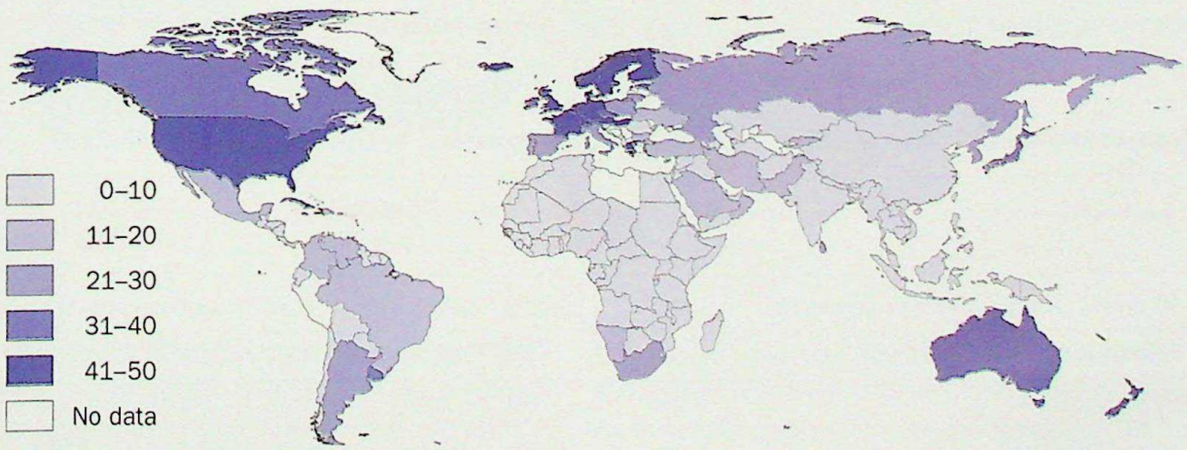
Analysis of the blood supply in relation to the population reveals that 83% of the world's population has access to only 40% of the global blood supply (Figure 1).

The blood donation rate per 1000 population is almost 20 times higher in developed countries (high HDI) than in countries with a low HDI (Map 1).

Figure 1: Global population and global blood supply, 1998–1999



Map 1: Number of whole blood donations per 1000 population, 1998–1999

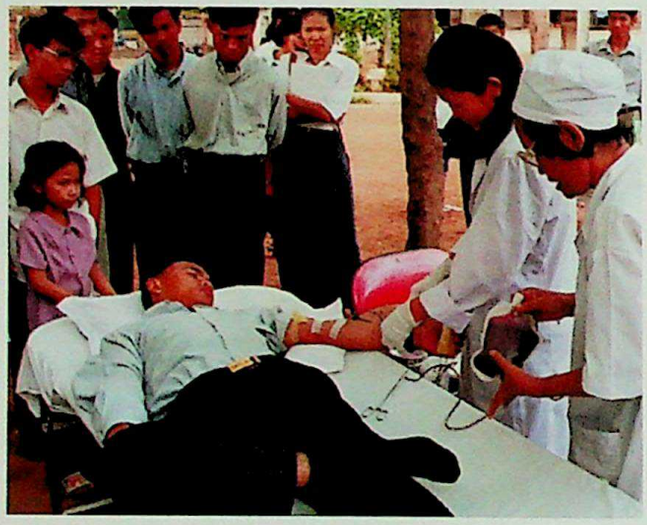


Organization and management

The safety and adequacy of the blood supply is dependent on the commitment of each national health authority to the establishment of a well-organized, nationally-coordinated blood programme. This requires official recognition of a specific organization with sole responsibility for blood transfusion services, an adequate budget and a national blood policy and plan, supported by a legislative and regulatory framework that governs all activities.

GDBS data indicates marked differences globally in the formulation and implementation of national blood policies. In the developed world (high HDI), 94% of countries with strong government commitment and support reported the implementation of a national blood policy and plan. In comparison, national policies have been implemented in only 59% of low and medium HDI countries, particularly those with hospital-based services. Only 20% of countries reported that all aspects of a well-organized BTS were in place.

A key indicator of a well-organized and coordinated national blood programme is a successful programme for the recruitment and retention of voluntary non-remunerated blood donors. Using this indicator, a marked difference is evident between countries with a nationally-coordinated blood transfusion service and those without, regardless of HDI classification.



Blood donors

In 1975, the World Health Assembly passed Resolution WHA 28.72 urging all Member States to promote the development of national blood transfusion services based on voluntary non-remunerated blood donation.

Regular, voluntary non-remunerated donors from low-risk populations are the safest blood donors. A number of studies have shown that family/replacement and paid donors have a higher incidence and prevalence of transfusion-transmissible infections than voluntary non-remunerated donors.

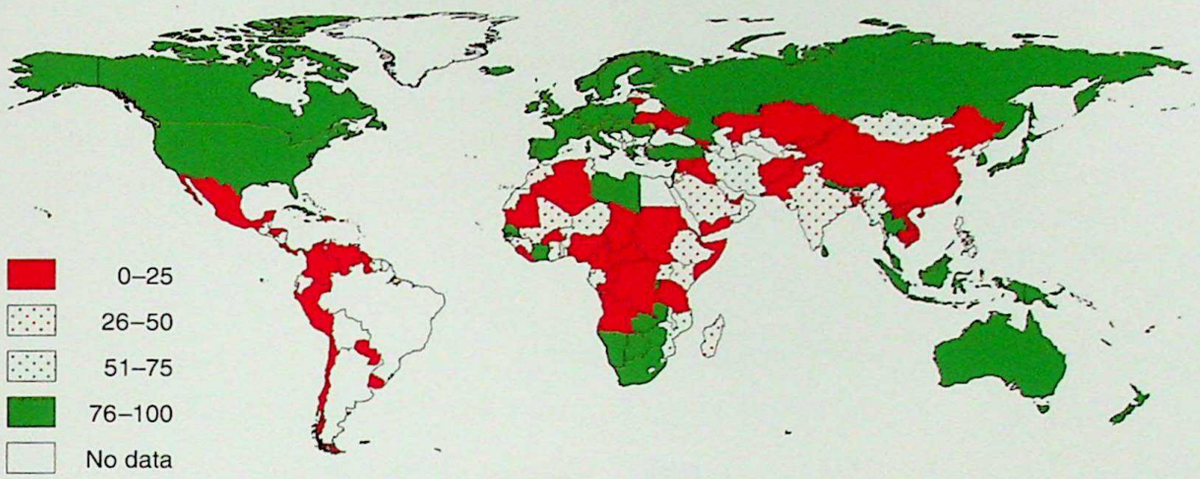
Unfortunately, the World Health Assembly Resolution has not been translated into reality in many low and medium HDI countries since it was adopted more than 25 years ago, as indicated by Table 2 and Map 2.

In low and medium HDI countries less than 40% of blood donations were from voluntary non-remunerated blood donors. In contrast, 98% of donations in high HDI countries were from voluntary non-remunerated blood donors.

Table 2: Estimated number (in millions) and percentage of donations, by type of donation, 1998–1999

	Low HDI countries		Medium HDI countries		High HDI countries	
Voluntary non-remunerated donations	0.4m	31%	11.6m	40%	43.9m	98%
Family/replacement donations	0.8m	61%	11.7m	41%	1.0m	2%
Paid donations	0.1m	8%	5.6m	19%	0.03m	n/a
Total donations	**1.3m**	**100%**	**28.9m**	**100%**	**44.93m**	**100%**

Map 2: Percentage of voluntary, non-remunerated blood donations, 1998–1999

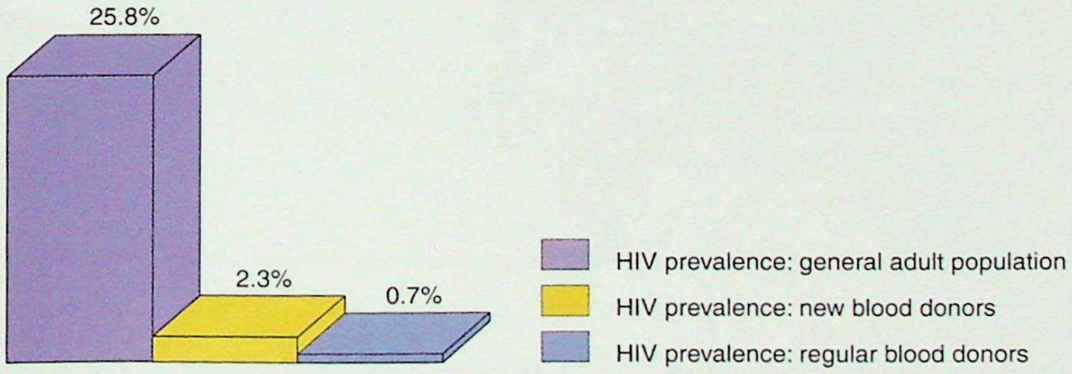


The analysis clearly illustrates that the lack of a well-organized blood donor programme based on voluntary non-remunerated blood donation leads to dependence on family/replacement blood donors. This paves the way for a 'hidden' paid and unsafe donation system since families may pay others to donate.

Globally, there were about 6 million donations from paid donors and 13.5 million from family/replacement donors. Up to 60–70% of donations in the developing world were given by family/replacement or paid donors, often in countries where the seroprevalence of HIV and other infectious agents, such as hepatitis B and hepatitis C, is relatively high.

Best practice has shown that, even in high prevalence areas for infections such as HIV, a well-organized programme of voluntary non-remunerated blood donation and effective donor selection procedures can achieve a low prevalence of infectious disease markers in the blood donor population. This is clearly demonstrated by model blood transfusion services such as those in Zimbabwe (Figure 2) and South Africa.

Figure 2: HIV prevalence in blood donors compared with the general adult population in Zimbabwe, 1998–1999



Blood screening

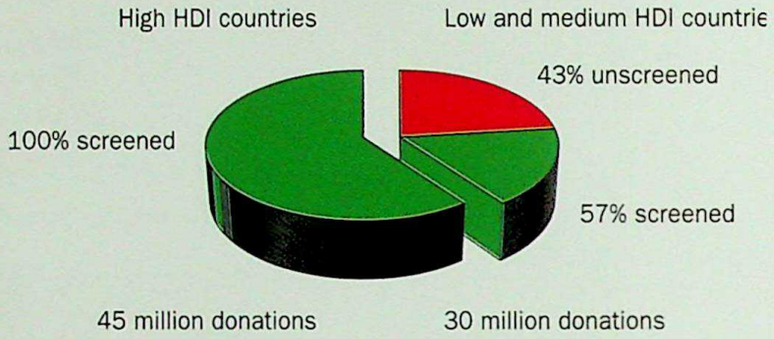
The WHO strategy for blood safety recommends that all donated blood should be tested for HIV, hepatitis B and syphilis. Where feasible and appropriate, all donated blood should also be screened for hepatitis C, malaria and Chagas disease. Screening for transfusion-transmissible infections, coupled with appropriate donor selection, has a major impact on reducing the risk and further spread of these infections.

GDBS data indicates that more than 40% of donated blood was not screened for TTIs in low and medium HDI countries. This means that around 80% of the world's population had access to only 20% of the global supply of safe, screened blood.

Millions of patients who are transfused with untested blood are therefore at risk of transfusion-transmissible infections.

Up to 13 million units of the global blood supply were not screened for all relevant transfusion-transmissible infections, mainly in low or medium HDI countries

Figure 3: Screening of the global blood supply, 1998–1999



The most common causes of poor blood screening programmes include irregular supplies of high quality reagents and test kits, inadequately trained and skilled staff, and an absence of quality assurance programmes and screening strategies for transfusion-transmissible infections.

Analysis of the data regarding ABO and RhD grouping and compatibility testing indicates that more than 70% of countries perform these tests. However, there is no information on the level of standardization of these test procedures.

ABO incompatibility remains one of the major causes of transfusion-associated mortality, often resulting from inappropriate testing and a lack of standard operating procedures.

Analysis of other key elements in the immuno-haematology laboratory reveals a general lack of traceability because of inadequate documentation of patients requiring transfusion and failure to preserve patients' blood samples.

This highlights the need for the implementation of:

- Appropriate testing of all donated blood for relevant transfusion-transmissible infections and blood group serology
- Good laboratory practice
- Adequate procedures for the identification of blood donors, blood units, blood samples and the recipients of blood and blood products.

The clinical use of blood

Transfusion should be prescribed only to treat conditions that might result in mortality or significant morbidity and that cannot be prevented or managed effectively by other means. The effective clinical use of blood and blood products therefore requires a reduction in unnecessary transfusions and the use of intravenous replacement fluids and other simple alternatives to transfusion, wherever possible.

A number of studies in both the developed and developing world have reported considerable variations in prescribing practice, often with a high number of unnecessary transfusions.

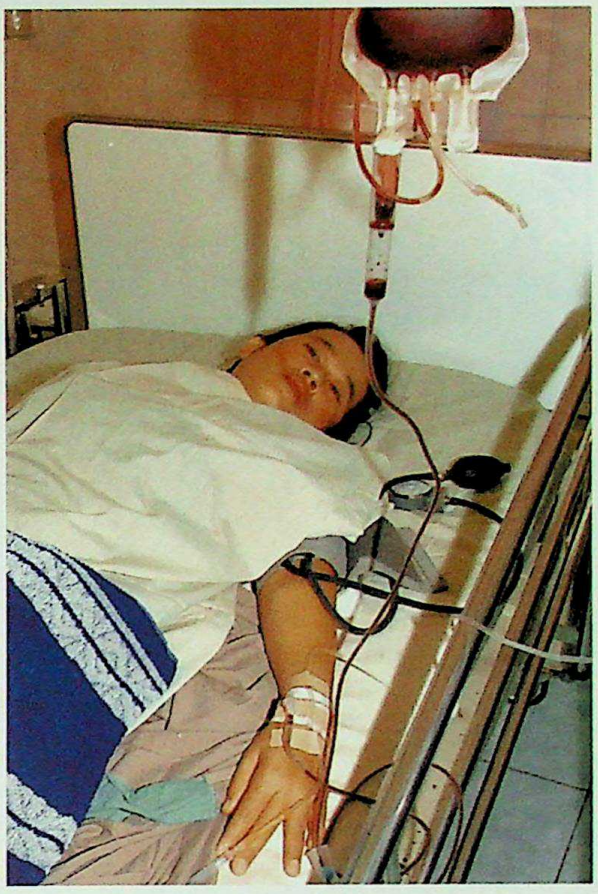
The inappropriate use of blood and blood products, coupled with the transfusion of unscreened or improperly screened units, particularly in countries with poor blood programmes, increases the risk of TTIs to recipients. It also widens the gap between supply and demand and contributes to shortages of blood and blood products for patients requiring transfusion.

National policies and guidelines are required to encourage the appropriate clinical use of blood, together with systems for the monitoring and evaluation of clinical transfusion practice. GDBS data indicates that the majority (>60%) of low and medium HDI countries do not have national policies and guidelines on clinical blood usage.

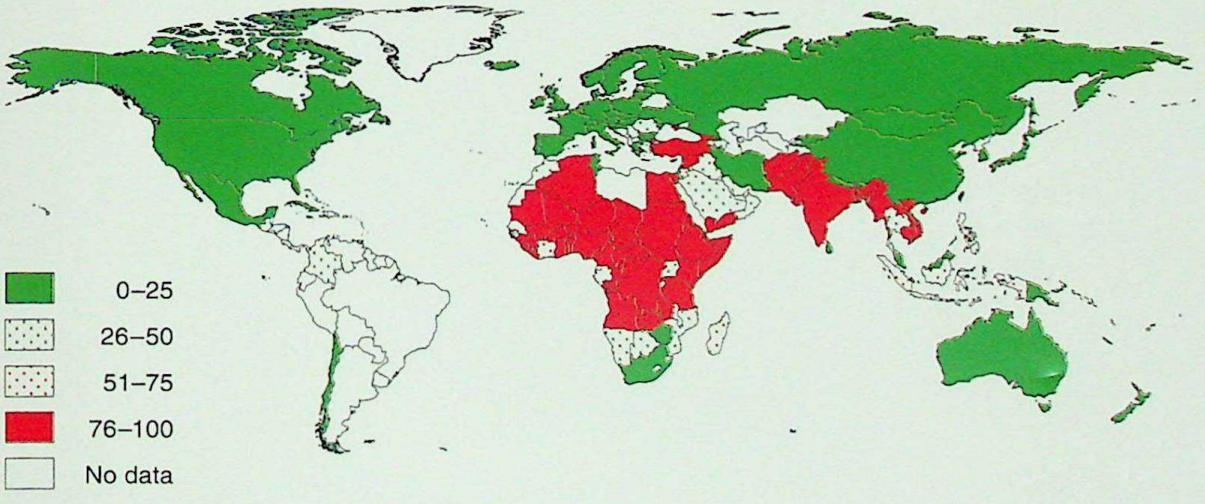
The appropriate use of blood and blood products is also dependent on consistent, adequate supplies of plasma substitutes, including crystalloid and colloid solutions. While more than 70% of countries report that plasma substitutes are available, field observations in many low and medium HDI countries suggest that these are often not readily accessible to patients when needed.

Effective clinical transfusion practice requires that whole blood is separated into its various components so that the right component is available for the right patient. The availability and use of blood components is limited in many low and medium HDI countries, as shown in Map 3, often as a result of a lack of organization, a poor infrastructure and a low level of awareness about the appropriate clinical use of blood.

GDBS data indicates that the use of whole blood is ten times higher in low and medium HDI countries than in the developed world, resulting in inadequate provision of life-saving support for patients requiring specialized treatment with blood component therapy.



Map 3: Percentage of blood transfused as whole blood, 1998–1999



Training

Blood transfusion services require a comprehensive, multi-disciplinary approach to training for all personnel, including donor recruitment and blood collection staff, laboratory staff, medical officers and quality managers. Training is also required for clinicians who prescribe transfusion.

Inadequate training jeopardizes the safety of blood and blood products and adversely affects the quality of care for patients requiring transfusion.

Analysis of GDBS data suggests that the facilities and infrastructure required for appropriate training are not

available universally, despite a recognition of training needs in both the developed and developing world. Globally, 72% of countries cannot meet identified training needs and many workers remain unfamiliar with quality concepts and the application of quality management tools that can improve efficiency without extra effort or resources.

A new initiative by WHO

Recognizing the need for capacity-building, WHO initiated the Quality Management Project (QMP) for Blood Transfusion Services in 2000.

This global project aims to improve blood safety through regional training programmes in quality management, the establishment of Regional External Quality Assessment Schemes and the creation of Regional Quality Networks.

Conclusions

The data generated from the GDBS has been invaluable in assisting countries to prioritize their needs in strengthening their blood safety programmes. It has also been an important tool for major programme initiatives by the WHO Blood Transfusion Safety Team, including the GCBS and the Quality Management Project, and will assist in monitoring progress, setting priorities and allow for re-planning of activities to achieve global blood safety. The data has also been used extensively in the preparation of WHO guidelines, recommendations, learning materials and other documents.

The Global Database on Blood Safety is a dynamic, ongoing project. WHO has recently modified the GDBS questionnaire to widen its scope and it is being distributed to national health authorities for data collection for the period 2000–2001.

Acknowledgements

WHO wishes to thank the Ministry of Health and Welfare, Government of Japan, for its continued support to the GDBS project.

The invaluable effort of all WHO regional and country offices, national health authorities, directors of blood transfusion services and national blood programme managers is also fully acknowledged.

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Guidelines for the Safe Transport of Infectious Substances and Diagnostic Specimens

Geneva, 1997

WORLD HEALTH ORGANIZATION

Division of Emerging and Other Communicable Diseases Surveillance and Control

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This document was prepared by the Directors of WHO Collaborating Centres for Biosafety and other advisers.

Table of Contents

Introduction

These guidelines are applicable to the transport of infectious substances and diagnostic specimens both nationally and internationally. They provide information for identifying and classifying the material to be transported and for its safe packaging and transport. The guidelines stress the importance of developing a working relationship between the groups involved – the sender, the carrier and the receiver – in order to provide for the safe and expeditious transport of this material.

Postal, airline and other transport industry personnel hold concerns about the possibility of their becoming infected as the result of exposure to infectious microorganisms that may escape from broken, leaking or improperly packaged material. The packaging of infectious materials for transport must therefore address these concerns and be designed to minimise the potential for damage during transport. In addition, the packaging will serve to ensure the integrity of the materials and timely processing of specimens.

There are no recorded cases of illness attributable to the release of specimens during transport, although there are reported incidents of damage to the outer packaging of properly packaged materials. The shipment of unmarked and unidentified infectious materials, improperly packaged, obviously increases the overall potential for exposure to all persons.

The international regulations for the transport of infectious materials by any mode of transport are based upon the Recommendations of the United Nations Committee of Experts on the Transport of Dangerous Goods (UN). The Universal Postal Union (UPU) reflects these recommendations in its regulations, particularly for packaging. The International Civil Aviation Organization (ICAO) and the International Air Transport Association (IATA) have also incorporated the UN Recommendations in their respective regulations, as have other international transport organizations. The World Health Organization serves in an advisory capacity to these bodies. This document provides practical guidance to facilitate compliance with current international regulations. If, at a future date, any modification is made in the section of the UN Recommendations on the Transport of Dangerous Goods dealing with infectious substances and diagnostic specimens, these guidelines will be updated accordingly.

Definitions

For the purpose of describing transport safety measures the terms "infectious substances" and "infectious materials" are considered synonymous. The term "infectious substances" will be used in this document.

Infectious substances

An infectious substance is defined as a substance containing a viable microorganism, such as a bacterium, virus, rickettsia, parasite or fungus, that is known or reasonably believed to cause disease in humans or animals*.

With respect to packaging and transport situations, infectious substances include:

1. all cultures containing or suspected of containing an agent which may cause infection;

2. human or animal samples that contain such an agent in quantities sufficient to cause infection, should an exposure to them occur due to a transport mishap;

3. sample(s) from a patient with a serious disease of unknown cause;

4. other specimens not included above and designated as infectious by a qualified person, e.g. a physician, scientist, nurse, etc.

* This definition is taken from the current UN Recommendations on the Transport of Dangerous Goods. Prions are not included in this definition although they are considered to be infectious agents.

Diagnostic specimens

A diagnostic specimen is defined as any human or animal material including, but not limited to, excreta, blood and its components, tissue and tissue fluids collected for the purposes of diagnosis, but excluding live infected animals.

Diagnostic specimens resulting from medical practice and research are considered a negligible threat to the public health.

Diagnostic specimens obtained from patients with suspected infectious diseases may contain limited quantities of an infectious agent. There are very few agents which may be the source of an infection as a result of a transport mishap. *If exposure to the specimen due to transport mishap could result in an infection, the diagnostic specimen must be packaged, labelled and transported as an infectious substance.* Diagnostic specimens collected during an investigation of an outbreak of a serious disease of unknown cause must be handled as infectious substances.

Packaging, Labelling and Documentation for Transport

Because of the distinction of risks between infectious substances and diagnostic specimens, there are variations to the packaging, labelling and documentation requirements. The packaging requirements are determined by the UN and are contained in ICAO and IATA regulations in the form of Packaging Instructions (PI) 602 and 650. The requirements are subject to change and upgrade by these organisations. The current packaging requirements are described below. UN-approved packaging systems are available commercially.

Basic triple packaging system

The system consists of three layers as follows.

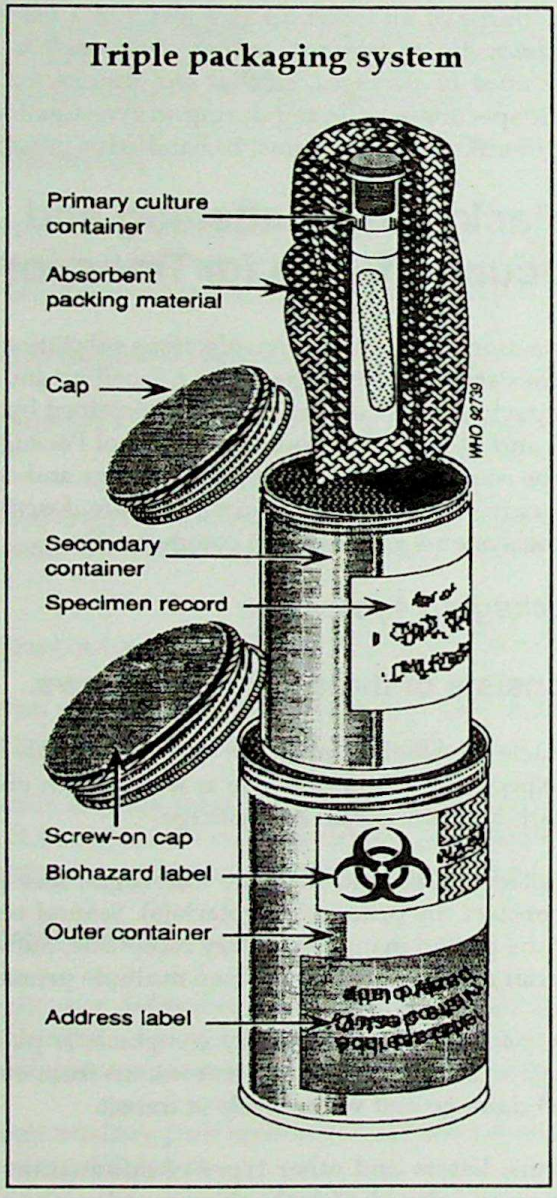
1. Primary receptacle. A labelled primary watertight, leak-proof receptacle containing the specimen. The receptacle is wrapped in enough absorbent material to absorb all fluid in case of breakage.

2. Secondary receptacle. A second durable, watertight, leak-proof receptacle to enclose and protect the primary receptacle(s). Several wrapped primary receptacles may be placed in one secondary receptacle. Sufficient additional absorbent material must be used to cushion multiple primary receptacles.

3. Outer shipping package. The secondary receptacle is placed in an outer shipping package which protects it and its contents from outside influences such as physical damage and water while in transit.

Specimen data forms, letters and other types of information that identify or describe the specimen and also identify the shipper and receiver should be taped to the outside of the secondary receptacle.

Figure 1


Triple packaging system
Primary culture container
Absorbent packing material
Cap
Secondary container
Specimen record
Screw-on cap
Biohazard label
Outer container
Address label
WHO 92739

Requirements for infectious substances

The basic triple packaging system is used with the following additional specifications and labelling and documentation requirements.

Infectious substances may only be transported in packaging which meets the UN class 6.2 specifications and packaging instruction (PI)602. This ensures that strict performance tests which include a nine metre drop test and a puncture test have been met. The outer shipping package must bear the UN Packaging Specification Marking (Figure 2). UN-approved packaging supplier listings may be obtained from carriers or from the appropriate national ministry or department, e.g. the Ministry of Transport, etc.

Figure 2

Packaging Specification Marking

example: 

4H"/Class 6.2/94
GB/2470

The packaging marking consists of:

- the United Nations packaging symbol
- type of packing
- the text "Class 6.2"
- the last two digits of the year of manufacture of the packaging
- State authority
- manufacturer's code.

Hand carriage of infectious substances is strictly prohibited by international air carriers, as is the use of diplomatic pouches for that purpose.

The maximum net quantity of infectious substances which can be contained in an outer shipping package is 50 mL or 50g if transport is by passenger aircraft. Otherwise, the limit per package is 4L-4Kg for transport by cargo aircraft or other carriers. Primary receptacles exceeding 50 mL in combination packing must be oriented so the closures are upward, and labels (arrows) indicating the "UP" direction must be placed on two opposite sides of the package. The passenger aircraft quantify limits do not apply to blood or blood products for which there is no reason to believe they contain infectious substances, when in receptacles of not more than 500 mL each and with a total volume of not more than 4L in the outer package.

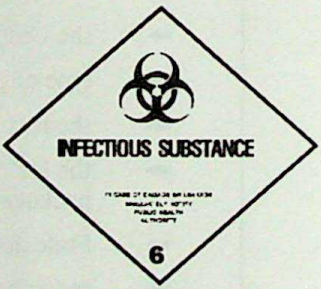
Figure 3

Hazard labels for dangerous goods

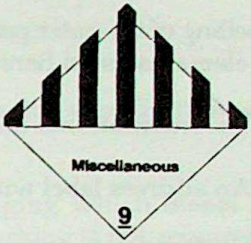
For all dangerous goods to be shipped by airfreight, specific hazard label(s) must be affixed to the outside of each package. The following hazards labels are of importance for culture collections or other institutions shipping biological substances.

Hazard labels for infectious substances and for genetically modified microorganisms which meet the IATA definition of an infectious substance:

Name:	Infectious Substance
Minimum dimensions:	100 x 100 mm
For small packages:	50 x 50 mm (black and white)



Hazard label for noninfectious genetically modified microorganisms and for carbon dioxide, solid (dry ice):

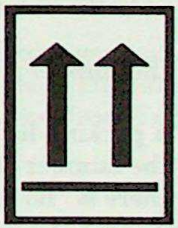


Name:	Miscellaneous
Minimum dimensions:	100 x 100 mm
For small packages:	50 x 50 mm (black and white)

Hazard label for liquid nitrogen:

Name:	Non-flammable gas
Minimum dimensions:	100 x 100 mm
For small packages:	50 x 50 mm (green and white)

Packages containing liquid cultures of infectious organisms and genetically modified microorganisms must be packed so that the closure(s) of the inner packaging(s) are upward; the upright position of the packaging must be indicated by two "Package Orientation" labels (black or red arrows). The labels must be affixed on opposite sides of the packaging. A label "THIS SIDE UP" or "THIS END UP" may also be displayed on the top cover of the package:



Name:	Package Orientation
Minimum dimensions:	74 x 105 mm (black or red and white)

For small packages of infectious substances dimensions may be halved.

In case shipments include only freeze-dried cultures the quantity should be given in g or mg. not in ml. The "PACKAGE ORIENTATION" labels should be affixed to avoid any delay.

Labelling of the outer package for shipment of infectious substances must include the elements listed hereafter.

1. The International Infectious Substance Label.

2. An address label with the following information:

   - the receiver's (consignee) name, address and telephone number
   - the shipper's (consignor) name, address and telephone number
   - the UN shipping name (Infectious Substances Affecting Humans or Animals as the case may be) followed by the scientific name of the substance
   - the UN Number (Humans – UN2814, Animals UN2900)
   - temperature storage requirements (optional).

   If the outer package is further packed in an overpack (with dry ice for instance) both outerpack and overpack must carry the above information, and the overpack must have a label stating "INNER PACKAGES COMPLY WITH PRESCRIBED SPECIFICATIONS".

3. Required shipping documents – these are obtained from the carrier and are fixed to the outer package:

   - the shipper's Declaration of Dangerous Goods (Figure 4 is one example)
   - a packing list/proforma invoice which includes the receiver's address, the number of packages, detail of contents, weight, value (note: state that there is "no commercial value" as the items are supplied free of charge)
   - An airwaybill if shipping by air.

4. An import and/or export permit and/or declaration if required.

5. If the outer package contains primary receptacles exceeding 50 mL in combination at least two "Orientation Labels" (arrows) must be placed on opposite sides of the package showing correct orientation of the package.

Requirements for diagnostic specimens

The basic triple packaging system is used with the following specifications and labelling requirements.

Diagnostic specimens may be transported in packaging which meets the packaging instruction (PI)650. The UN specification marking is not required.

Primary receptacles may contain up to 500 mL each, the total volume in the outer package not to exceed 4L.

Labelling of the outer package for the shipment of diagnostic specimens must include the following.

1. An address label with the following information:
   - the receiver's (consignee) name, address and telephone number
   - the shipper's (consignor) name, address and telephone number
   - the statement "Diagnostic Specimen, Not Restricted, Packed in Compliance with Packing Instruction 650".
2. Required shipping documents – these are obtained from the carrier and are fixed to the outer package:
   - a packing list/proforma invoice which includes the receiver's address, the number of packages, detail of contents, weight, value (note: state that there is "no commercial value" as the items are supplied free of charge)
   - an airwaybill if shipping by air.
3. An import and/or export permit and/or declaration (if required).

Note: The infectious substance label and the shipper's declaration of dangerous goods are not required for diagnostic specimens.

Requirements for Air Mail

Infectious substances and diagnostic specimens may be shipped by registered air mail. The basic triple packaging system is used with the same requirements as for other means of conveyance.

The address label must display the word "LETTRE" and the green Customs Declaration Label for Postal Mail is required for international mailing. Diagnostic specimens are to be identified with the violet UPU "PERISHABLE BIOLOGICAL SUBSTANCES" label. Infectious substances are to be identified with the International Infectious Substance label (see Figure 3). Infectious substances must also be accompanied with a shipper's Declaration of Dangerous Goods form (see Figure 4 at the end of the document).

Because of local/international restrictions, prior contact should be made with the local post office to ascertain whether the packaged material will be accepted by the postal service.

Refrigerants

Ice or dry ice when used in a shipment must be placed outside the secondary receptacle. If wet ice is used it should be in a leak-proof container and the outer package must also be leak-proof.

The secondary receptacle must be secured within the outer package to prevent damage after the refrigerant has melted or dissipated. Dry ice must **not** be placed inside the primary or secondary receptacle because of the risk of explosions. An overpack (a specially designed insulated outer package) may be used to contain dry ice. The outer package must permit the release of carbon dioxide gas if dry ice is used. UN Packing Instruction 904 must be observed.

If dry ice is used for infections substances, the details must appear on the shipper's Declaration for Dangerous Goods. In particular, the outer most packing must carry the "MISCELLANEOUS" hazard label for dry ice (see Figure 3).

If liquid nitrogen is used as a refrigerant, special arrangements must be made in advance with the carrier. Primary receptacles must be capable of withstanding extremely low temperatures and appropriate packaging requirement of the carrier must be observed. In particular, the outer most packing must carry the "NON-FLAMMABLE GAS" label for liquid nitrogen (see Figure 3).

Local Surface Transport

Examples include transport of specimens from a doctor's office/surgery to a laboratory, from a hospital to a diagnostic laboratory or from one laboratory to another. Such courier services may be operated by a hospital, a laboratory, a health service or other approved agency or organisation.

The principle of safe transport by this means is the same as for air or international transport - the material should not have any possibility of escaping from the package under normal conditions of transport.

The following practices should be observed:

1. specimen containers should be watertight and leak-proof;

2. if the specimen container is a tube, it must be tightly capped and placed in a rack to maintain it in an upright position;

3. specimen containers and racks should be placed in robust, leak-proof plastic or metal transport boxes with secure, tight fitting covers;

4. the transport box should be secured in the transport vehicle;

5. each transport box should be labelled appropriately consistent with its contents;

6. specimen data forms and identification data should accompany each transport box;

7. a spill kit containing absorbent material, a chlorine disinfectant, a leak-proof waste disposal container and heavy duty reusable gloves should be kept in the transport vehicle.

Note: The practices 1 - 7 described above are not intended to supersede local or national requirements.

Transport Planning

It is the responsibility of the sender to ensure the correct designation, packaging, labelling and documentation of all infectious substances and diagnostic specimens.

The efficient transport and transfer of infectious materials requires good coordination between the sender, the carrier and the receiver (receiving laboratory), to ensure that the material is transported safely and arrives on time and in good condition. Such coordination depends upon well-established communication and a partner relationship between the three parties.

All have specific responsibilities to carry out in the transport effort.

The sender

1. makes advance arrangements with the receiver of the specimens including investigating the need for an import permit;

2. makes advance arrangements with the carrier to ensure:

   - that the shipment will be accepted for appropriate transport

   - that the shipment (direct transport if possible) is undertaken by the most direct routing, avoiding arrival at weekends;

3. prepares necessary documentation including permits, dispatch and shipping documents;

4. notifies the receiver of transportation arrangements once these have been made, well in advance of expected arrival time.

The carrier

1. provides the sender with the necessary shipping documents and instructions for their completion;

2. provides advice to the sender about correct packaging;

3. assists the sender in arranging the most direct routing and then confirms the routing;

4. maintains and archives the documentation for shipment and transport;

5. monitors required holding conditions of the shipment while in transit;

6. notifies the sender of any anticipated (or actual) delays in transit.

The receiver

1. obtains the necessary authorisation(s) from national authorities for the importation of the material;

2. provides the sender with the required import permit(s), letter(s) of authorisation, or other document(s) required by the national authorities;

3. arranges for the most timely and efficient collection on arrival;

4. immediately acknowledges receipt to the sender.

Shipments should not be dispatched until:

- advance arrangements have been made between the sender, carrier and receiver

- the receiver has confirmed with the national authorities that the material may be legally imported

- the receiver has confirmed that there will be no delay incurred in the delivery of the package to its destination.

Detailed information on response and emergency safety measures in transport-associated accidents can be found in *Laboratory Biosafety Manual*, Second edition (1993). Geneva: World Health Organization: (pp. 52 - 54).

Figure 4A. Standard shipment of infectious substances

Shipper's Declaration for Dangerous Goods

Shipper
World Health Organization
20, avenue Appia
CH-1211 Geneva
Switzerland

Air Waybill No. 117-4812'9550

Page 1 of 1 Page

Shipper's Reference Number *(optional)*

Consignee
Karolinska Hospital
Clinical Microbiology
Stockholm 17176, Sweden
Attn: Dr Göran Kronvall
Tel: 468 51 77 4910/Fax: 468 308 099

Transport details

This shipment is within the limitations prescribed for: *(delete non-applicable)*

Passenger and Cargo Aircraft

Airport of Departure:

Airport of Destination:

Warning
Failure to comply in all respects with the applicable Dangerous Goods Regulations may be in breach of the applicable law, subject to legal penalties. This Declaration must not, in any circumstances, be completed and/or signed by a consolidator, a forwarder or an IATA cargo agent.

Shipment type: *(delete non-applicable)*

Non-Radioactive

Nature and Quantity of Dangerous Goods *(see sub-Section 8.1 of IATA Dangerous Goods Regulations)*

Dangerous Goods Identification: Proper Shipping Name	Class or Division	UN or ID No.	Packing Group	Subsidiary Risk	Quantity and type of packing	Packing Inst.	Authorization
Infectious substance, affecting humans (Streptococcus Pneumonia)	6.2	UN 2814			1 fibreboard box x 2g	602	

SPECIMEN

Additional Handling Information
Emergency contact: P Munger - Tel: 4122 791 2179
Prior arrangements as required by the IATA Dangerous Goods Regulations 1.3.3.1 have been made.

I hereby declare that the contents of this consignment are fully and accurately described above by the proper shipping name, and are classified, packaged, marked and labelled/placarded, and are in all respects in proper condition for transport according to applicable international and national governmental regulations.

Two completed and signed copies of this Declaration must be handed to the operator

Name/Title of Signatory
P Munger, Shipping and Logistics Unit

Place and Date
Geneva, 3 Jun 19[illegible]

Signature *(see warning above)*

Distribution: One copy to accompany AWB
One copy to be filed at airport of departure (with AWB-copy)

Form 3134 1995 IFAC-1VI Shipper's Declaration - 3 954

Figure 4B. Shipment of infectious substances using dry ice

Shipper's Declaration for Dangerous Goods

Shipper World Health Organization 20, avenue Appia CH-1211 Geneva Switzerland	Air Waybill No. 117-4812'9550 Page 1 of 1 Page Shipper's Reference Number *(optional)*
Consignee Karolinska Hospital Clinical Microbiology Stockholm 17176, Sweden Attn: Dr Göran Kronvall Tel: 468 51 77 4910/Fax: 468 308 099	
Transport details This shipment is within the limitations prescribed for: *(delete non-applicable)* **Passenger and Cargo Aircraft** — XXXXXX Airport of Departure: Airport of Destination:	**Warning** **Failure to comply in all respects with the applicable Dangerous Goods Regulations may be in breach of the applicable law, subject to legal penalties. This Declaration must not, in any circumstances, be completed and/or signed by a consolidator, a forwarder or an IATA cargo agent.** Shipment type: *(delete non-applicable)* Non-Radioactive — XXXXXXXX

Nature and Quantity of Dangerous Goods *(see sub-Section 8.1 of IATA Dangerous Goods Regulations)*

Dangerous Goods Identification							
Proper Shipping Name	Class or Division	UN or ID No.	Packing Group	Subsidiary Risk	Quantity and type of packing	Packing Inst.	Authorization
Infectious substance, affecting humans (Streptococcus Pneumonia)	6.2	UN 2814			1 fibreboard box x 2g	602	
Dry Ice	9	UN 1845	III		10 kg	904	
		OVERPACK USED					

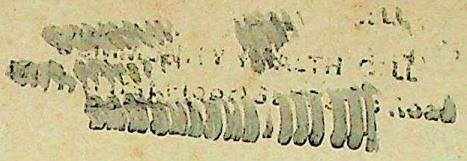
SPECIMEN

Additional Handling Information
Emergency contact: P Munger - Tel: 4122 791 2179
Prior arrangements as required by the IATA Dangerous Goods Regulations 1.3.3.1 have been made.

I hereby declare that the contents of this consignment are fully and accurately described above by the proper shipping name, and are classified, packaged, marked and labelled/placarded, and are in all respects in proper condition for transport according to applicable international and national governmental regulations. *Two completed and signed copies of this Declaration must be handed to the operator*	Name/Title of Signatory P Munger, Shipping and Logistics Unit Place and Date Geneva, 3 June 19.. Signature *(see warning above)*

Distribution: One copy to accompany AWB
One copy to be filed at airport of departure (with AWB-copy)

Form 3256 1995 IFAG VI Shipper's Declaration 2 Sig



BUREAU OF HYGIENE AND TROPICAL DISEASES

Keppel Street, London, WC1E 7HT, England

Supplement to *Tropical Diseases Bulletin* and *Abstracts on Hygiene*—
January 1972

[*superseding supplement dated January 1970.*]

It is the policy of the Bureau to provide, in the *Tropical Diseases Bulletin* and in *Abstracts on Hygiene*, a selection of abstracts of articles which appear in the world's principal medical and scientific journals and which make significant contributions to knowledge of all aspects of public health and of associated fields of microbiology, environmental conditions, nutrition, entomology and laboratory procedures. These abstracts are prepared by named specialists, who write critical comments on the articles when they appear to be justified.

Periodicals perused regularly in the Bureau for the selection of abstracts are listed herein. It will be realized from the foregoing that it depends on the nature and quality of the contents of a particular issue of a journal whether any of the articles are abstracted; in certain instances, articles which in themselves are excellent, may be excluded because they do not relate to any new developments, or for that and other reasons are recorded by title only or by use of the authors' summaries.

Titles have been brought up to date as far as possible to December, 1971. Abbreviations of titles are based on those contained in the *World List of Scientific Periodicals*, 1900–1960, Volumes 1–3 [1963–5, London: Butterworths]. Some modifications introduced in the *World List* supplements *New Periodical Titles*, and certain minor changes which appear more suitable for use in the Bulletins* have, however, been adopted.

The publications in this list are incorporated eventually in the serials collection of the library of the London School of Hygiene and Tropical Medicine. Full details of the holdings of the library, which include periodicals additional to those normally reviewed in the Bulletins, are contained in Section 7 of the *Dictionary Catalogue of the London School of Hygiene and Tropical Medicine* [1965, Boston (Mass.): G. K. Hall] and the first supplement [1971].

Other publications which are abstracted or reviewed in the Bulletins but are excluded from this list are reports of international and national medical and scientific organizations, Government departments, health authorities and other bodies; reprints of articles from journals not regularly taken; published theses; and books.

* *Tropical Diseases Bulletin* and *Abstracts on Hygiene*.

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Abstracts of Bulgarian Scientific Literature.	Sofia	*Abstr. Bulg. Scient. Lit.*
Académie Royale des Sciences d'Outre-Mer. Classe des Sciences Naturelles et Médicales.	Bruxelles	*Acad. R. Sci. Outre-Mer. Classe Sci. Naturelles Méd.*
Acarologia.	Paris	*Acarologia*
Accident Analysis and Prevention.	Oxford	*Accid. Anal. Prev.*
L'Acqua nell'Agricoltura, nell'Igiene, nell'Industria.	Roma	*Acqua Agric. Ig. Ind.*
Acta Dermato-Venereologica.	Stockholm	*Acta Derm.-Vener.*
Acta Medica. [Igaku Kenkyu.]	Fukuoka	*Acta Med.* Fukuoka
Acta Médica Costarricense.	San José	*Acta Méd. Costarric.*
Acta Medica Iranica.	Teheran	*Acta Med. Iran.*
Acta Medica Iugoslavica.	Beograd	*Acta Med. Iugosl.*
Acta Medica Philippina.	Manila	*Acta Med. Philipp.*
Acta Medica Polona.	Warszawa	*Acta Med. Pol.*
Acta Medica Scandinavica.	Stockholm	*Acta Med. Scand.*
Acta Médica Venezolana.	Caracas	*Acta Méd. Venez.*
Acta Medicinae Okayama.	Okayama	*Acta Med. Okayama*
Acta Microbiologica Academiae Scientiarum Hungaricae.	Budapest	*Acta Microbiol. Hung.*
Acta Microbiologica Hellenica.	Athens	*Acta Microbiol. Hellen.*
Acta Microbiologica Polonica, Series A and B.	Warszawa	*Acta Microbiol. Pol., Ser. A* (or *Ser. B*)
Acta Parasitologica Polonica.	Warszawa	*Acta Parasit. Pol.*
Acta Pathologica et Microbiologica Scandinavica, Section A and B.	Copenhagen	*Acta Path. Microbiol. Scand., Sect. A* (or *Sect. B*)
Acta Protozoologica.	Warszawa	*Acta Protozool.*
Acta Scholae Medicinalis Universitatis in Kioto.	Kyoto	*Acta Sch. Med. Univ. Kioto*
Acta Socio-Medica Scandinavica.	Stockholm	*Acta Socio-Med. Scand.*
Acta Tropica.	Basel	*Acta Trop.*
Acta Virologica.	Praha	*Acta Virol.*
Acta Zoologica Mexicana.	Mexico	*Acta Zool. Mex.*
Acta Zoologica et Pathologica Antverpiensia.	Antwerpen	*Acta Zool. Path. Antverp.*
Advances in Applied Microbiology.	New York	*Adv. Appl. Microbiol.*
Advances in Biological and Medical Physics.	New York	*Adv. Biol. Med. Phys.*
Advances in Carbohydrate Chemistry.	New York	*Adv. Carbohyd. Chem.*
Advances in Chemotherapy.	New York	*Adv. Chemother.*
Advances in Enzymology.	New York	*Adv. Enzymol.*
Advances in Fertility Control.	Amsterdam	*Adv. Fert. Control*
Advances in Food Research.	New York	*Adv. Fd Res.*
Advances in Genetics.	New York	*Adv. Genet.*
Advances in Immunology.	New York	*Adv. Immunol.*
Advances in Insect Physiology.	London	*Adv. Insect Physiol.*
Advances in Microbial Physiology.	London	*Adv. Microbial Physiol.*
Advances in Parasitology.	London	*Adv. Parasit.*
Advances in Pest Control Research.	New York	*Adv. Pest Control Res.*
Advances in Protein Chemistry.	New York	*Adv. Protein Chem.*
Advances in Virus Research.	New York	*Adv. Virus Res.*
Afghan Journal of Public Health.	Kabul	*Afghan J. Publ. Hlth*
African Journal of Medical Sciences.	Oxford	*Afr. J. Med. Sci.*
Africana Bulletin.	Warszawa	*Africana Bull.*
Aging.	Washington	*Aging*
Agricultural and Biological Chemistry.	Tokyo	*Agric. Biol. Chem.*
Air Pollution Control Association Abstracts.	Pittsburgh	*A.P.C.A. Abstr.*
Alexandria Medical Journal.	Alexandria	*Alexandria Med. J.*
American Industrial Hygiene Association Journal.	Chicago	*Am. Ind. Hyg. Ass. J.*
American Journal of Clinical Nutrition.	Philadelphia	*Am. J. Clin. Nutr.*
American Journal of Clinical Pathology.	Philadelphia	*Am. J. Clin. Path.*
American Journal of Diseases of Children.	Chicago	*Am. J. Dis. Child.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
American Journal of Epidemiology.	Baltimore	*Am. J. Epidem.*
American Journal of Human Genetics.	Baltimore	*Am. J. Hum. Genet.*
American Journal of Medical Sciences.	Philadelphia	*Am. J. Med. Sci.*
American Journal of Medicine.	New York	*Am. J. Med.*
American Journal of Pathology.	Boston	*Am. J. Path.*
American Journal of Physiology.	Baltimore	*Am. J. Physiol.*
American Journal of Public Health.	New York	*Am. J. Publ. Hlth*
American Journal of Sociology.	Chicago	*Am. J. Sociol.*
American Journal of Tropical Medicine and Hygiene.	Baltimore	*Am. J. Trop. Med. Hyg.*
American Review of Respiratory Disease.	Baltimore	*Am. Rev. Resp. Dis.*
Anais da Academia Brasileira de Ciencias.	Rio de Janeiro	*Anais Acad. Bras. Cienc.*
Anais da Escola Nacional de Saúde Pública e de Medicina Tropical.	Lisboa	*Anais Escola Nac. Saúde Públ. Med. Trop.*
Anais da Faculdade de Medicina de Pôrto Alegre. [Universidade do Rio Grande do Sul.]	Pôrto Alegre	*Anais Fac. Med. Pôrto Alegre*
Anais da Faculdade de Medicina da Universidade do Paraná.	Curitiba, Brasil	*Anais Fac. Med. Univ. Paraná*
Anais de Microbiologia.	Rio de Janeiro	*Anais Microbiol.*
Angewandte Parasitologie.	Jena	*Angew. Parasit.*
Annales de Dermatologie et de Syphiligraphie.	Paris	*Ann. Derm. Syph.*
Annales de l'Institut Pasteur.	Paris	*Ann. Inst. Pasteur*
Annales de l'Institut Pasteur de Lille.	Paris	*Ann. Inst. Pasteur Lille*
Annales Medicinae Experimentalis et Biologiae Fenniae.	Helsinki	*Ann. Med. Exp. Biol. Fenn.*
Annales de la Nutrition et de l'Alimentation.	Paris	*Ann. Nutr. Aliment.*
Annales de Parasitologie Humaine et Comparée.	Paris	*Ann. Parasit. Hum. Comp.*
Annales des Sociétés Belges de Médecine Tropicale, de Parasitologie et de Mycologie.	Bruxelles	*Ann. Soc. Belg. Méd. Trop.*
Annales de l'Université d'Abidjan.	Abidjan	*Ann. Univ. Abidjan*
Annali dell'Istituto Superiore di Sanità.	Roma	*Ann. Ist. Sup. Sanità*
Annali di Medicina Navale.	Roma	*Ann. Med. Nav.*
Annali della Sanità Pubblica.	Roma	*Ann. Sanità Pubbl.*
Annali Sclavo. [Rivista di Microbiologia e di Immunologia.]	Siena	*Ann. Sclavo*
Annals of Clinical Biochemistry.	London	*Ann. Clin. Biochem.*
Annals of Clinical Research.	Helsinki	*Ann. Clin. Res.*
Annals of the Entomological Society of America.	Baltimore	*Ann. Ent. Soc. Am.*
Annals of Human Genetics.	London	*Ann. Hum. Genet.*
Annals of Internal Medicine.	Lancaster, Pa	*Ann. Intern. Med.*
Annals of Life Insurance Medicine.	Zurich	*Ann. Life Insur. Med.*
Annals of Occupational Hygiene.	Oxford	*Ann. Occup. Hyg.*
Annals of Tropical Medicine and Parasitology.	Liverpool	*Ann. Trop. Med. Parasit.*
Annual of Czechoslovak Medical Literature.	Praha	*A. Czech. Med. Lit.*
Annual Review of Biochemistry.	Palo Alto, Calif.	*A. Rev. Biochem.*
Annual Review of Entomology.	Palo Alto, Calif.	*A. Rev. Ent.*
Annual Review of Genetics.	Palo Alto, Calif.	*A. Rev. Genet.*
Annual Review of Microbiology.	Palo Alto, Calif.	*A. Rev. Microbiol.*
Annual Review of Physiology.	Palo Alto, Calif.	*A. Rev. Physiol.*
Antimicrobial Agents and Chemotherapy.	Ann Arbor	*Antimicrob. Agents Chemother.*
Antonie van Leeuwenhoek.	Amsterdam	*Antonie van Leeuwenhoek*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Applied Ergonomics.	Guildford, Surrey	*Appl. Ergonom.*
Applied Microbiology.	Baltimore	*Appl. Microbiol.*
Applied Statistics.	Edinburgh	*Appl. Statist.*
Arbeitsmedizin Sozialmedizin Arbeitshygiene.	Stuttgart	*Arbmed. Sozmed. Arbhyg.*
Archiv für die Gesamte Virusforschung.	Wien	*Arch. Ges. Virusforsch.*
Archiv für Protistenkunde.	Jena	*Arch. Protistenk.*
Archives Belges de Médecine Sociale, Hygiène, Médecine du Travail et Médecine Légale.	Bruxelles	*Arch. Belg. Méd. Soc.*
Archives of Biochemistry and Biophysics.	New York	*Arch. Biochem. Biophys.*
Archives of Dermatology.	Chicago	*Arch. Derm.*
Archives of Disease in Childhood.	London	*Arch. Dis. Childh.*
Archives of Environmental Health.	Chicago	*Arch. Envir. Hlth*
Archives de l'Institut Pasteur d'Algérie.	Alger	*Arch. Inst. Pasteur Algér.*
Archives de l'Institut Pasteur Hellénique.	Athens	*Arch. Inst. Pasteur Hellén.*
Archives de l'Institut Pasteur de Madagascar.	Tananarive	*Arch. Inst. Pasteur Madagascar*
Archives de l'Institut Pasteur de Tunis.	Tunis	*Arch. Inst. Pasteur Tunis*
Archives de l'Institut Razi.	Teheran	*Arch. Inst. Razi*
Archives of Internal Medicine.	Chicago	*Arch. Intern. Med.*
Archives des Maladies Professionnelles de Médecine du Travail et de Sécurité Sociale.	Paris	*Arch. Mal. Prof. Méd. Trav.*
Archives of Pathology.	Chicago	*Arch. Path.*
Archives Roumaines de Pathologie Expérimentale et de Microbiologie.	Bucuresti	*Arch. Roum. Path. Exp. Microbiol.*
Archivio Italiano di Scienze Mediche Tropicali e di Parassitologia.	Tripoli	*Arch. Ital. Sci. Med. Trop. Parassit.*
Arquivos Brasileiros de Nutrição.	Rio de Janeiro	*Arq. Bras. Nutr.*
Archivos Dominicanos de Pediatria.	Santo Domingo	*Arch. Dom. Pediat.*
Arquivos da Escola de Veterinária da Universidade Federal de Minas Gerais.	Belo Horizonte	*Arq. Escola Vet. Univ. Fed. Minas Gerais*
Arquivos de Gastroenterologia.	São Paulo	*Arq. Gastroenterol.*
Arquivos de Higiene e Saúde Pública.	São Paulo	*Arq. Hig. Saúde Públ.*
Archivos del Hospital Vargas.	Caracas	*Arch. Hosp. Vargas*
Archivos Latinoamericanos de Nutricion.	Caracas	*Arch. Lat.-am. Nutr.*
Archivum Immunologiae et Therapiae Experimentalis.	Warszawa	*Arch. Immun. Ther. Exp.*
Armed Forces Medical Journal India.	New Delhi	*Armed Forces Med. J. India*
Assignment Children.	Neuilly-sur-Seine	*Assignment Child.*
Atmospheric Environment.	Oxford	*Atmos. Envir.*
Australian Journal of Biological Sciences.	Melbourne	*Aust. J. Biol. Sci.*
Australian Journal of Experimental Biology and Medical Science.	Adelaide	*Aust. J. Exp. Biol. Med. Sci.*
Australian Journal of Zoology.	Melbourne	*Aust. J. Zool.*
Australian and New Zealand Journal of Medicine.	Sydney	*Aust. N.Z. J. Med.*
Australian Veterinary Journal.	Sydney	*Aust. Vet. J.*
Bacteriological Proceedings.	Ann Arbor	*Bact. Proc.*
Bacteriological Reviews.	Baltimore	*Bact. Rev.*
Baltimore Health News.	Baltimore	*Baltimore Hlth News*
Bëilage zu den Beiträgen zur Silikose-Forschung (Pneumokoniose).	Bochum	*Bëil. Silikosforsch. (Pneumokon.)*
Beiträge zur Silikose-Forschung (Pneumokoniose).	Bochum	*Beitr. Silikosforsch. (Pneumokon.)*
Berufsdermatosen.	Aulendorf	*Berufsdermatosen*
Biken Journal.	Osaka	*Biken J.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Biochemical Genetics.	New York	*Biochem. Genet.*
Biochemical Journal.	London	*Biochem. J.*
Biochemical Medicine.	Baltimore	*Biochem. Med.*
Biochemistry.	Easton, Pa	*Biochemistry*
Biochimica et Biophysica Acta.	Amsterdam	*Biochim. Biophys. Acta*
Biochimie.	Paris	*Biochimie*
Biological Reviews.	Cambridge	*Biol. Rev.*
Biophysical Journal.	New York	*Biophys. J.*
Blood.	Baltimore	*Blood*
Boletim da Campanha de Erradicação da Variola.	Rio de Janeiro	*Bolm Camp. Errad. Variola*
Boletim do Centro de Estudos do Hospital dos Servidores Estado.	Rio de Janeiro	*Bolm Cent. Estud. Hosp. Serv. Estado*
Boletim Clínico e de Estatístico Hospital do Ultramar.	Lisboa	*Bolm Clin. Estat. Hosp. Ultramar*
Boletín de la Asociación Médica de Puerto Rico.	San Juan	*Bol. Asoc. Méd. P. Rico*
Boletín Chileno de Parasitología.	Santiago de Chile	*Bol. Chil. Parasit.*
Boletin Dermatologico Sanitario.	Caracas	*Bol. Derm. Sanit.*
Boletín de Higiene y Epidemiología.	Habana	*Bol. Hig. Epidem.* Havana
Boletín Informativo de la Dirección de Malariología y Saneamiento Ambiental.	Maracay	*Bol. Inf. Dir. Malariol. Saneam. Ambient.*
Boletín de la Oficina Sanitaria Panamericana.	Washington	*Bol. Of. Sanit. Pan-am.*
Bollettino dell'Istituto Sieroterapico Milanese.	Milano	*Boll. Ist. Sieroter. Milan.*
Bordeaux Médical.	Bordeaux	*Bordeaux Méd.*
Brasil-Médico.	Rio de Janeiro	*Bras.-Méd.*
British Dental Journal.	London	*Br. Dent. J.*
British Hospital Journal and Social Service Review.	London	*Br. Hosp. J. Social Serv. Rev.*
British Journal of Addiction.	London	*Br. J. Addict.*
British Journal of Cancer.	London	*Br. J. Cancer*
British Journal of Clinical Practice.	London	*Br. J. Clin. Practice*
British Journal of Dermatology.	London	*Br. J. Derm.*
British Journal of Diseases of the Chest.	London	*Br. J. Dis. Chest*
British Journal of Experimental Pathology.	London	*Br. J. Exp. Path.*
British Journal of Haematology.	Oxford	*Br. J. Haemat.*
British Journal of Hospital Medicine.	London	*Br. J. Hosp. Med.*
British Journal of Industrial Medicine.	London	*Br. J. Ind. Med.*
British Journal of Mathematical and Statistical Psychology.	London	*Br. J. Math. Statist. Psychol.*
British Journal of Medical Education.	London	*Br. J. Med. Educ.*
British Journal of Nutrition.	London	*Br. J. Nutr.*
British Journal of Pharmacology.	London	*Br. J. Pharmacol.*
British Journal of Preventive and Social Medicine.	London	*Br. J. Prev. Social Med.*
British Journal of Psychiatry.	London	*Br. J. Psychiat.*
British Journal of Social Work.	London	*Br. J. Social Wk*
British Journal of Sociology.	London	*Br. J. Sociol.*
British Journal of Venereal Diseases.	London	*Br. J. Vener. Dis.*
British Medical Bulletin.	London	*Br. Med. Bull.*
British Medical Journal.	London	*Br. Med. J.*
British Nutrition Foundation Information Bulletin.	London	*Br. Nutr. Found. Inf. Bull.*
British Standards Institution News.	London	*B.S.I. News*
Bruxelles-Médical.	Bruxelles	*Brux.-Méd.*
B.T.T.A. Review. [British Thoracic and Tuberculosis Association.]	London	*BTTA Rev.*
Bulletin de l'Académie Nationale de Médecine.	Paris	*Bull. Acad. Natn. Méd.*
Bulletin de l'Académie Polonaise des Sciences. Série des Sciences Biologiques.	Warszawa	*Bull. Acad. Pol. Sci. Sér. Sci. Biol.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Bulletin of the Calcutta School of Tropical Medicine.	Calcutta	*Bull. Calcutta Sch. Trop. Med.*
Bulletin of the Chest Disease Research Institute, Kyoto University.	Kyoto	*Bull. Chest Dis. Res. Inst. Kyoto Univ.*
Bulletin of Endemic Diseases.	Baghdad	*Bull. Endem. Dis.*
Bulletin of Entomological Research.	London	*Bull. Ent. Res.*
Bulletin of Epizootic Diseases of Africa.	Muguga	*Bull. Epizoot. Dis. Afr.*
Bulletin of the History of Medicine.	Baltimore	*Bull. Hist. Med.*
Bulletin d'Hygiène. Health Bulletin.	Montreal	*Bull. Hyg.* Montreal
Bulletin de l'Institut Fondamental d'Afrique Noire, Series A and B.	Dakar	*Bull. Inst. Fond. Afr. Noire, Ser. A* (or *Ser. B*)
Bulletin de l'Institut National de la Santé et de la Recherche Médicale.	Paris	*Bull. Inst. Natn. Santé Rech. Méd.*
Bulletin de l'Institut Pasteur.	Paris	*Bull. Inst. Pasteur*
Bulletin of the Institute of Marine Medicine in Gdańsk.	Gdańsk	*Bull. Inst. Mar. Med. Gdańsk*
Bulletin of the Institute for Medical Research, Federation of Malaya.	Kuala Lumpur	*Bull. Inst. Med. Res. Fed. Malaya*
Bulletin of the Institute of Public Health.	Tokyo	*Bull. Inst. Publ. Hlth.* Tokyo
Bulletin of the Medical Library Association.	Cleveland	*Bull. Med. Libr. Ass.*
Bulletin Mensuel de Statistiques Sociales.	Paris	*Bull. Mens. Statist. Sociales*
Bulletin du Ministere de la Santé Publique et de la Famille.	Bruxelles	*Bull. Minist. Santé Publ. Famille*
Bulletin on Narcotics.	Geneva	*Bull. Narcot.*
Bulletin of National Institute of Hygienic Sciences.	Tokyo	*Bull. Natn. Inst. Hyg. Sci.* Tokyo
Bulletin of the New York Academy of Medicine.	New York	*Bull. N.Y. Acad. Med.*
Bulletin of the Ophthalmological Society of Egypt.	Cairo	*Bull. Ophthal. Soc. Egypt*
Bulletin of the Osaka Medical School.	Osaka	*Bull. Osaka Med. Sch.*
Bulletin. Ross Institute Information and Advisory Service.	London	*Bull. Ross Inst. Inf. Advis. Serv.*
Bulletin der Schweizerischen Akademie der Medizinischen Wissenschaften.	Basel	*Bull. Schweiz. Akad. Med. Wiss.*
Bulletin Scientifique. Conseil des Académies de la RPF de Yougoslavie, Section A.	Zagreb	*Bull. Sci. Cons. Acads RPF Yougosl., Sect. A*
Bulletin des Séances. Académie Royale des Sciences d'Outre-Mer.	Bruxelles	*Bull. Séanc. Acad. R. Sci. Outre-Mer*
Bulletin de la Société Française de Dermatologie et de Syphiligraphie.	Paris	*Bull. Soc. Fr. Derm. Syph.*
Bulletin de la Société Médicale d'Afrique Noire de Langue Française.	Dakar	*Bull. Soc. Méd. Afr. Noire Lang. Fr.*
Bulletin de la Société de Pathologie Exotique.	Paris	*Bull. Soc. Path. Exot.*
Bulletin de la Société Scientifique d'Hygiène Alimentaire. [L'Alimentation et la Vie.]	Paris	*Bull. Soc. Scient. Hyg. Aliment.*
Bulletin of the Tokyo Medical and Dental University.	Tokyo	*Bull. Tokyo Med. Dent. Univ.*
Bulletin of the World Health Organization *see* World Health Organization publications		
Bulletin of the Yamaguchi Medical School.	Ube	*Bull. Yamaguchi Med. Sch.*
Bulletin of Zoological Nomenclature.	London	*Bull. Zool. Nom.*
Cahiers d'Office de la Recherche Scientifique et Technique Outre-Mer. Entomologie Médicale et Parasitologie.	Paris	*Cah. O.R.S.T.O.M. Ent. Méd. Parasit.*
Cahiers de Sociologie et de Démographie Médicales.	Paris	*Cah. Sociol. Démog. Méd.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
California Vector Views.	Berkeley	*Calif. Vector Views*
Canadian Journal of Biochemistry.	Ottawa	*Can. J. Biochem.*
Canadian Journal of Microbiology.	Ottawa	*Can. J. Microbiol.*
Canadian Journal of Public Health.	Toronto	*Can. J. Publ. Hlth*
Canadian Journal of Zoology.	Ottawa	*Can. J. Zool.*
Canadian Medical Association Journal.	Toronto	*Can. Med. Ass. J.*
Cardiovascular Research Center Bulletin.	Houston	*Cardiovasc. Res. Cent. Bull.*
Časopis Lékařů Českých.	Praha	*Čas. Lék. Česk.*
CCA Journal on Alcoholism. [Camberwell Council on Alcoholism.]	London	*CCA J. Alcohol.*
Cellular Immunology.	New York	*Cellular Immunol.*
Center for Disease Control Surveillance: Diphtheria; Foodborne Outbreaks; Hepatitis; Influenza-Respiratory Disease; Malaria; Measles; Neurotropic Viral Diseases; Rubella; Shigella; Tetanus; Trichinosis; Zoonoses.	Atlanta, Ga	*CDC* [*Diphth.*] *Surv.*, etc.
Central African Journal of Medicine.	Salisbury, Rhodesia	*Cent. Afr. J. Med.*
Československá Epidemiologie, Mikrobiologie, Imunologie.	Praha	*Čslká Epidem. Mikrobiol. Imunol.*
Československá Hygiena.	Praha	*Čslká Hyg.*
Ceylon Journal of Medical Science.	Colombo	*Ceylon J. Med. Sci.*
Ceylon Medical Journal.	Colombo	*Ceylon Med. J.*
Ceylon Veterinary Journal.	Peradeniya	*Ceylon Vet. J.*
Chemical Reviews.	Baltimore	*Chem. Rev.*
Chicago Medical School Quarterly.	Chicago	*Chicago Med. Sch. Q.*
Children in the Tropics.	Dakar	*Childn Trop.*
Chinese Journal of Microbiology.	Taipei	*Chinese J. Microbiol.*
Clean Air.	Brighton	*Clean Air*
Clinical and Experimental Immunology.	Oxford	*Clin. Exp. Immunol.*
Clinical Pharmacology and Therapeutics.	St. Louis	*Clin. Pharmacol. Ther.*
Clinical Science.	London	*Clin. Sci.*
Clinical Toxicology.	New York	*Clin. Toxicol.*
Community Health.	London	*Community Hlth*
Community Medicine.	London	*Community Med.*
Comptes Rendus des Séances de la Société de Biologie.	Paris	*C.R. Séanc. Soc. Biol.*
Computers in Biology and Medicine.	Oxford	*Comput. Biol. Med.*
Computers and Biomedical Research.	New York	*Comput. Biomed. Res.*
Country Profiles.	New York	*Country Profiles*
Current Literature on Venereal Disease.	Atlanta	*Curr. Lit. Ven. Dis.*
Current Publications in Population/Family Planning.	New York	*Curr. Publs Popul./Family Plann.*
Current Therapeutic Research.	New York	*Curr. Ther. Res.*
Current Topics in Microbiology and Immunology.	Berlin	*Curr. Topics Microbiol. Immunol.*
Cytobios.	Cambridge	*Cytobios*
Dairy Science Abstracts.	Shinfield	*Dairy Sci. Abstr.*
Danish Medical Bulletin.	Copenhagen	*Dan. Med. Bull.*
Dar es Salaam Medical Journal.	Dar es Salaam	*Dar es Salaam Med. J.*
Demography.	Chicago	*Demography*
Dermatologia. [Revista Mexicana.]	Mexico	*Dermatologia.* Mexico
Dermatología Venezolana.	Caracas	*Derm. Venez.*
Deutsche Medizinische Wochenschrift.	Stuttgart	*Dt. Med. Wschr.*
Developmental Medicine and Child Neurology.	Tadworth	*Devl Med. Child Neur*
Diabetes.	New York	*Diabetes*
Drugs & Society.	London	*Drugs & Society*
East African Medical Journal.	Nairobi	*E. Afr. Med. J.*
Egészségtudomány.	Budapest	*Egészségtudomány*
Endeavour.	London	*Endeavour*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Endocrinology.	Glendale, Calif.	*Endocrinology*
Entomologia Experimentalis et Applicata.	Amsterdam	*Entomologia Exp. Appl.*
Entomological Review. [Translation of Entomologischeskoye Obozreniye.]	Washington	*Ent. Rev.*
Environmental Health.	London	*Envir. Hlth*
Environmental Research.	New York	*Envir. Res.*
Enzymologia.	Den Haag	*Enzymologia*
Ergebnisse der Mikrobiologie und Immunitätsforschung *see* Current Topics in Microbiology and Immunology.		
Ergonomics.	London	*Ergonomics*
Ethiopian Medical Journal.	Addis Ababa	*Ethiop. Med. J.*
European Journal of Biochemistry.	Berlin	*European J. Biochem.*
European Journal of Clinical Investigation.	New York	*European J. Clin. Invest.*
Excerpta Medica: Public Health, Social Medicine and Hygiene.	Amsterdam	*Excerpta Med., Publ. Hlth*
Experientia.	Basel	*Experientia*
Experimental Cell Research.	New York	*Expl Cell Res.*
Experimental Parasitology.	New York	*Expl Parasit.*
Family Physician.	Tel-Aviv	*Family Physn*
Family Planning.	London	*Family Plann.*
Federation Proceedings. Federation of American Societies for Experimental Biology.	Washington	*Fedn Proc. Fedn Am. Socs Exp. Biol.*
Folia Facultatis Medicae Universitatis Comenianae Bratislaviensis.	Bratislava	*Folia Fac. Med. Univ. Comenianae Bratisl.*
Folia Microbiologica.	Praha	*Folia Microbiol.* Praha
Folia Parasitologica.	Praha	*Folia Parasit.*
Foreign Agriculture.	Washington	*Foreign Agric.*
Fukushima Journal of Medical Science.	Fukushima	*Fukushima J. Med. Sci.*
Gaceta Médica de Caracas.	Caracas	*Gac. Méd. Caracas*
Gastroenterology.	Baltimore	*Gastroenterology*
Gazeta Médica da Bahia.	Bahia	*Gaz. Méd. Bahia*
Gazette of the Institute of Medical Laboratory Technology.	London	*Gaz. Inst. Med. Lab. Technol.*
G.E.N. [Sociedad Venezolana de Gastroenterología.]	Caracas	*G.E.N.*
Genetical Research.	Cambridge	*Genet. Res.*
Genetics.	Austin, Texas	*Genetics*
German Medicine.	Stuttgart	*Germ. Med.*
Gesundheitswesen und Desinfektion.	Hannover	*Gesundhwes. Desinfekt.*
Ghana Medical Journal.	Accra	*Ghana Med. J.*
Gigiena i Sanitariya.	Moskva	*Gig. Sanit.*
Gigiena Truda i Professional'nye Zabolevaniya.	Moskva	*Gig. Truda Prof. Zabol.*
Giornale di Batteriologia, Virologia ed Immunologia.	Torino	*Gior. Batt. Virol. Immunol.*
Giornale di Igiene e Medicina Preventiva.	Genova	*Gior. Ig. Med. Prev.*
Giornale Italiano di Dermatologia: Minerva Dermatologica.	Torino	*Gior. Ital. Derm. Minerva Derm.*
Giornale di Malattie Infettive e Parassitarie.	Torino	*Gior. Mal. Infett. Parassit.*
Glasnik Zavoda za Zdravstvenu Zaštitu SRS.	Beograd	*Glasn. Zav. Zdrav. Zašt. SRS.*
Glaxo Volume.	Greenford	*Glaxo Vol.*
Godishen Zbornik na Meditsinskiot Fakultet vo Skopje.	Skopje	*God. Zb. Med. Fak. Skopje*
Good Health for South Australia.	Adelaide	*Good Hlth S. Aust.*
Greater London Research Quarterly Bulletin of the Research and Intelligence Unit.	London	*Greater London Res. Q. Bull.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Gunma Journal of Medical Sciences.	Maebashi	*Gunma J. Med. Sci.*
Gut.	London	*Gut*
Hamdard Medical Digest.	Karachi	*Hamdard Med. Dig.*
Harefuah. [Journal of the Israel Medical Association.]	Jerusalem	*Harefuah*
Harvard Public Health Alumni Bulletin.	Boston	*Harv. Publ. Hlth Alumni Bull.*
Hawaii Medical Journal.	Honolulu	*Hawaii Med. J.*
Health.	Canberra	*Health.* Canberra
Health.	London	*Health.* London
Health in Bristol.	Bristol	*Hlth Bristol*
Health Bulletin.	Edinburgh	*Hlth Bull.* Edinburgh
Health Bulletin. Department of Health, Victoria.	Melbourne	*Hlth Bull. Dep. Hlth Vict.*
Health Education Journal.	London	*Hlth Educ. J.*
Health Information Digest. [Central Council for Health Education.]	London	*Hlth Inf. Dig.*
Health Laboratory Science.	New York	*Hlth Lab. Sci.*
Health Physics.	Oxford	*Hlth Phys.*
Health Services Research.	Chicago	*Hlth Servs Res.*
Health Trends.	London	*Hlth Trends*
Health Visitor.	London	*Hlth Visitor*
Heart.	London	*Heart*
Helminthologia.	Bratislava	*Helminthologia*
Helminthological Abstracts, Series A—Animal Helminthology.	St. Albans	*Helminth. Abstr., Ser. A*
Heredity. [An International Journal of Genetics.]	London	*Heredity.* London
Histochemical Journal.	London	*Histochem. J.*
Hospital	Rio de Janeiro	*Hospital.* Rio de Janeiro
HSMHA Health Reports. [Health Services and Mental Health Administration.]	Rockville, Md	*HSMHA Hlth Rep.*
Human Biology.	Detroit	*Hum. Biol.*
Humangenetik.	Berlin	*Humangenetik*
Human Pathology.	Philadelphia	*Human Path.*
Igaku Kenkyu *see* Acta Medica.		
Igiena. [Revista a Societătii de Igiena şi Sanatate Publica.]	Bucuresti	*Igiena*
Igiene Moderna.	Parma	*Ig. Mod.*
Igiene e Sanità Pubblica.	Roma	*Ig. Sanità Pubbl.*
Illinois Biological Monographs.	Urbana	*Illinois Biol. Monogr.*
Immunochemistry.	Oxford	*Immunochemistry*
Immunology.	Oxford	*Immunology*
Index-Catalogue of Medical and Veterinary Zoology.	Washington	*Index-Cat. Med. Vet. Zool.*
Indian Journal of Biochemistry.	New Delhi	*Indian J. Biochem.*
Indian Journal of Experimental Biology.	New Delhi	*Indian J. Exp. Biol.*
Indian Journal of Helminthology.	Lucknow	*Indian J. Helminth.*
Indian Journal of Industrial Medicine.	Calcutta	*Indian J. Ind. Med.*
Indian Journal of Medical Education.	Vellore	*Indian J. Med. Educ.*
Indian Journal of Medical Research.	New Delhi	*Indian J. Med. Res.*
Indian Journal of Medical Sciences.	Bombay	*Indian J. Med. Sci.*
Indian Journal of Public Health.	Calcutta	*Indian J. Publ. Hlth*
Indian Journal of Tuberculosis.	New Delhi	*Indian J. Tuberc.*
Indian Medical Forum.	Calcutta	*Indian Med. Forum*
Industrial Health.	Kawasaki	*Ind. Hlth.* Kawasaki
Industrial Hygiene Review.	New York	*Ind. Hyg. Rev.*
Industrial Medicine and Surgery.	Sheboygan, Wis.	*Ind. Med. Surg.*
Industrial Safety and Health Bulletin.	New Delhi	*Ind. Saf. Hlth Bull.*
Infection and Immunity.	Baltimore	*Infection & Immunity.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
International Archives of Allergy and Applied Immunology.	Basel	*Int. Arch. Allergy Appl. Immunol.*
International Atomic Energy Agency Bulletin.	Wien	*Int. Atom. Energy Ag. Bull.*
International Digest of Health Legislation.	Geneva	*Int. Dig. Hlth Legisl.*
International Journal of Dermatology.	Philadelphia	*Int. J. Derm.*
International Journal of Environmental Studies.	London	*Int. J. Envir. Stud.*
International Journal of Health Education.	Geneva	*Int. J. Hlth Educ.*
International Journal of Health Services.	Westport, Conn.	*Int. J. Hlth Serv.*
International Journal of Leprosy.	Washington	*Int. J. Lepr.*
International Journal for Parasitology.	Oxford	*Int. J. Parasit.*
International Journal of Protein Research.	Copenhagen	*Int. J. Protein Res.*
International Journal of Social Psychiatry.	London	*Int. J. Social Psychiat.*
International Journal of Systematic Bacteriology.	Ames, Iowa	*Int. J. Syst. Bact.*
International Journal for Vitamin and Nutrition Research.	Bern	*Int. J. Vitamin Nutr. Res.*
International Labour Office Panorama.	Geneva	*ILO Panorama*
International Labour Review.	Geneva	*Int. Labour Rev.*
International Planned Parenthood News.	London	*IPP News*
International Review of Cytology.	New York	*Int. Rev. Cytol.*
International Review of Experimental Pathology.	New York	*Int. Rev. Exp. Path.*
International Social Security Review.	Geneva	*Int. Social Secur. Rev.*
Internationale Zeitschrift für Angewandte Physiologie Einschliesslich Arbeitsphysiologie.	Berlin	*Int. Ztschr. Angew. Physiol.*
Internationales Archiv für Arbeitsmedizin.	Berlin	*Int. Arch. Arbmed.*
IPPF Medical Bulletin. [International Planned Parenthood Federation.]	London	*IPPF Med. Bull.*
Irish Journal of Medical Science.	Dublin	*Ir. J. Med. Sci.*
Israel Journal of Medical Sciences.	Jerusalem	*Israel J. Med. Sci.*
Izvestiya na Mikrobiologischeskiya Institut.	Sofia	*Izv. Mikrobiol. Inst.* Sofia
Japanese Journal of Bacteriology.	Tokyo	*Jap. J. Bact.*
Japanese Journal of Experimental Medicine.	Tokyo	*Jap. J. Exp. Med.*
Japanese Journal of Industrial Health.	Tokyo	*Jap. J. Ind. Hlth*
Japanese Journal of Medical Science and Biology.	Tokyo	*Jap. J. Med. Sci. Biol.*
Japanese Journal of Microbiology.	Tokyo	*Jap. J. Microbiol.*
Japanese Journal of Parasitology.	Tokyo	*Jap. J. Parasit.*
Japanese Journal of Tuberculosis and Chest Diseases.	Tokyo	*Jap. J. Tuberc. Chest Dis.*
Japanese Journal of Veterinary Science.	Tokyo	*Jap. J. Vet. Sci.*
Jikeikai Medical Journal.	Tokyo	*Jikeikai Med. J.*
Jordan Medical Journal.	Amman	*Jordan Med. J.*
Journal of the Air Pollution Control Association.	Pittsburgh	*J. Air Pollut. Control Ass.*
Journal of Alcoholism.	London	*J. Alcohol.*
Journal of the American Dietetic Association.	Chicago	*J. Am. Diet. Ass.*
Journal of the American Medical Association.	Chicago	*J. Am. Med. Ass.*
Journal of the American Statistical Association.	Boston	*J. Am. Statist. Ass.*
Journal of the American Water Works Association.	Lancaster, Pa	*J. Am. Wat. Wks Ass.*
Journal of Animal Ecology.	Oxford	*J. Anim. Ecol.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Journal of Applied Bacteriology.	Reading	*J. Appl. Bact.*
Journal of Applied Physiology.	Washington	*J. Appl. Physiol.*
Journal of the Association of Physicians of India.	Bombay	*J. Ass. Physns India*
Journal of Bacteriology.	Baltimore	*J. Bact.*
Journal of Biological Chemistry.	Baltimore	*J. Biol. Chem.*
Journal of Biosocial Science.	London	*J. Biosoc. Sci.*
Journal of Cell Biology.	New York	*J. Cell Biol.*
Journal of Cell Science.	London	*J. Cell Sci.*
Journal of Chromatographic Science.	Evanston, Ill.	*J. Chromat. Sci.*
Journal of Chromatography.	Amsterdam	*J. Chromat.*
Journal of Chronic Diseases.	Oxford	*J. Chron. Dis.*
Journal of Clinical Investigation.	Boston	*J. Clin. Invest.*
Journal of Clinical Pathology.	London	*J. Clin. Path.*
Journal of Communicable Diseases.	Delhi	*J. Com. Dis.*
Journal of Economic Entomology.	Baltimore	*J. Econ. Ent.*
Journal of the Egyptian Medical Association.	Cairo	*J. Egypt. Med. Ass.*
Journal of the Egyptian Public Health Association.	Cairo	*J. Egypt. Publ. Hlth Ass.*
Journal of Endocrinology.	London	*J. Endocr.*
Journal of Entomology, Series A and B.	London	*J. Ent., Ser. A* (or *Ser. B*)
Journal of Experimental Biology.	Cambridge	*J. Exp. Biol.*
Journal of Experimental Medicine.	New York	*J. Exp. Med.*
Journal of the Faculty of Medicine.	Baghdad	*J. Fac. Med.* Baghdad
Journal of Food Science.	Chicago	*J. Fd Sci.*
Journal of the Formosan Medical Association.	Taipei	*J. Formosan Med. Ass.*
Journal of General Microbiology.	Cambridge	*J. Gen. Microbiol.*
Journal of General Physiology.	Baltimore	*J. Gen. Physiol.*
Journal of General Virology.	London	*J. Gen. Virol.*
Journal of Health and Social Behavior.	Albany	*J. Hlth Social Behav.*
Journal of Helminthology.	London	*J. Helminth.*
Journal of Histochemistry and Cytochemistry.	Baltimore	*J. Histochem. Cytochem.*
Journal of Hygiene.	Cambridge	*J. Hyg.* Cambridge
Journal of Hygiene, Epidemiology, Microbiology and Immunology.	Praha	*J. Hyg. Epidem. Microbiol. Immunol.*
Journal of Immunology.	Baltimore	*J. Immunol.*
Journal of the Indian Institute of Science.	Bangalore	*J. Indian Inst. Sci.*
Journal of the Indian Medical Association.	Calcutta	*J. Indian Med. Ass.*
Journal of Infectious Diseases.	Chicago	*J. Infect. Dis.*
Journal of Insect Physiology.	London	*J. Insect Physiol.*
Journal of the Institute of Actuaries.	London	*J. Inst. Actuaries*
Journal of Invertebrate Pathology.	New York	*J. Invertebr. Path.*
Journal of Investigative Dermatology.	Baltimore	*J. Invest. Derm.*
Journal of the Japan Dental Association.	Tokyo	*J. Japan Dent. Ass.*
Journal of the Kuwait Medical Association.	Kuwait	*J. Kuwait Med. Ass.*
Journal of Laboratory and Clinical Medicine.	St. Louis	*J. Lab. Clin. Med.*
Journal de Médecine de Montpellier.	Montpellier	*J. Méd. Montpellier*
Journal of the Medical Association of Thailand.	Dhouburi	*J. Med. Ass. Thailand*
Journal of Medical Education.	Chicago	*J. Med. Educ.*
Journal of Medical Entomology.	Honolulu	*J. Med. Ent.*
Journal of Medical Genetics.	London	*J. Med. Genet.*
Journal Médical Libanais.	Beyrouth	*J. Méd. Liban.*
Journal of Medical Microbiology.	Edinburgh	*J. Med. Microbiol.*
Journal of Medicinal Chemistry.	Easton, Pa	*J. Mednl Chem.*
Journal of Molecular Biology.	London	*J. Molec. Biol.*
Journal of the Nepal Medical Association.	Kathmandu	*J. Nepal Med. Ass.*
Journal of Neurochemistry.	Oxford	*J. Neurochem.*
Journal of Nutrition.	Philadelphia	*J. Nutr.*
Journal of Occupational Medicine.	Baltimore	*J. Occup. Med.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Journal of the Pakistan Medical Association.	Karachi	*J. Pakistan Med. Ass.*
Journal of Parasitology.	Lawrence, Kansas	*J. Parasit.*
Journal of Pathology.	Edinburgh	*J. Path.*
Journal of Pediatrics.	St. Louis	*J. Pediat.*
Journal of Pharmacy and Pharmacology.	London	*J. Pharm. Pharmac.*
Journal of the Philippine Medical Association.	Manila	*J. Philipp. Med. Ass.*
Journal of Physiology.	London	*J. Physiol.* London
Journal of Postgraduate Medicine.	Bombay	*J. Postgrad. Med.*
Journal of Protozoology.	New York	*J. Protozool.*
Journal of the Royal Army Medical Corps.	London	*J. R. Army Med. Cps*
Journal of the Royal Army Veterinary Corps.	London	*J. R. Army Vet. Cps*
Journal of the Royal College of General Practitioners.	London	*J. R. Coll. Gen. Practnrs*
Journal of the Royal College of Physicians of London.	London	*J. R. Coll. Physns Lond.*
Journal of the Royal Naval Medical Service.	Gosport	*J. R. Nav. Med. Serv.*
Journal of the Royal Statistical Society, Series A and B.	London	*J. R. Statist. Soc., Ser. A* (or *Ser. B*)
Journal of Science of Labour.	Tokyo	*J. Sci. Labour*
Journal of the Society of Health of Nigeria.	Lagos	*J. Soc. Hlth Nigeria*
Journal of Theoretical Biology.	London	*J. Theoret. Biol.*
Journal of Tropical Medicine and Hygiene.	London	*J. Trop. Med. Hyg.*
Journal of Tropical Pediatrics and Environmental Child Health.	London	*J. Trop. Pediat. Envir. Child Hlth*
Journal of Ultrastructure Research.	New York	*J. Ultrastruct. Res.*
Journal of Virology.	Baltimore	*J. Virol.*
Journal of Zoology.	London	*J. Zool.*
Kasmera.	Maracaibo	*Kasmera*
Keio Journal of Medicine.	Tokyo	*Keio J. Med.*
Kobe Journal of Medical Sciences.	Kobe	*Kobe J. Med. Sci.*
Korean Journal of Parasitology.	Seoul	*Korean J. Parasit.*
Korean Journal of Public Health.	Seoul	*Korean J. Publ. Hlth*
Kumamoto Medical Journal.	Kumamoto	*Kumamoto Med. J.*
Kurume Medical Journal.	Kurume-Shi	*Kurume Med. J.*
Laboratory Practice.	London	*Lab. Pract.*
Lancet.	London	*Lancet*
Lepro.	Osaka	*Lepro*
Leprosy in India.	New Delhi	*Lepr. India*
Leprosy Review.	London	*Lepr. Rev.*
Lotta contro la Tubercolosi.	Roma	*Lotta Tuberc.*
Malawi Medical Bulletin.	Blantyre	*Malawi Med. Bull.*
Maroc Médical.	Casablanca	*Maroc Méd.*
Maternal and Child Care.	London	*Maternal Child Care*
Mathematical Biosciences.	New York	*Mathl Biosci.*
Mayo Clinic Proceedings.	Rochester, Minn.	*Mayo Clin. Proc.*
Médecine d'Afrique Noire.	Dakar	*Méd. Afr. Noire*
Médecine Tropicale.	Marseille	*Méd. Trop.*
Medical and Biological Illustration.	London	*Med. Biol. Illust.*
Medical Bulletin. Standard Oil Company.	New Jersey	*Med. Bull. Standard Oil Co.*
Medical Care.	Philadelphia	*Med. Care*
Medical History.	London	*Med. Hist.*
Medical Journal of Australia.	Sydney	*Med. J. Aust.*

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Medical Journal of Malaya.	Singapore	*Med. J. Malaya*
Medical Journal of Mutual Aid Association.	Tokyo	*Med. J. Mut. Aid Ass.*
Medical Journal of Osaka University.	Osaka	*Med. J. Osaka Univ.*
Medical Journal of Zambia.	Ndola	*Med. J. Zambia*
Medical Laboratory Technology.	London	*Med. Lab. Technol.*
Medical Microbiology and Immunology.	Berlin	*Med. Microbiol. Immunol.*
Medical Proceedings.	Johannesburg	*Med. Proc.*
Medicina Cutánea.	Barcelona	*Medna Cutánea*
Medicina de Empresa.	Barcelona	*Medna Empresa*
Medicina del Lavoro.	Milano	*Medna Lav.*
Medicina. [Revista Mexicana.]	Mexico	*Medicina.* Mexico
Medicina y Seguridad del Trabajo.	Madrid	*Medna Segur. Trab.*
Medicina. [Stručni Časopis Liječnika Zdravstvenog Centra Rijeka.]	Rijeka	*Medicina.* Rijeka
Medicina Tropical.	Madrid	*Medna Trop.*
Medicine Today.	Karachi	*Med. Today*
Meditsinskaya Parazitologiya i Parazitarnye Bolezni.	Moskva	*Medskaya Parazit.*
Medycyna Doświadczalna i Mikrobiologia.	Warszawa	*Medycyna Dośw. Mikrobiol.*
Medycyna Pracy.	Warszawa	*Medycyna Pr.*
Mémoires de l'Institut Fondamental d'Afrique Noire.	Dakar	*Mém. Inst. Fond. Afr. Noire*
Memoirs of the College of Medicine National Taiwan University.	Taipei	*Mem. Coll. Med. Natn. Taiwan Univ.*
Memórias do Instituto Oswaldo Cruz.	Rio de Janeiro	*Mems Inst. Oswaldo Cruz*
Mens en Onderneming.	Leiden	*Mens Ondern.*
Mental Health.	London	*Ment. Hlth.* London
Metabolism. [Clinical and Experimental.]	New York	*Metabolism*
Methods of Biochemical Analysis.	New York	*Meth. Biochem. Anal.*
Microbiologia Española.	Madrid	*Microbiologia Esp.*
Microbiologia, Parazitologia, Epidemiologia.	Bucuresti	*Microbiologia Parazit. Epidem.*
Microbios.	Cambridge	*Microbios*
Mie Medical Journal.	Tsu City	*Mie Med. J.*
Mikrobiologija.	Beograd	*Mikrobiologija*
Mikrobiologiya.	Moskva	*Mikrobiologiya*
Milbank Memorial Fund Quarterly.	New York	*Milbank Meml Fund Q.*
Military Medicine.	Washington	*Milit. Med.*
Modern Medicine of Great Britain.	London	*Mod. Med. Gt Br.*
Monographies de l'Institut National de la Santé et de la Recherche Médicale.	Paris	*Monogrs Inst. Natn. Santé*
Morbidity and Mortality. [Weekly Report.]	Atlanta	*Morbid. Mortal.*
Mosquito News.	Albany	*Mosquito News*
Münchener Medizinische Wochenschrift.	München	*Münch. Med. Wschr.*
Munkavédelem.	Budapest	*Munkavédelem*
Mutation Research.	Amsterdam	*Mutat. Res.*
Mycopathologia et Mycologia Applicata.	Den Haag	*Mycopath. Mycol. Appl.*
Nagoya Journal of Medical Science.	Nagoya	*Nagoya J. Med. Sci.*
Nagoya Medical Journal.	Nagoya	*Nagoya Med. J.*
National Institute of Animal Health Quarterly.	Tokyo	*Natn. Inst. Anim. Hlth Q.*
Nature.	London	*Nature.* London
Nature New Biology.	London	*Nature New Biol.*
Nature Physical Sciences.	London	*Nature Phys. Sci.*
Nederlands Tijdschrift voor Geneeskunde.	Amsterdam	*Ned. Tijdschr. Geneesk.*
Nematologica.	Leiden	*Nematologica*
Népegészségügy.	Budapest	*Népegészségügy*
New England Journal of Medicine.	Boston	*New Engl. J. Med.*
New Zealand Medical Journal.	Wellington	*N. Z. Med. J.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
NLL Translations Bulletin. [National Lending Library.]	Boston Spa	*NLL Transl.Bull.*
Nordisk Hygienisk Tidskrift.	Lund	*Nord. Hyg. Tidskr.*
Nordisk Medicin.	Hälsingborg	*Nord. Med.*
Nuovi Annali d'Igiene e Microbiologia.	Roma	*Nuovi Ann. Ig. Microbiol.*
Nutrition Abstracts and Reviews.	Aberdeen	*Nutr. Abstr. Rev.*
Nutrition Newsletter.	Roma	*Nutr. Newsl.*
Nutrition Reviews.	New York	*Nutr. Rev.*
Occupational Health. [A Journal for Occupational Health Nurses.]	London	*Occup. Hlth*
Occupational Health Review.	Ottawa	*Occup. Hlth Rev.*
Occupational Psychology.	London	*Occup. Psychol.*
Occupational Safety Bulletin.	London	*Occup. Saf. Bull.*
Occupational Safety and Health.	London	*Occup. Saf. Hlth*
Öffentliche Gesundheitswesen.	Stuttgart	*Öff. Gesundhwesen*
Official Records of the World Health Organization *see* World Health Organization publications		
Onderstepoort Journal of Veterinary Research.	Pretoria	*Onderstepoort J. Vet. Res.*
Opuscula Medica.	Stockholm	*Opusc. Med.*
Organic Syntheses.	New York	*Org. Synth.*
Overseas Building Notes.	London	*Overseas Bldg Notes*
Paediatrica Indonesiana.	Jakarta	*Paediatrica Indones.*
Pahlaui Medical Journal.	Shivaz	*Pahlaui Med. J.*
Pakistan Medical Review.	Karachi	*Pakistan Med. Rev.*
PANS. [Pest Articles and News Summaries.]	London	*PANS*
Papua and New Guinea Medical Journal.	Port Moresby	*Papua New Guin. Med. J.*
Parasitologische Schriftenreihe.	Jena	*Parasit. Schriften.*
Parasitology.	Cambridge	*Parasitology*
Parassitologia.	Roma	*Parassitologia*
Parazitologicheskii Sbornik.	Leningrad	*Parazit. Sb.*
Pathologia et Microbiologia.	Basel	*Pathologia Microbiol.*
Pathology.	Sydney	*Pathology*
Pediatrics.	Evanston, Ill.	*Pediatrics*
Pesticides Documentation Bulletin.	Washington	*Pestic. Doc. Bull.*
Physics in Medicine and Biology.	London	*Physics Med. Biol.*
Physiological Reviews.	Washington	*Physiol. Rev.*
Population Newsletter.	New York	*Popul. Newsl.*
Population Studies.	London	*Popul. Stud.*
Postepy Higieny i Medycyny Doświadczalnej.	Warszawa	*Postepy Hig. Med. Doświad.*
Postgraduate Medical Journal.	London	*Postgrad. Med. J.*
Practitioner.	London	*Practitioner*
Problèmes Sociaux Congolais.	Lubumbashi	*Problèmes Soc. Congo.*
Proceedings of the Annual Meetings. New Jersey Mosquito Extermination Association.	Atlantic City, N. J.	*Proc. New Jers. Mosq. Exterm. Ass.*
Proceedings of the Helminthological Society of Washington.	Washington	*Proc. Helminth. Soc. Wash.*
Proceedings of the Microbiological Research Group of the Hungarian Academy of Sciences.	Budapest	*Proc. Microbiol. Res. Grp Hung. Acad. Sci.*
Proceedings of the Mine Medical Officers' Association.	Johannesburg	*Proc. Mine Med. Offrs Ass.*
Proceedings of the National Academy of Sciences of the United States of America.	Washington	*Proc. Natn. Acad. Sci. U.S.A.*
Proceedings of the Nutrition Society.	Cambridge	*Proc. Nutr. Soc.*
Proceedings and Papers of the Annual Conference of the California Mosquito Control Association.	Visalia, Calif.	*Proc. Pap. A. Conf. Calif. Mosq. Control Ass.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Proceedings of the Royal Society, Series B.	London	*Proc. R. Soc., Ser. B*
Proceedings of the Royal Society of Medicine.	London	*Proc. R. Soc. Med.*
Proceedings of the Society for Experimental Biology and Medicine.	New York	*Proc. Soc. Exp. Biol. Med.*
Proceedings of the United States National Museum.	Washington	*Proc. U.S. Natn. Mus.*
Progress in Hematology.	New York	*Prog. Hemat.*
Progress in Medical Genetics.	New York	*Prog. Med. Genet.*
Progress in Medical Laboratory Technique.	London	*Prog. Med. Lab. Tech.*
Progress in Medical Virology.	New York	*Prog. Med. Virol.*
Przeglad Epidemiologiczny.	Warszawa	*Przegl. Epidem.*
Przeglad Lekarski.	Kraków	*Przegl. Lek.*
Psychological Medicine.	London	*Psychol. Med.*
Public Health. [Journal of the New Zealand Branch of the Royal Society of Health.]	Auckland	*Publ. Hlth.* Auckland
Public Health.	Johannesburg	*Publ. Hlth.* Johannesburg
Public Health.	London	*Publ. Hlth.* London
Public Health Laboratory.	Pierre, South Dakota	*Publ. Hlth Lab.*
Public Health Papers *see* World Health Organization publications		
Publicações do Centro de Estudos Leprológicos.	Curitiba, Brasil	*Publções Cent. Estud. Leprol.*
Quaderni Sclavo di Diagnostica.	Siena	*Quad. Sclavo Diagn.*
Quarterly Journal of Medicine.	Oxford	*Q. J. Med.*
Queensland Journal of Agricultural and Animal Sciences.	Brisbane	*Qd J. Agric. Anim. Sci.*
Radovi Medicinskog Fakulteta u Zagrebu.	Zagreb	*Rad. Med. Fak. Zagr.*
Reports on Population/Family Planning.	New York	*Rept. Popul./Family Plann.*
Reproduction and Population Research Abstracts.	Bethesda, Md	*Reprod. Popul. Res. Abstr.*
Research Bulletin of the Meguro Parasitological Museum.	Tokyo	*Res. Bull. Meguro Parasit. Mus.*
Research in Reproduction.	London	*Res. Reprod.*
Research in Veterinary Science.	Oxford	*Res. Vet. Sci.*
Respiration Physiology.	Amsterdam	*Respn Physiol.*
Review of Applied Entomology, Series A and B.	London	*Rev. Appl. Ent., Ser. A* (or *Ser. B*)
Review of Czechoslovak Medicine.	Praha	*Rev. Czech. Med.*
Review of Medical and Veterinary Mycology.	Kew	*Rev. Med. Vet. Mycol.*
Review of Plant Pathology.	Kew	*Rev. Plant Path.*
Revista de la Asociación Médica Argentina.	Buenos Aires	*Revta Asoc. Méd. Argent.*
Revista da Associação Médica Brasileira.	São Paulo	*Revta Ass. Méd. Bras.*
Revista da Associação Médica de Minas Gerais.	Belo Horizonte	*Revta Ass. Méd. Minas Gerais*
Revista de Biologia Tropical.	San José	*Revta Biol. Trop.*
Revista Brasileira de Leprologia.	São Paulo	*Revta Bras. Leprol.*
Revista Brasileira de Malariologia e Doenças Tropicais.	Rio de Janeiro	*Revta Bras. Malar. Doenç. Trop.*
Revista Brasileira de Medicina.	Rio de Janeiro	*Revta Bras. Med.*
Revista Brasileira de Pesquisas Médicas e Biológicas.	São Paulo	*Revta Bras. Pesquisas Méd. Biol.*
Revista Cubana de Medicina.	Habana	*Revta Cub. Med.*
Revista Cubana de Medicina Tropical.	Habana	*Revta Cub. Med. Trop.*
Revista Cubana de Pediatría.	Habana	*Revta Cub. Pediat.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Revista Ecuatoriana de Higiene y Medicina Tropical.	Guayaquil	*Revta Ecuat. Hig. Med. Trop.*
Revista Ecuatoriana de Medicina y Ciencias Biológicas.	Quito	*Revta Ecuat. Med. Cienc. Biol.*
Revista da Faculdade de Medicina Veterinaria, Universidade de São Paulo.	São Paulo	*Revta Fac. Med. Vet. Univ. S. Paulo*
Revista de la Facultad de Ciencias Médicas de la Universidad Nacional de Córdoba.	Córdoba, Argentina	*Revta Fac. Cienc. Méd. Univ. Nac. Córdoba*
Revista de la Facultad de Medicina.	Maracaibo	*Revta Fac. Med.* Maracaibo
Revista de Farmácia e Bioquímica.	Belo Horizonte	*Revta Farm. Bioquím.*
Revista Goiana de Medicina.	Goiânia	*Revta Goiana Med.*
Revista do Hospital das Clínicas de Faculdade de Medicina da Universidade de São Paulo.	São Paulo	*Revta Hosp. Clín. Fac. Med. Univ. S. Paulo*
Revista Ibérica de Parasitología.	Granada	*Revta Ibér. Parasit.*
Revista do Instituto Adolfo Lutz.	São Paulo	*Revta Inst. Adolfo Lutz*
Revista do Instituto de Medicina Tropical de São Paulo.	São Paulo	*Revta Inst. Med. Trop. S. Paulo*
Revista de Investigación en Salud Pública.	Mexico	*Revta Invest. Salud Públ.*
Revista Latinoamericana de Microbiologia y Parasitologia.	Mexico	*Revta Lat.-am. Microbiol.*
Revista Médica del Hospital Colonia.	Mexico	*Revta Méd. Hosp. Colonia*
Revista Paulista de Medicina.	São Paulo	*Revta Paul. Med.*
Revista de Sanidad e Higiene Pública.	Madrid	*Revta Sanid. Hig. Públ.*
Revista de la Sanidad Militar Argentina.	Buenos Aires	*Revta Sanid. Milit. Argent.*
Revista de la Sanidad de Policia.	Lima	*Revta Sanid. Polic.*
Revista de Saúde Pública.	São Paulo	*Revta Saúde Públ.*
Revista de la Sociedad Peruana de Dermatología.	Lima	*Revta Soc. Peruana Derm.*
Revista da Sociedade Brasileira de Medicina Tropical.	Rio de Janeiro	*Revta Soc. Bras. Med. Trop.*
Revista Venezolana de Sanidad y Asistencia Social.	Caracas	*Revta Venez. Sanid. Asist. Social*
Revista del Viernes Médico.	Lima	*Revta Viernes Méd.*
Revue d'Epidemiologie Médecine Sociale et Santé Publique.	Paris	*Revue Epid. Méd. Soc. Santé Publ.*
Revue Française des Affaires Sociales.	Paris	*Revue Fr. Aff. Sociales*
Revue d'Immunologie.	Paris	*Revue Immunol.*
Revue de l'Institut d'Hygiène des Mines.	Hasselt	*Revue Inst. Hyg. Mines*
Revue Internationale des Services de Santé des Armées de Terre, de Mer et de l'Air.	Liège	*Revue Int. Servs Santé Armées*
Revue Internationale du Trachome.	Paris	*Revue Int. Trachome*
Revue Médicale Minière.	Douai	*Revue Méd. Min.*
Revue Roumaine d'Inframicrobiologie.	Bucuresti	*Revue Roum. Inframicrobiol.*
Rivista Italiana d'Igiene.	Pisa	*Riv. Ital. Ig.*
Rivista di Parassitologia.	Roma	*Riv. Parassit.*
Romanian Medical Review.	Bucuresti	*Rom. Med. Rev.*
Royal Society of Health Journal.	London	*R. Soc. Hlth J.*
Rural Medicine.	London	*Rural Med.*
Safety in Mines Research Establishment. Abstracts of Current Publications.	Sheffield	*SMRE Abstracts*
Safety in Mines Research Establishment. Research Reports.	Sheffield	*SMRE Res. Rep.*
St. Luke's Hospital Gazette.	Malta	*St. Luke's Hosp. Gaz.*
Salud Occupacional.	Lima	*Salud Occup.*
Salud Pública de México.	Mexico	*Salud Públ. Méx.*
Santé Publique.	Bucuresti	*Santé Publ.*
Saúde Pública. Boletim dos Serviços de Saúde Pública.	Lisboa	*Saúde Públ. Bolm. Servs Saúde Públ.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Sbornik Trudove na Nauchno-Issledovatelskija Khigienen Institut.	Sofia	*Sb. Trud. Nauchno-Issled. Khig. Inst.*
Scandinavian Journal of Infectious Diseases.	Stockholm	*Scand. J. Infect. Dis.*
Scandinavian Journal of Respiratory Diseases.	Copenhagen	*Scand. J. Resp. Dis.*
Schweizerische Medizinische Wochenschrift.	Basel	*Schweiz. Med. Wschr.*
Science.	Washington	*Science.* Washington
Scientific American.	New York	*Scient. Am.*
Scottish Medical Journal.	Glasgow	*Scott. Med. J.*
Scripta Scientifica Medica. [Annual Scientific Papers of the Higher Medical Institute.]	Varna	*Scr. Sci. Med.*
Securitas.	Roma	*Securitas*
Selected Papers of the Royal Netherlands Tuberculosis Association.	Den Haag	*Selecd Pap. R. Neth. Tuberc. Ass.*
Shikoku Acta Medica.	Tokushima	*Shikoku Acta Med.*
Singapore Medical Journal.	Singapore	*Singapore Med. J.*
Social Biology.	Chicago	*Social Biol.*
Social and Economic Administration.	Exeter	*Social Econ. Adm.*
Social Psychiatry.	Berlin	*Social Psychiat.*
Social Science and Medicine.	Oxford	*Social Sci. Med.*
Social Science Research Council Newsletter.	London	*SSRC Newsl.*
Social Service Quarterly.	London	*Social Serv. Q.*
South African Journal of Medical Sciences.	Johannesburg	*S. Afr. J. Med. Sci.*
South African Journal of Obstetrics and Gynaecology.	Cape Town	*S. Afr. J. Obstet. Gynaec.*
South African Journal of Surgery.	Johannesburg	*S. Afr. J. Surg.*
South African Medical Journal.	Cape Town	*S. Afr. Med. J.*
South East Asian Journal of Tropical Medicine and Public Health.	Bangkok	*S.E. Asian J. Trop. Med. Publ. Hlth*
South Pacific Commission. Technical Papers.	Noumea	*S. Pac. Com. Tech. Pap.*
Southern Medical Journal.	Birmingham, Ala	*Sth. Med. J.*
Sovetskoe Zdravookhranenie.	Moskva	*Sov. Zdravookhr.*
Städtehygiene.	Hamburg	*Städtehygiene*
Stain Technology.	Geneva, N.Y.	*Stain Technol.*
Statistical News.	London	*Statist. News*
Staub.	Düsseldorf	*Staub*
Studia Pneumologica et Phtiseologica Cechoslovaca.	Praha	*Studia Pneumol. Phtiseol. Cechoslov.*
Studies in Family Planning.	New York	*Stud. Family Plann.*
Sudan Medical Journal.	Khartoum	*Sudan Med. J.*
Texas Reports on Biology and Medicine.	Galveston	*Tex. Rep. Biol. Med.*
Theoretical Population Biology.	New York	*Theoret. Popul. Biol.*
Thorax.	London	*Thorax*
Tijdschrift voor Diergeneeskunde.	Utrecht	*Tijdschr. Diergeneesk.*
Tohoku Journal of Experimental Medicine.	Sendai	*Tohoku J. Exp. Med.*
Tokushima Journal of Experimental Medicine.	Tokushima	*Tokushima J. Exp. Med.*
Toxicon.	Oxford	*Toxicon*
Transactions of the Royal Entomological Society.	London	*Trans. R. Ent. Soc.*
Transactions of the Royal Society of Tropical Medicine and Hygiene.	London	*Trans. R. Soc. Trop. Med. Hyg.*
Transactions of the Society of Occupational Medicine.	London	*Trans. Soc. Occup. Med.*
Tropical Animal Health and Production.	Edinburgh	*Trop. Anim. Hlth Prod.*
Tropical Doctor.	London	*Trop. Doctor*
Tropical and Geographical Medicine.	Haarlem	*Trop. Geogr. Med.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
Tropical Health.	Kampala	*Trop. Hlth*
Tropical Medicine.	Nagasaki	*Trop. Med.*
Tropical Medicine and Hygiene News.	Bethesda, Md	*Trop. Med. Hyg. News*
Trudy Gel'mintologicheskoi Laboratorii, Akademiya Nauk SSSR.	Moskva	*Trudy Gel'mint. Lab.*
Trudy Nauchno-Issledovatel'skogo Instituta Epidemiologii i Mikrobiologii.	Sofia	*Trudy Nauchno-Issled.Inst. Epidem. Mikrobiol.*
Trudy Zoologischeskogo Instituta, Akademiya Nauk SSSR.	Leningrad	*Trudy Zool. Inst.* Leningrad
Tubercle.	London	*Tubercle.* London
Tunisie Médicale.	Tunis	*Tunis. Méd.*
Türk Hijiyen ve Tecrübî Biyoloji Dergisi.	Ankara	*Türk Hij. Tecr. Biyol. Derg.*
Ulster Medical Journal.	Belfast	*Ulster Med. J.*
Union Internationale contre le Cancer. Technical Report Series.	Geneva	*UICC Techn. Rep. Ser.*
United States National Museum Bulletin.	Washington	*U.S. Natn. Mus. Bull.*
United States Public Health Service Publications.	Washington	*U.S.P.H.S. Publs*
University of California Publications in Entomology.	Berkeley	*Univ. Calif. Publs Ent.*
University of California Publications in Zoology.	Berkeley	*Univ. Calif. Publs Zool.*
University of Kansas Science Bulletin.	Lawrence	*Univ. Kansas Sci. Bull.*
Věstnik Československé Společnosti Zoologické.	Praha	*Věst. Čsl. Spol. Zool.*
Vestnik Dermatologii i Venerologii.	Moskva	*Vest. Derm. Vener.*
Veterinary Annual.	Bristol	*Vet. A.*
Veterinary Bulletin.	Weybridge	*Vet. Bull.* Weybridge
Virology.	New York	*Virology*
Vital and Health Statistics. [U.S. DHEW Publications.]	Rockville, Md	*Vital Hlth Statist.*
Voprosy Leprologii i Dermatologii.	Rostov-na-Don	*Vop. Leprol. Derm.*
Voprosy Pitaniya.	Moskva	*Vop. Pitan.*
Vox Sanguinis. [Journal of Blood Transfusion, Immunohaematology and Immunopathology.]	Basel	*Vox Sang.*
Wakayama Medical Reports.	Wakayama	*Wakayama Med. Rep.*
Water Pollution Abstracts.	London	*Wat. Pollut. Abstr.*
Well-being. [A Review of Health and Education.]	Wakefield	*Well-being*
West African Medical Journal.	Ibadan	*W. Afr. Med. J.*
West Indian Medical Journal.	Kingston, Jamaica	*W. Indian Med. J.*
Wiadomości Parazytologiczne.	Warszawa	*Wiad. Parazyt.*
Work-Environment-Health.	Helsinki	*Wk-Envir.-Hlth*
World Health Organization publications:		
Bulletin of the World Health Organization.	Geneva	*Bull. Wld Hlth Org.*
Official Records of the World Health Organization.	Geneva	*Off. Rec. Wld Hlth Org.*
Public Health Papers.	Geneva	*Publ. Hlth Pap. Wld Hlth Org.*
Weekly Epidemiological Record.	Geneva	*Wkly Epidem. Rec.*
WHO Chronicle.	Geneva	*WHO Chron.*
World Health.	Geneva	*Wld Hlth*
World Health Organization Monograph Series.	Geneva	*Wld Hlth Org. Monogr. Ser.*
World Health Organization Technical Report Series.	Geneva	*Wld Hlth Org. Techn. Rep. Ser.*
World Health Statistics Report.	Geneva	*Wld Hlth Statist. Rep.*

Title of Periodical	*Place of Publication*	*Abbreviations used in Bulletins*
World Medical Instrumentation.	London	*Wld Med. Instrum.*
World Medical Journal.	New York	*Wld Med. J.*
Yale Journal of Biology and Medicine.	New Haven, Conn.	*Yale J. Biol. Med.*
Yokohama Medical Bulletin.	Yokohama	*Yokohama Med. Bull.*
Yonago Acta Medica.	Yonago	*Yonago Acta Med.*
Your Health.	Calcutta	*Your Hlth.* Calcutta
Zbornik Lekárskej Fakulty Košice.	Bratislava	*Zb. Lek. Fak. Košice*
Zdravstveni Vestnik.	Ljubljana	*Zdravst. Vest.*
Zdrowie Publiczne.	Warszawa	*Zdrow. Publ.*
Zeitschrift für Allgemeine Mikrobiologie, Morphologie, Physiologie, Genetik und Ökologie der Microorganismen.	Berlin	*Ztschr. Allg. Mikrobiol.*
Zeitschrift für Ärztliche Fortbildung.	Jena	*Ztschr. Arztl. Fortbild.*
Zeitschrift für die Gesamte Hygiene und ihre Grenzgebiete.	Berlin	*Ztschr. Ges. Hyg.*
Zeitschrift für Immunitätsforschung, Allergie und Klinische Immunologie.	Stuttgart	*Ztschr. Immunforsch.*
Zeitschrift für Parasitenkunde.	Berlin	*Ztschr. Parasitkde*
Zeitschrift für Präventivmedizin.	Zürich	*Ztschr. Prävmed.*
Zeitschrift für Tropenmedizin und Parasitologie.	Stuttgart	*Ztschr. Tropenmed. Parasit.*
Zeitschrift für Unfallmedizin und Berufskrankheiten.	Zürich	*Ztschr. Unfmed. Berufskr.*
Zentralblatt für Arbeitsmedizin und Arbeitsschutz.	Darmstadt	*Zentbl. Arbmed. Arbschutz*
Zentralblatt für Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene.		
Abteilung I. Originale, Reihe A und B.	Stuttgart	*Zentbl. Bakt. I. Orig., Ser. A* (or *Ser. B*)
Abteilung I. Referate.	Stuttgart	*Zentbl. Bakt. I. Ref.*
Abteilung II.	Jena	*Zentbl. Bakt. Abt. II.*
Zentralblatt für Verkehrs-Medizin Verkehrs-Psychologie Luft- und Raumfahrt-Medizin.	München	*Zentbl. Verkehrsmed.*
Zhurnal Mikrobiologii, Epidemiologii i Immunobiologii.	Moskva	*Zh. Mikrobiol. Epidem. Immunobiol.*

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High Blood Pressure

What is meant by `Blood Pressure' ?

Our heart is a kind of a pump. With each heartbeat, pure (oxygen rich) blood is pumped into the arteries of the body. Due to this pumping action, pressure is generated in the blood vessels, which is called `blood pressure'. Just as water comes out of a tap under pressure, similarly blood flows in the blood vessels of the body under certain pressure. During each minute, the heart beats, that is contracts and relaxes, about 70 to 80 times. While the heart is contracting, naturally the pressure in the arteries is higher. This is called the `upper' or *systolic* blood pressure, that is the pressure when the heart is contracting. This is often in the range of 110 to 130 millimetres of mercury. This means that this blood pressure is equal to the pressure that would be generated by a column of mercury say, 110 or 130 millimetres high. When the heart relaxes, the blood pressure is less. This is called the `lower' or *diastolic* blood pressure, that is the pressure when the heart is relaxed. In an adult this is usually in the range of 70 to 90 millimetres of mercury. The blood pressure is noted down as 130/90, 110/70 and so on. A blood pressure of 130/90 means an `upper'(systolic) pressure of 130 millimetres of mercury and a `lower'(diastolic) pressure of 90 millimetres of mercury.

What is meant by `High Blood Pressure' ?

The Joint National Committee on High Blood Pressure (an expert committee) unanimously decided that an `upper' (systolic) B.P. of 140 mm. of mercury or more, and a `lower'(diastolic) B.P. of 90 mm. of mercury or more, should be termed High Blood Pressure. This is irrespective of the persons sex or age. If the B.P. of a pregnant woman rises by 30 mm. compared to her pre-pregnancy B.P., then that too would be called High Blood Pressure. By the same logic, if the B.P. of any person rises by 30 mm. compared to his/her previous B.P., then it needs to be treated. Before labelling a person as having High Blood Pressure, her/his B.P. should be measured at least twice, and that too at an interval of a few weeks. The reason is that anxiety or the very process of the B.P. being measured by a doctor, can temporarily raise the B.P. of a person. If two measurements show a raised B.P., then such a person should be termed as having High Blood Pressure. A B.P. of upto 130/85 is considered normal, and above this upto 139/89 is considered `high normal'. A person with `high normal' B.P. should have his/her B.P. measured every six months.

What is meant by `Low Blood Pressure' ?

There is actually no disease such as `low blood pressure'. Some healthy and active people have a usual B.P. of 90/50 or even less (especially women). This itself carries no risk. However, if a person's blood pressure drops sharply, compared to her/his usual B.P., then this is indicative of serious illness . For example due to a heart attack, a persons B.P. may fall suddenly, and this is a sign of a life-threatening situation.

Why and how does the ailment of High Blood Pressure develop?

There is a network of innumerable fine blood vessels throughout our body. Sometimes, due to specific reasons, these small blood vessels become narrowed. Therefore, the heart has to pump blood at a higher pressure through such vessels, to ensure adequate blood supply to various organs. This is what we know as High Blood Pressure. Why do the blood vessels become narrowed ? We know the actual reason for this in only about 5 percent of patients with high B.P.. In the remaining patients, there is the combined effect of various factors. Among these factors, we have no control over some which are mentioned below.

1)Heredity: If neither parent, one parent or both parents have High Blood Pressure, then the probability of an offspring having the same ailment is 3%, 30% and 45% respectively. Among

those who have a family history of high B.P., the probability of having this disease increases five-fold.

2) Defects in arterial walls: The cells in the walls of arteries (blood vessels which carry blood from the heart to other parts of the body) sometimes malfunction. There is an imbalance in these cells between salt (sodium chloride) and calcium. Due to this, these arteries contract and the blood pressure increases. In such persons, an excess of salt in the diet tends to increase the B.P.

3) Ageing : The walls of arteries become less elastic with increasing age. Due to such hardening of arteries, the B.P. increases.

4) Male sex : Men are more prone to develop high blood pressure than women in the below-forty age group.

We obviously have no control over the above mentioned factors. However, certain factors like overweight due to faulty diet and lifestyle; lack of exercise; excess salt in the diet; competition and related mental stress; compulsive addiction to tobacco products; - all increase the likelihood of a person developing high blood pressure. Such lifestyle related risk factors are socially conditioned causes of High Blood Pressure - and definitely can be prevented.

What are the ill effects of High Blood Pressure on the body ?

The heart of a person with High Blood Pressure has to pump blood continuously with increased force, which causes strain to the heart. If this strain becomes excessive, the heart may fail to pump blood adequately - known as heart failure. Besides this, flow of blood at a higher pressure leads to injury to the internal lining of small blood vessels. Therefore, a layer of a fatty substance called cholesterol tends to become deposited more rapidly. If the arteries supplying blood to vital organs like the brain, heart or kidneys become narrowed by such cholesterol deposition, then the circulation of blood to such organs is decreased. This increases the likelihood of disease related to these critical organs. The delicate blood vessels of the retina of the eye, kidneys, brain or heart may not be able to stand the sustained high blood pressure . They may rupture leading to serious ailments such as loss of vision, paralysis, kidney disease, or heart attack. If the raised B.P. is brought down and kept normal by appropriate treatment, then naturally these risks can be avoided.

The raising of which is more dangerous - `lower' or `upper' type of Blood Pressure ?

Some years back it was believed that the raising of the `lower' (diastolic) B.P. is more dangerous. However recent research has shown that raising of either `upper'(systolic) or `lower' (diastolic) blood pressure are equally dangerous.

How does one recognise High Blood Pressure ?

Some patients with high B.P. may have complaints like headache, shortness of breath on exertion, palpitation (awareness of heartbeat) , or bleeding from the nose. Presence of any of these should prompt one to get one's blood pressure measured by a doctor. However, most patients of High Blood Pressure do not have any symptoms at all - while the body continues to undergo the strain of raised B.P. Hence High Blood Pressure has been called a silent disease. Therefore it is best to get one's B.P. measured periodically, from youth onwards. If any parents or siblings have high B.P., if there is a background history of diabetes, overweight or tobacco addiction, then the B.P. should be measured at a young age. If there is no such predisposing factor, still one should get one's B.P. measured once in a year after the age of forty.

If the B.P. is found to be raised during such an examination, then the measurement is repeated after two weeks, twice during the morning. (The morning B.P. is more significant for diagnosis of the ailment). Treatment is initiated only if the B.P. is found to be consistently high. Those with mildly raised B.P. have sometimes been found to have raised B.P. in the doctors clinic, but a normal B.P. when measured at home by a nurse or oneself. Hence before finalising a diagnosis of `mildly raised B.P.', it is preferable to measure the B.P. at the patients home.

Besides measuring the blood pressure, are other tests also necessary ?

The treating doctor should naturally do a complete physical examination. This may sometimes reveal the cause of raised B.P.. High Blood Pressure may lead to adverse effects on organs like the retina of the eye, kidneys, heart or brain. Of these, to determine adverse changes in the blood vessels of the brain requires sophisticated tests. However, the retina of the eye can be easily examined. Routine examination of the urine and level of a substance called creatinine in the blood gives an indication about damage to the kidneys. The E.C.G. (electrical examination of the heart) and Echocardiogram (sonography of the heart) give information about adverse changes in the heart. All these tests yield information about damage to vital organs. If the B.P. is only mildly raised, then only changes in lifestyle (weight control, reducing stress, restricting salt intake, regular exercise, ceasing tobacco intake etc.) may be sufficient to control the problem and it may be possible to avoid drugs.

If the person also has diabetes or if the level of cholesterol in the blood is raised, then the adverse effects of high B.P. are aggravated. Therefore it is essential to test for levels of blood sugar and cholesterol.

If high B.P. is detected at a young age, then it is necessary to perform tests and sonography of the kidneys. This is because there is a higher likelihood of kidney disease in such patients.

If an abnormality is detected in any of these tests, then additional investigations may need to be done. High Blood Pressure is not a short-term illness, and it may have life-threatening consequences. Therefore it is important not to be negligent regarding these tests, nor should one try to avoid expense by skipping them.

How is High Blood Pressure treated ?

Much research has been done on High Blood Pressure. The ailment of High B.P. can be effectively kept under control by means of modern treatment. In milder forms of the disease, measures besides drugs are also effective.

Highly effective and comparatively safe drugs are available today to treat high B.P. Still, measures like appropriate diet, lifestyle, regular exercise, freedom from mental stress and avoidance of addictions are as important as medication. An unhealthy lifestyle tends to cause not only high B.P., but other diseases also. Instead of taking separate medication for each such disease, it is logical to take a holistic approach and adopt a healthy lifestyle which would minimise the need for drugs.

If a single medical examination shows a B.P. above 140/90, there is no need to immediately start medication. According to recent research, the treatment depends on the following conditions.

It has been demonstrated recently, that of those persons with mildly raised B.P. (systolic 140 to 159 mm./ diastolic 90 to 99 mm.), twenty percent show raised B.P. when examined by a doctor but normal B.P. when examined at home by a nurse or themselves. Therefore such persons should confirm by repeated measurements at home or by the doctor, as to whether there actually is a problem of high B.P. .

There is a group of patients whose B.P. is in the range 140 to 159 systolic / 90 to 99 mm. diastolic, and who have no risk factors like overweight, addiction to tobacco, diabetes, raised cholesterol level, or death of a blood relative from heart disease below the age of fifty. At the same time if tests show no adverse effects of raised B.P. on the retina, kidneys, heart then recent research shows that there is no need to initiate medication. Such persons should keep a watch on their B.P. by having it measured periodically, and get regular exercise, keep their weight under control, avoid mental stress so that the B.P. can be brought down further.

Next are those patients who have a B.P. which is higher than 160 systolic / 100 diastolic but not excessively raised, and who do not have the above mentioned risk factors nor do they have

adverse effects on retina, kidneys and heart. In such persons also an attempt should be made to lower the B.P. below 140/90 by means of weight reduction, regular exercise, avoiding tobacco, preventing mental stress, reducing salt in the diet etc. This should be given a trial for three to six months and only if this does not succeed, should medication for the high B.P. be started.

Does reducing one's weight bring down raised blood pressure ?

Yes! To some extent this is possible. Especially among those whose weight is above the 'ideal' , a weight reduction of each kilogram leads to a fall in B.P. of 1.5 to 2 mm. You can calculate your desirable weight by means of the following formula:

Ideal weight (in kilograms) = Height (in centimetres) - 100

A weight that is five to ten percent above or below this is acceptable. Changes in the diet and regular exercise both help in weight reduction.

Each kilogram of excess weight means that there are 800 grams excess fat deposited in the body. To reduce this in a month, one should reduce 7200 calories from ones food intake for the month (one gram of fat equals 9 calories). This means 240 calories every day. Thus reducing ones daily diet by 240 calories would lead to a weight loss of one kilogram in a month.

The following food portions contain 100 calories each:

One serving bowl (`vati') of cooked rice (30 grams of uncooked rice) ; a chapati of six inches diameter (30 grams of flour) ; a plain dosa ; two idlis ; two slices of bread ; a serving bowl (`vati') of cooked dal (30 grams uncooked dal) ; one large egg ; a banana ; two and a half teaspoons of butter ; two teaspoons of oil or ghee.

Some principles may be kept in mind relevant to reducing weight and maintaining this reduction - sweet and oily foodstuffs are undesirable from the health point of view. They should be absolutely restricted. A person should not use more than 20 grams of oil in a day or 0.6 kg. oil in a month for cooking. Taking additional butter, ghee also means adding fat, which should be avoided. Ghee obtained from milk consumed in the house may be taken, not more than one teaspoon per day. Other animal fats should be avoided ; non-vegetarian foods excepting fish should be cut down. One should not consume more than 15 eggs in a month.

One should eat at specified mealtimes only. Between-meal snacks should be totally avoided. Foods like leafy vegetables, raw salads which contain mainly water and fibre should be increased in the diet.

Does exercising help ?

Besides dieting, the other means of reducing excess weight is exercise. To reduce a kilogram of weight, one has to burn or expend 7200 extra calories. You can determine how much your present weight is above the ideal, i.e. how many excess kilograms you need to shed. Of this excess weight, decide how much you are going to shed in the next three months by means of dieting. The remaining should be reduced by exercising. Calories being used up per minute in various types of exercise are as follows: housework, gardening, painting - 2 to 5 calories; masonry type of work - 4 to 5 calories ; walking quickly, climbing stairs, bicycling, playing tennis, carpentry - 5 to 10 calories. On this basis you may determine which exercise you should do, and for how long you need to do it daily. Among these, walking is the easiest and safest form of exercise. You may start with that. It is necessary to walk continuously at least half an hour daily to achieve any weight reduction. Once you are accustomed to walking half to one hour daily, you can move to other forms of exercise based on your doctors advice.

You can determine whether your heart can bear exercise or not by the following simple test. First measure your pulse rate per minute while sitting down. Then measure it again while standing up. If there is a difference of more than 20 beats between the two rates, then do not indulge in strenuous exercise like running and jogging. Another simple test is - climb up and down a single stair thirty times in one minute. Then measure your pulse rate. If it exceeds 120 then do not take up exercises like jogging or running. First reduce your weight by walking regularly. Then take up running etc. only after consulting your doctor. Perhaps your doctor might advise a test called 'stress test' before recommending exercise. During this test one is required to walk in a single place on a moving belt. During this walking, electrical tracings of the heart are taken (E.C.G.). Gradually the speed and gradient of the moving belt are increased, making the exercise more strenuous for the heart. Whether the heart can bear the strain of this exercise or not, is revealed by the E.C.G..

After having reduced one's weight, one needs to exercise in a specific way only 10 minutes per day, three times a week, in order to remain healthy. This has been proved by the world renowned expert, Dr. Lawrence Morehouse. How to achieve this is given later in this booklet, which you can practice.

Besides weight reduction, are there other methods to reduce raised B.P. ?

Yes, there are! These are beneficial to at least some extent for most patients. It is possible to avoid medication in case of mildly raised B.P., by taking measures related to diet and exercise, and adopting other non-drug methods detailed below. In others with severely raised B.P., the dosage of medications can be reduced by these means.

Other measures

- Reduce salt in the diet ; do not take salt in your plate. Avoid salty items like pickles, papad, wafers etc.
- Raised B.P. can be brought down by reducing mental stress. One should try to change one's mentality and outlook, not becoming unnecessarily tense, spending some time every day quietly and with a relaxed mind (one's favourite music can help in this). One should try to breathe 'naturally' - that is expanding one's abdomen while inhaling and contracting the abdomen while exhaling. This can help in reducing raised B.P..
- Stopping tobacco intake in any form (smoking, pan masala, chewing tobacco) helps to reduce raised B.P.. A substance called Nicotine in tobacco causes the blood vessels to occasionally contract. This leads to injury to the delicate inner lining of these vessels, and the process of deposition of a layer of fat there, is accelerated. This leads to narrowing of the blood vessels and increased probability of their becoming blocked by a clot. Stopping tobacco intake reduces these adverse effects.

Are effective medicines available to treat High Blood Pressure ?

Today a number of effective medicines are available to treat high B.P. Which medication to take, and in what dosage, depends on a number of factors. The level of raised B.P., age of the patient, whether diabetes exists or not, financial situation etc.

Though the drugs available today to treat high B.P. are comparatively safe, yet they can cause side effects. Therefore if one experiences any new complaint after starting medication, the doctor should be informed. Diuretics (medicines which increase urination) can cause frequent urination and a feeling of weakness. Some other drugs may cause increase or decrease in the pulse rate, cough, swelling over ankles, dizziness after standing up or shortness of breath. If any of these is experienced then the doctor should be informed; the doctor may change the medication if necessary. On the whole, of the drugs introduced in the last 5-10 years, one or the other usually suits the patient and does not cause significant side effects even if taken lifelong.

What is the period of treatment for High Blood Pressure ?

About five percent of patients with high B.P. have an underlying cause in form of disease of the kidneys or hormonal glands. If this disease is treated then the B.P. comes down to normal and further treatment is not required. However, the remaining 95% of patients with high B.P. require lifelong treatment. Alteration in diet and lifestyle, regular exercise, freedom from addictions, reducing mental stress - these measures should be permanently continued. Mildly raised B.P. may be controlled just with these measures. However in cases of moderately to severely raised B.P., medication has to be taken continuously and lifelong. Claiming that 'my B.P. is presently normal' or 'I feel all right now' and stopping medication on this basis is incorrect and can be dangerous. This is because the B.P. may be maintained at normal level only due to the effect of the drugs. Also, as mentioned before, high B.P. is a silent disease which may cause damage without producing symptoms - so it is incorrect to rely on one's subjective judgement about being normal.

After starting treatment, is it necessary to undergo medical examination and other tests from time to time ?

Yes. However, the nature of tests depends on the situation of the particular patient. If the B.P. is excessively raised, then it should be measured once a week. Once the B.P. is under control, it may be measured once in two to four months only.

If initial examinations show defects in the heart, kidneys or retina, then regular examinations may be advised as necessary.

Can one prevent High Blood Pressure ?

Yes. All the non-drug measures mentioned above for treatment of high B.P. are effective in preventing the disease if adopted from youth onwards. Only kidney disease or hormonal imbalances cannot be influenced by these measures. Those whose parents have high B.P. need to specially take the precautions related to diet, exercise and mental stress.

At the broader level, a sedentary and consumerist culture is on the rise in our society. Unhealthy food, lifestyle and addiction to tobacco products are contributing to a situation almost like an epidemic of high B.P.. To check this, it is necessary to change the basic social conditions in which we live today.

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HYPER ACIDITY & ULCER-TROUBLE

What is acidity ?

Though 'Acidity-problem', 'Pitta-problem' are the terms quite frequently used, they are not scientifically correct terms. All of us have acidity in our stomach, since hydrochloric-acid, which is essential for digestion of food in the stomach, is produced daily in our stomach. When this hydrochloric acid is produced in excess, it is called as hyperacidity. Some people say that they suffer from Pitta-problem. This is described as : headache accompanied by Uneasiness in stomach and relieved by vomiting. But this is a form of migrainous headache and is different from hyper-acidity.

Hyperacidity leads to a variety of symptoms e.g. discomfort, burning or pain in the stomach above the umbilicus or in the centre of the chest behind the sternum, vomiting, excessive burping are common amongst these. Patients call these symptoms by a variety of names : 'acidity-problem,' 'Pitta-problem,' 'gas-trouble' These symptoms can occur in other diseases also, for example, in certain diseases of the pancreas or gall-bladder.

Hence the point to remember here is that if any of the symptoms appear repeatedly, doctor's advice should be definitely sought for. It is dangerous to just keep taking widely advertized ~~~ : 'anti-acidity drugs.' It is necessary to find out the cause behind these symptoms--whether they are due to hyperacidity or gastritis (inflammation of stomach lining) or ulcer or gall-bladder or pancreatic disease. Moreover, especially after the forties, the possibility of cancer of the stomach has to be kept in mind.

What is ulcer ? Is it different from hyperacidity ?

The accompanying diagram shows the upper part of our gastro-intestinal track or food-track. It consists of gullet or oesophagus which carries food from the mouth to the stomach. From the stomach, the food enters the first part of the small-intestine, called as the duodenum. All these parts are lined from inside by a tender lining. This lining is

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sometimes damaged and develops ar breach in its continuity, a kind of a small wound, called ulcer. The accompanying diagram shows common sites of ulcer in the oesophagus, the stomach and the duodenum. Ulcer is not the same as hyper-acidity. Ulcer occurs if hyperacidity remains untreated. In some of the patients, however, there is no excess of acid, yet they develop ulcer. On the otherhand, in some of the persons hyperacidity does not develop into an ulcer, even if not treated !

How to suspect an ulcer ?

The symptoms of ulcer are the same as those of hyper-acidity. If you get these symptoms repeatedly, or for more than a week, suspect ulcer. Rarely, however, some ulcer - patients have no symptoms; in this unfortunate lot, the ulcer grows without giving any warning signals, and the ulcer is diagnosed only when it leads to serious complications like : perforation (a hole) in the stomach or duodenum at the ulcer-site, causing sudden agonizing pain in upper abdomen or bleeding from the ulcer causing coffee-coloured vomiting or frank-blooddy-vomiting or passing of black-coloured stools.

Why does ulcer occur ?

The internal lining of the stomach secretes digestive juices called pepsin and hydrochloric acid. A delicate, complex mechanism ensures that these juices digest food, but do not erode the tender internal lining. But if this delicate balance breaks down, these juices start eroding this lining. This leads to ulcer-formation. This delicate balance breaks down due to the following reasons :-

1) Increased acid secretion : Psychological tensions, consumption of tobacco, alcohol causes causes increased secretion of hydrochloric acid and psp sin. In some of the families, there is a tendency towards excessive secretion of acid. Ulcer tends to occur more frequently in such families.

2) Irregular faulty diet : The stomach is used to secrete hydrochloric acid at our usual meal-times. If we do not take food at this time, in absence of the food, the acid acts on the stomach-lining and erodes it. If this irregularity occurs repeatedly, ulcer develops.

If food is eaten in a hurry, the alkaline saliva is not mixed with food in adequate quantity and this increases the acidity in the stomach and thereby helps form an ulcer.

Hot, spicy food also tends to damage the internal lining of the upper G.I. track.

The causation of ulcer is summed up in a saying in medicine : " hurry, worry, curry causes ulcer."

3) Deficiency in the buffer-mechanism : The internal lining of the stomach and duodenum is protected from the eroding effect of the hydrochloric acid, by a buffer-mechanism or 'mucosal resistance.' This happens due to the infection of the bacteria called H.Pylori. This breakdown of mucosal resistance may also occur due to consumption of tobacco in any form (from cigarette to Gutkha), alcohol, aspirin and similar drugs used in the treatment of rheumatism, as well as by steroid-group of drugs.

4) Defect in the valve between the stomach and the gullet : We have a kind of valve which prevents regurgitation of the acidic contents of the stomach back into the gullet. Some times, it gets defective. As a result, the acidic contents of the stomach flow back into the oesophagus. The internal-lining of the oesophagus is not used to the hydrochloric acid or the pepsin. It is therefore, easily eroded and an ulcer is found at the lower end of the gullet. Initially, there is burning sensation in the chest (heart-burn) behind the sternum, and reguragition of sour, acidic stomach-contents. As ulcer develops, there is pain in the centre of the chest. just above the stomach.

How is ulcer diagnosed ?

If symptoms of hyperacidity/ulcer described above occurs, repeatedly, and especially if there is pain in the upper abdomen, consult your doctor at the earliest. Doctor would ask you several questions about your work, food-habits, and would examine you in detail to arrive at a preliminary diagnosis. Ideally, for a definite diagnosis, an investigation called endoscopy of the upper gastrointestinal track, should be done.

(For the sake of brevity, it is called as gastroscopy). In this investigation, the interior of the upper gastrointestinal track (GI-track) is seen with the help of an endoscopic tube which consists of fibre-optic tubules. The patient is asked to swallow one end of this tube till this end reaches the stomach. The interior of the GI track is seen through the other end by a specially trained doctor, by passing light thro' it. If there is any ulcer or any other abnormality, it is directly seen and a definite diagnosis is made. Gastroscopy however, costs from Rs 500/- to Rs 1,000/-; and hence many a times, especially in patients below 40 years of age, the diagnosis of ulcer is made without this test, on the basis of patient's complaints, his/her life-style & physical examination. Based on this 'clinical-diagnosis,' anti-ulcer treatment is given. Gastroscopy is done later, if there is no relief after standard anti-ulcer treatment. In patients above 40 years of age, however, gastroscopy is necessary before starting any treatment, since at this age, there is a possibility of cancer of the stomach and this has to be ruled out before starting anti-ulcer treatment.

Is there any pain or any other trouble while undergoing gastroscopy ? Is it safe ? :

Gastroscopy is a trouble-free, risk-free investigation. The throat is numbed by local anaesthetic spray or by swallowing an anaesthetic liquid, so that there is no pain while swallowing the endoscopic tube. A sedative injection is given if the patient is very anxious. The additional advantage of gastroscopy is that if necessary, a part of the suspicious-looking lining can be taken out (biopsy) painlessly for examination under a microscope. Such microscopic examination of this tissue is necessary for the diagnosis of diseases like : cancer. This biopsy does not cause pain and takes only 10-15 minutes.

X-ray examination is some times advised for diagnosis of ulcer; Why ? :-

Ulcer can be diagnosed by a special x-ray examination called Barium-x-ray. In this examination, the patient suspected to be having an ulcer is given a glass of 'Barium-meal' to drink which is radio-opaque because it contains a Barium-salt. This barium salt, sort of paints the internal lining of

the gastro-intestinal track and when an x-ray is taken the GJ track is visualized very well on the x-ray film. If there is an ulcer, it is seen on the x-ray film. This test is also painless, but endoscopy is more reliable as in endoscopy the ulcer is seen directly, whereas in x-ray it is seen in the x-ray photograph. However, endoscopy requires a trained, experienced specialist.

The x-ray of barium has the advantage that it gives a permanent photo-record of the ulcer which can be seen indirectly by other experts and can be compared later with a subsequent Barium-x-ray. Endoscopy can also be recorded on a video-film. In India, this facility is now available in some places, but costs around Rs: 1,000/- extra. Such record is usually not necessary.

What happens, if ulcer is not diagnosed early enough ?

As diagnosis is delayed, the ulcer increases in size and depth. Ultimately the ulceration may eat up the whole depth of the stomach, or duodenum to cause a perforation in the wall at the ulcer-site. Secondly, untreated ulceration may erode a blood-vessel and this may cause heavy bleeding. Both these complications are life-threatening and specialized treatment including surgery is urgently needed. Such urgent surgery is of course, riskier than planned surgery.

A long standing ulcer is the stomach, especially at an older age can become cancerous. (=Duodenal-ulcer, however, never becomes cancerous, and ulcer of the oesophagus rarely becomes cancerous).

It would thus be clear that it is necessary that ulcer is diagnosed early enough.

What is the treatment of ulcer ?

Until a few years back, the treatment of ulcer was cumbersome to the patients and the results unsatisfactory. It consisted of heavy, frequent doses of antacid tablets or liquid, which would neutralize the acid in the stomach. Such treatment was prolonged and repetitive. A lot of dietary restrictions were also required. However, now, the treatment of ulcer has been revolutionized during last decade.

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Now ulcer is treated with two types of drugs : (1) those which almost stop the secretion of hydrochloric acid. When there is no acid or pepsin, ulcers heal in 6-8 weeks. These acid-inhibitor drugs include ranitidine, famotidine and Omeprazale. Omeprazole is the drug of choice if the ulcer is in the oesophagus. (2) drugs which act against the bacterium called H.Pylorie. We have seen earlier that this bacterium reduces the mucosal resistance to hydrochloric acid. These drugs are indicated especially when ulcer recurs after treatment with the acid-inhibitor drugs. For a simple case of ulcer a course of 6 to 8 weeks of acid-inhibitors is needed. The doctor would decide if the acid-stopping drugs is needed for more days and whether, in addition, drugs to eliminate H.Pylorie are needed or not. These drugs are to be given only for ~~about ten~~ seven days.

If the ulcer is in the stomach, after the course of acid-inhibitors, gastroscopy is needed to ensure that the ulcer has healed. If it has not healed, this may be because it is cancerous. No such endoscopic examination is usually needed after treatment of duodenal or oesophegeal ulcer as the former never turns malignant and the latter rarely becomes malignant (cancerous).

In case of ulcer of the oesophugus, omeprazole is preferred. To prevent the reflux of contents of stomach, into the oesophagus causing 'water-brash' and heartburn, a drug called Cisapride is also given. Raising the cot on the head-side by putting wooden blocks below the cot on that side helps to prevent this regurgitation during sleep.

The following simple exercise may also help to correct the defective 'valve' between the oesophagus and the stomach by strengthening the concerned muscles. : Inhale fully by pushing the abdomen forward. While exhaling, allow the abdomen to retract and at the end of exhalation, actively retract it furtherest by drawing inwards and upwards the upper abdomen to the full. Do this twenty to thirty minutes at a time, thrice a day. This abdominal breathing also tones up parasympathetic nervous system thus helping reduce acid secretion. This abdominal breathing is in fact the natural breathing and is seen in children. As we grow up in the tension-filled world, this natural pattern is changed to 'emergency,' chest-breathing, which is associated with increased acid-secretion.

Do anti-ulcer drugs have any side-effects ?

Acid inhibitors are generally devoid of any side effects. If taken for prolonged period, some people develop indigestion.

The drugs which eliminate H.Pylorie may cause nausea, vomiting, diarrhoea. If you experience any side effects, consult your doctor.

Are there any dietary or other restrictions to be followed ?

Eating habits should be regular. Do not fast. The stomach-seretions, are released by reflex action at the accustomed time. If there is no food in the stomach then the acid and pepsin, erode the lining of the stomach and the duodenum.

Tobacco in any form must be totally stopped. So is the case with alcohol. Chillis and spices should be cut down.

Keep away from tension causing situations and relations and learn to take things easy.

It should be borne in mind that acid-inhibitors will help heal the ulcer. But if after this treatment, there is excessive secretion of acid once again due to psychological tension, or wrong habits, the ulcer will recur. Hence it is quite essential to have good, regular habits and to keep away tensions. Otherwise the ulcer disease continues to recur requiring repeated, prolonged drug treatment; and the risk of complications remains.

Why do some patients have to undergo surgery for ulcer ?

Despite proper drug-treatment, if there is no relief, surgery for ulcer is required. However, now a days, this rarely happens because, the currently available anti-ulcer drugs are very effective. If treatment is not properly taken or not taken at all, complications like bleeding from the ulcer or perforation occur requiring urgent surgery. Now a days, operations are restricted only for such rare events.

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If drug-treatment is taken haphazardly, the affected area may contract, leading to the narrowing the food-pipe. To enable food to pass forward properly, surgery is needed to relieve this narrowing.

In case of inadequate or delayed treatment of gastric (stomach) ulcer, it may turn cancerous. In that case a big operation is required.

However, in general, thanks to the new powerful, appropriate drugs, the need for surgery has come down drastically in recent years.

Can ulcer be completely cured ?

In many patients, a 6 to 8 weeks course of acid-inhibiting drugs cures the ulcer. If this does not occur or if ulcer recurs, then alongwith the powerful acid-inhibitors omeprazol for one month, other drugs (amoxycillin like antibiotic, metronidazole) are given for a week. These antimicrobials eliminate H.Pylorie and the ulcer is generally cured. However, in a few patients, it recurs. In such cases, there is generally some cause- irregular, wrong food habits, psychological pensions, indulgence in tobacco, alcohol, or ulcer-causing drugs like : aspirin, brufen, steroids, etc. Unless any such cause is removed, ulcer will recur. It should be clear that treatment of ulcer does not consist of mere drug therapy.

GAS - TROUBLE ... ?

What is exactly meant by gas-trouble ?

Many complain about 'gas-trouble' or 'indigestion'. A variety of complaints are included in this-restlessness or feeling of bloating in the stomach, burning in upper abdomen, ragurgitation of food and heart-burn, excessive burping, sense of incomplete evacuation after stools, excessive passage of gas, foul eructations.....all such complaints are called as 'indigestion' or 'gas-trouble.'

The first thing to note is that genuine indigestion due to inadequaty of digestive juices is very rare and most of the complaints mentioned above occur due to problems other than indigestion as such. True indigestion can occur due to: (i) alcoholism--alcoholism leads to inflammation of the stomach lining (gastritis) and of the pancreas. Hence stomach and pancrease produce less of their digestive juices causing indigestion as such. (ii) Rarely, gall-bladder stone may put pressure on the nearby pancreatic duct which carries pancreatic digestive juice to the intestine. Less of pancreatic juice thus reaches the intestine for digestion and this causes indigestion. Indigestion occurs in some other rare disorders. In case of deficiency of digestive juices, the stools are bulky and float in the water, and there are other symptoms due to the main disease. In contrast, the usual 'indigestion' is not due to deficiency of digestive juices but is caused by inflammation of the internal lining of the stomach and intestine. It leads to symptoms described above except those related to passage of stools.

Why does gas trouble occur ?

Inflammation or irritation of the internal lining of the stomach is the main cause of 'gas-trouble.' The internal lining of the stomach secretes the digestive juices-hydrochloric acid and pspsin. Irritation of this lining due to the excessive secretion of these juices or due to

irritants like excessive chillis, tea, coffee etc. causes symptoms towards 'gas-trouble.' Irregular diet and sleep, psychological tensions, tobacco, alcohol increase the acidity and hence this irritation. Repeated consumption of alcohol is especially likely to cause stomach irritation. Due to this irritation, the stomach becomes very sensitive and even normal, usual food causes a sensation of bloating in the stomach or other symptoms, mentioned above, commonly called as 'gas-trouble,' or 'indigestion.'

Some patients feel that excess gas has accummulated in the stomach and get relieved after burping. In reality, there is no excess gas in the stomach. In all healthy persons, some air is swallowed during eating or drinking; and this does not cause any trouble. But if the internal lining of the stomach has become extra sensitive due to irritation, even this normal amount of swallowed air causes a sensation of bloated stomach.

If there is repeated burping, this means excess of air has been swallowed. While smoking cigarrettes a bidis or while drinking tea/coffee frequently, especially from the saucer, excess air is unconsciously swallowed. An anxious person also unconsciously swallows excess air. If any of these causes is identified, this type of 'gas-trouble' can be prevented by appropriate behavioural change.

What is the treatment if the gas-trouble is due to irritation of the stomach-lining ?

The cause of irritation of stomach-lining, hyper-acidity has to be found out. As mentioned above, tobacco, alcohol consumption, excessive tea/coffee, spicy-food, aspirin-like painkiller drugs cause this irritation. Avoidance of these irritants would relieve the gas-trouble. If there is no relief, consult your doctor. The doctor would give an antacid which neutralizes excess of acid and has a soothening effect on stomach lining. Popular antacid brands are: Gelucil, Digel. Antacids must be taken in adequate dosage. Two tablets or 1 teaspoonfuls 4 to 6 times a day. A medicine called dimethicon (brand name : Dimol) 1 2 teaspoonfuls helps to bring out swallowed air.

while burping out the swallowed air, —
the mouth should be closed, keeping only a small gap between the lips for the air to escape out. Otherwise, with a widely open mouth, burping leads to more air being swallowed than coming out.

A simple home remedy to take out swallowed air is to mix a quarter teaspoonful of soda-bicarb with a cup of water, and a few lemon-drops, and drink it. (Any readymade carbonated drink would also do). Bubbles of carbon-dioxide come out of the stomach and the trapped air also comes out alongwith it. This gives relief. But some times instead, the carbondioxide also gets trapped in the stomach. If this occurs, the gas-trouble instead becomes more pronounced. It is, therefore, better to use dimethicon.

If the symptoms of hyperacidity are severe, or if there is no relief from simple measures mentioned above, doctor will advise a course of an acid-inhibitor drug : ranitidine for 6 to 8 weeks. Wrong habits mentioned above, should also be changed.

A warning is needed here : uneasiness, pain in upper abdomen can rarely occur due to a heart-attack ! Hence if gas-trouble is not relieved by antacid, or by soda-bicarb or by burping etc., consult your doctor immediately.

Can gas-trouble occur due to any other reason ?

Yes, the important causes are as follows-

Diarrhoea : Ill-formed stools or increased frequency, mucus in stools, sense of incomplete evacuation after passing motion,-such complaints are also called as 'indigestion' or 'gas-trouble.' Generally, this problem occurs only for a couple of days and then stops on its own. If it continues, take doctor's advice. This is because, this continued trouble is due to some infection of the GI track and appropriate drug is required to treat this infection.

'Indigestion' is sometimes accompanied by foul eructations. But they generally stop in a couple of days & no drug treatment is generally required.

Excessive gas per rectum : This is one more type of 'gas-trouble.' Let us see why this occurs.

In the large intestine, about 7 to 10 litres of gas is accumulated every day. A part of it consists of swallowed air reaching upto the large-intestine; another part comprises of the gases diffused from the blood-vessels lining the intestinal mucosa; and the remaining is formed in the intestine. This gas is mainly made up of methane, carbondioxide, and hydrogen; and hence is generally devoid of foul smell. A large part of this gas is absorbed back into the blood and during 24 hours, in a normal person, about half-a-litre is passed per rectum, a little at a time, 10-12 times in 24 hours.

When more gas is passed out than this, it is because of increased peristaltic movements of the intestine, because of which the gas does not get adequate time to get absorbed into the blood and hence more than usual amount passes out through the rectum. This increased peristaltic movements are due to increased sensitivity of the intestinal lining due to some irritation or infection. A hypersensitive intestine pushes forward its contents more rapidly. One consequence of this faster motion is that partially digested food enters the large intestine, instead of after completing the digestion. Bacteria in the large intestine act upon this partially digested food, and in the process produce foul smelling gas. Foul smelling gas is also occurs when certain dals are consumed, which release the foul smelling hydrogen sulfide gas due to the action of bacteria in the large-intestine on these dals.

To prevent such foul-smelling gases, one has to find out, which kinds of foodstuffs cause this type of gas in a particular person. Secondly, intestinal irritation or infection has to be treated to reduce the increased peristaltic movements of the intestines.

Constipation can also cause gas-trouble. To prevent constipation, consume adequate amount of green leafy vegetable, other vegetables, fruits and water. Secondly, at least some exercise should be done every day, especially by those who have a sedentary life-style. Simple long, walk every day relieves gas-trouble of many people.

Many medicines for gas-trouble are advertised in the lay-media. Should these be used ?

It should be clear from the discussion so far, that gas-trouble is mainly due to wrong habits. If they are changed, gas-trouble would disappear. Use of Antacids and dimethicon are useful temporary measures. But the real solution lies in leaving unhealthy habits. But some of the drug-companies promote certain drugs through aggressive advertising, as 'remedies' for 'gas-trouble'. Some doctors also prescribe 'enzymes' to increase digestion. But all such medicines are wasteful; and by & large ineffective. There is hardly ever a deficiency of digestive juices leading to gas-trouble. Secondly, most 'digestive' enzyme preparations contain enzymes in too small a quantity. Most proprietary 'Ayurvedic' preparation that are advertised in the lay-media are generally useless. However, tablets like 'Gasex' contain activated charcoal, which absorbs gases to a certain extent; and this may give some relief. Yet, if the gas-trouble occurs repeatedly, it's underlying cause has to be found out and treated.

In conclusion, it is to be borne in mind that one has to first identify exactly, what one means by gas-trouble and to find out the underlying cause to be remedied. To suffer unnecessarily, or to merely keep on taking medicines is not adviseable.

Part - II

REVIEW OF LITERATURE ON CYANIDE POISONING AND TREATMENT

SYMPTOMS : Early stages of acute poisoning resembles an anxiety state with headache, giddiness, excitement and tachycardia. Tachypnoea is a sign of low cyanide concentrations. In severe poisoning drowsiness, coma and convulsions precede death. Reduced oxygen consumption diminishes the arteriovenous oxygen content difference, rendering the retinal artery and vein a similar colour. Palpitations, hypotension, pulmonary oedema and hypoxic ECG changes can occur and a smell of burnt or bitter almonds may be detected on the breath.[1] (The ability to detect such a smell is, however, genetically determined and lacking in a large fraction of the population.[6] In one test 3 out of 5 pathologists and 9 out of 11 members of a biochemistry department could not identify cyanide by smell.[2])

Clinical abnormalities can occur when cyanide exposure is high or when there is abnormality of detoxification or when there is a combination of both factors. Abnormalities in detoxification may arise from paucity of substrate arising from malnutrition. Such situations can give rise to tobacco amblyopia, Leber's hereditary optic atrophy, inherited optic atrophy and subacute combined degeneration of the cord.[4*]

LETHAL DOSAGE AND BLOOD CONCENTRATIONS : The minimum lethal dose is 0.5 mg/kg of body weight and the minimum lethal concentration in air is 0.2 - 0.3 mg/1 (200 - 300 ppm).[7*] Oral ingestion of 250 mg of cyanide salt is usually fatal within minutes as is inhalation of 50 ml (1.85 mmol) of HCN gas.[1]

A blood cyanide level of greater than 0.2 microgram/ml is considered toxic. Acute toxicity may occur as blood cyanide concentrations approach 0.5 micrograms/ml. Fatalities are usually associated with concentrations exceeding 1.0 microgram per ml (John D. Bauer, Casarette & Doull).

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* See also additional notes at the end.

TOXIC ACTION : Cyanide has a high affinity for the ferric ion of cytochrome oxidase (a_3) within the mitochondria. By combining with the aa_3 complex, cyanide prevents O_2 from reoxidizing reduced cytochrome a_3 thus inhibiting electron transfer and preventing both oxidative phosphorylation and oxygen utilisation and cellular respiration (for conversion of glucose to energy).[1] As a result of the inhibition of oxidative phosphorylation, mitochondrial O_2 utilisation ceases and arteriovenous O_2 differences are abolished. The loss of ATP generation in the mitochondrial electron transport chain evokes anaerobic metabolism (Pasteur effect). This increases lactic acid generation leading to lactic acidosis. The buffering of lactic acid leads to a progressive fall in plasma bicarbonate concentration. All organs are affected; eventually there is central nervous system anoxia and finally, death.[2]

Cyanide may also have a direct, though reversible, toxic effect on pancreatic P cells resulting in hyperglycaemia.[2]

DETOXIFICATION : The major pathway for detoxification is the formation of thiocyanate from the combination of sulphur with the cyanide-cytochrome complex by the enzyme thiocyanate oxidase in the liver. The respiratory enzyme is released and thiocyanate undergoes renal excretion. The rate limiting step is the production of sulphur from the limited body store of thiosulphate by the rhodenase catalysed reaction which may also explain recurrent and prolonged toxic symptoms despite antidotal therapy.[1,10]

An alternative pathway is the conversion of hydroxocobalamin (Vitamin B_{12a}) by cyanide ions to cyanocobalamin (Vitamin B_{12}), which undergoes renal excretion, and to hydrogen cyanide which is excreted via the lungs.[1]

ANTIDOTES : The intrinsic toxicity of antidotes should be carefully considered in case the diagnosis of cyanide poisoning proves to be erroneous. It must also be borne in mind that as of 1977 in only 4 out of 61 cases reported in the last 100 years, was the magnitude of poisoning by cyanide documented quantitatively, and that inferences of a causal relationship between antidotal use and successful outcome were till 1977 based on such data.[2] The situation has improved somewhat subsequently.

SODIUM NITRITE : This oxidises nearly 30 - 35% of blood haemoglobin with a ferrous (2^+) ion to methaemoglobin (M-Hb) with a ferric (3^+) ion which has a greater attraction for free CN^- ions than does cytochrome oxidase. M-Hb thus binds the CN^- ion to form cyanomethaemoglobin thereby decreasing CN^- combination with cytochrome oxidase. The weak CN-MHb bond allows the slow release of cyanide within the liver, where increased sulphur requirements for detoxification are met by exogenous thiosulphate which has slower tissue penetration than cyanide. Normal M-Hb blood levels are 1%, and 300 mg of sodium nitrite will produce a level of 10% when cyanosis will appear. The optimum theraputic M-Hb level of 25% may be increased to 40% if symptoms of cyanide poisoning are severe. Greater levels may produce anoxia, coma and death.[1]

Although attended with low allergy risk, sodium nitrite has considerable intrinsic toxicity and its use in patients with cardiovascular collapse or vascular haemorrhage is hazardous. It involves a large sodium load and presents problems in monitoring therapy whilst maintaining near toxic levels of M-Hb. A child treated with nitrite died because of overwhelming methaemoglobinemia.[2]

Recent observation,[4,5] that the antidotal combination of sodium nitrite and thiosulphate with or without M-Hb formation were equally effective against cyanide poisoning has triggered investigations which seem to indicate that the antidotal action of sodium nitrite is due to vasogenic action rather than methaemoglobin formation.

SODIUM THIOSULPHATE : It combines with cyanide in the presence of the enzyme thiosulphate transulphurase (rhodanase) and O_2 to produce relatively nontoxic thiocyanate. It is relatively nontoxic although impurities in production may produce allergic reaction in 1 out of 1000 or 10000 persons. As a single agent its efficacy is about that of sodium nitrite but in acute poisoning accepted practice is to use it in combination with sodium nitrite, which increases its efficacy, in those patients for whom the diagnosis of overwhelming cyanide poisoning is clearly established.[1]

sodium thiosulphate + cyanomethaemoglobin

$$\xrightarrow[\text{thiocyanate oxidase}]{\text{rhodanase}}$$ methaemoglobin + thiocyanate

OXYGEN : Inhalation of 100% oxygen increases arterial PO_2 and increases tissue O_2 delivery. It may reverse the binding of cyanide with cytochrome oxidase and may also help increase the conversion of cyanide to thiocyanate by thiosulphate.[2] Oxygen can markedly enhance the efficacy of the nitrite - thiosulphate combination, so that oxygen should be made an integral part of antidotal combination in cyanide poisoning therapy.[3] Oxygen toxicity is unlikely with use over periods less than 48 hours.[1]

HYDROXOCOBALAMIN (VITAMIN B_{12a}) : It combines with cyanide forming cyanocobalamin (Vitamin B_{12}) but has limited protein binding and a short half-life of 5 min.[1] It has the overwhelming advantage that it is essentially nontoxic although in large doses it may produce facial acne. Thus even in the face of erroneous diagnosis or dosage, the patient is not at increased risk because of therapy. It has been called the most promising antidote.[2] For maximum effect it must be given in equimolar proportions that are approximately 50 times the ingested amount of cyanide.[7]

There seems to be disagreement over its use in combination with sodium thiosulphate. In reference 1 we find the statement that it is inactivated when mixed with soduum thiosulphate, whereas in reference 2 we find the assertion that in animal studies this agent is found to be especially useful when combined with thiosulphate.

COBALT EDETATE : It rapidly chelates free plasma and tissue bound cyanide producing cobalticyanide and monocobalt which are excreted within 24 hours renally. While not free from side effects, it can be employed in severe poisoning without close biochemical monitoring or reduction in oxygen carrying capacity.[1] High concentrations of cobalt salts have their own intrinsic toxicity, therefore, great caution must be exercised in their use.[2]

A simple chemical test on gastric aspirates to establish oral cyanide poisoning is described in reference 2. A detailed description of the mechanisms of cyanide toxicity and antagonisms is given in reference 5. Reference 8 deals with the treatment of cyanide poisoning by the administration of 4-dimethyl-aminophenol (DMAP) whose action is similar to that of sodium nitrite in that it helps in the oxidation of the ferrous form of blood haemoglobin to

to methaemoglobin. Use of pyruvate[3], mercatopyruvate[3-5] and chlorpromazine[3] as antidotes is also described in the literature.

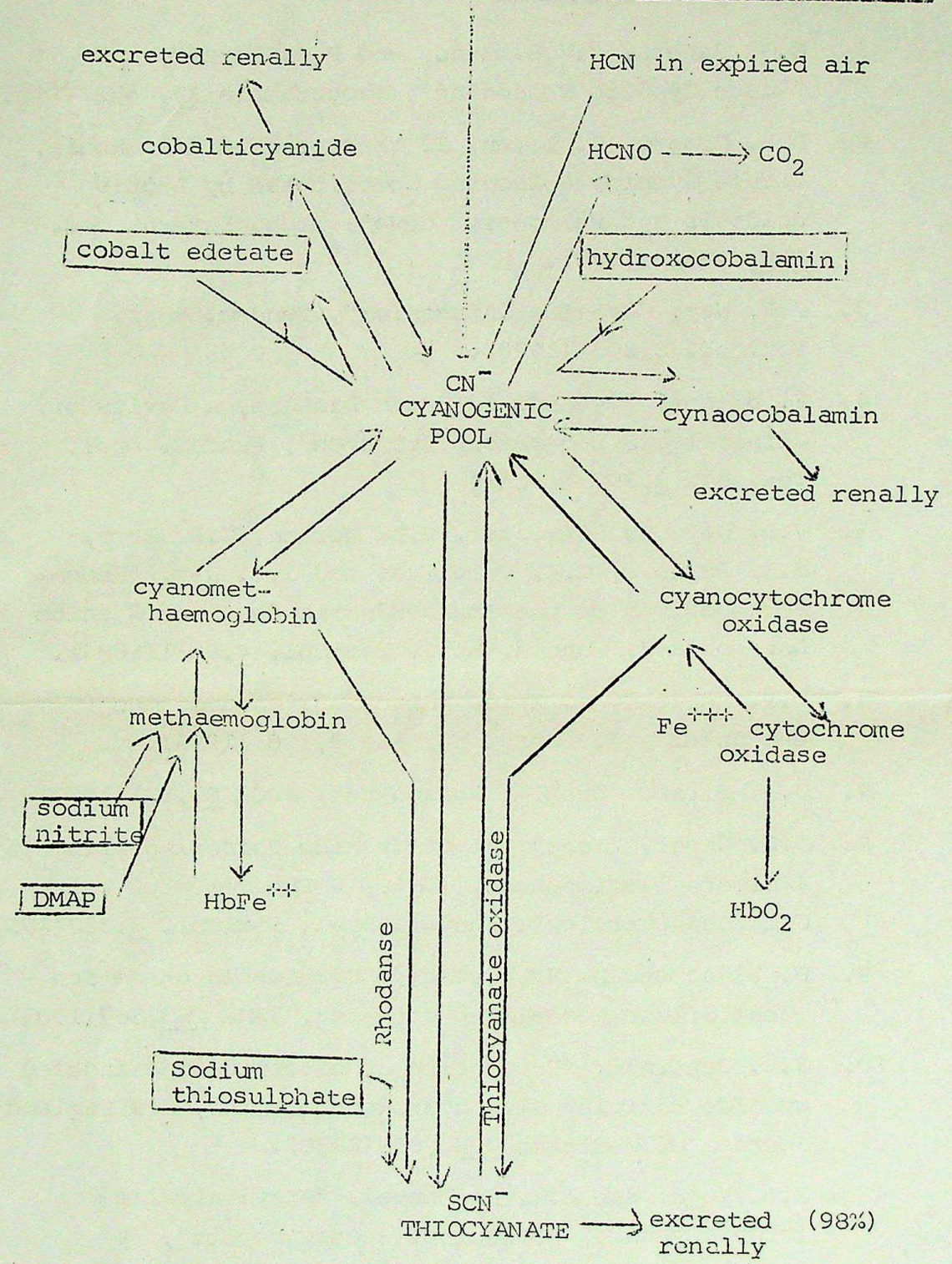
ADDITIONAL NOTES : The current OSHA exposure limit to hydrogen cyanide is 10 ppm (eight-hour time-weighted average) although a reduction to 5 ppm has been recommended. Short-term inhalation of air levels of 50 ppm HCN causes acute symptoms of gastric and respiratory tract disturbance; 130 ppm can be lethal. Lower doses, in the range of 10 to 20 ppm can cause complaints similar to those experienced at 50 ppm although longer exposure times may be required to elicit them.[9]

It has been suggested that vitamin B_{12} may be a protective factor in cyanide neurotoxic effects. Long-term cyanide intoxication leads to thyroid enlargement and interferes with iodine metabolism. It can also lead to weight loss, easy fatigue and sleep disturbance. It must be remembered that cyanide exposure inhibits a wide variety of enzyme systems in addition to the cytochrome oxidase system.[9]

Reference 11 contains a detailed account of the treatment of an episode of acute acrylonitrile poisoning. This is an important paper as it documents that a single incidence of acute cyanide poisoning can give rise to recurrent cyanide toxicity. As a result of this recurrence the patient required 15 treatments with sodium nitrite and sodium thiosulphate during a 72 hour period, along with additional therapy involving hydroxocobalamin and supplemental O_2, with constant monitoring of methaemoglobin levels. This case emphasizes that prolonged treatment of cyanide poisoning may be required and that many doses of sodium nitrite and sodium thiosulphate can be given safely over a prolonged period with adequate monitoring.

AGENT	MECHANISM		POTENTIAL TOXICITY
Sodium nitrite	$NaNO_2$ + Hb	M-Hb	Tachycardia, vomiting, hypotension, severe methaemoglobinemia, hypoxia, vascular collapse
	M-Hb + CN^-	CyanoM-Hb	
Sodium thiosulphate	$Na_2S_2O_3$ + CN^- (rhodanase)		none known
Oxygen	more O_2 in arterial blood more O_2 in tissues may reverse CN^- binding with cytochrome potentiates activity of sodium thiosulphate		Oxygen toxicity unlikely when used for less than 48 hours
Hydroxocobalamin	$OH-B_{12}$ + CN	$CN-B_{12}$	none known
Cobalt salts	chelates cyanide		significant loss of Ca^{++}, Mg^{++}, plus intense purgation, cardiac toxicity

UPTAKE OF CYANIDE (PER INHALATION, ORALLY, PERCUTANEOUS



FATE OF CYANIDE ION IN THE BODY

Adapted from:
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Focus on therapeutic endoscopy

Interview with Dr. V.G. Mohan Prasad, gastroenterologist.

"The therapeutic use of endoscopy, a revolution in treatment and cure, is yet to gain acceptance in India though it has tremendous potential," says Coimbatore-based **Dr. V.G. Mohan Prasad**, consultant gastroenterologist, hepatologist and endoscopist, who recently released a CD-Rom on therapeutic endoscopy. The CD-Rom, which details the procedures of various therapeutic uses of endoscopy, is a pioneering effort in the medical world.

Dr. Mohan Prasad has undergone training in the United States (Granada Hills Community Hospital, Los Angeles), Japan (Chiba University, Kyoto) and the Netherlands (Academic Medical Centre, Amsterdam), and is a member of the International Association for the Study of Liver and the American Gastroenterologic Association. He has published several research papers on liver diseases, dyspepsia, cancer and therapeutic endoscopy procedures. In Chennai, Dr. Mohan Prasad spoke to **Asha Krishnakumar** of the various types of jaundice as also ulcers, their causes and treatment methods, of pharmacological developments made in the treatment of liver diseases, and of the uses of therapeutic endoscopy. Excerpts from the interview:

► *What are the common liver disorders in India and how do they manifest themselves?*

Among the most common liver disorders are hepatitis, alcoholic liver diseases and liver carcinoma. Jaundice is an important manifestation of most liver disorders and is of three types: haemolytic, which occurs rarely and has a genetic predisposition; obstructive jaundice; and hepatitis. In haemolytic jaundice the red blood corpuscles (RBCs) swell and start breaking down into sclerocytes. This releases haemoglobin, which gets converted by the liver into a yellow pigment called bilirubin. The excess bilirubin enters the bloodstream giving a yellow colour to the sclera (the white fibrous outer layer of the eyeball) and urine. People with haemolytic jaundice have a higher chance of developing gall-bladder stones.

The 'obstructive' type occurs when there is an obstruction in the flow of bile. The bile secreted in the liver is taken to the gall bladder through bile ducts and stored there. The gall bladder releases it into the intestine through the common bile duct, to aid in digestion. A block in this pathway – from the liver down to the duodenum – can cause obstructive jaundice. Roundworm or stones in the gall bladder can migrate into the bile duct, run amok in the intestine and cause problems. Obstruction can also be caused by cancer of the bile duct, duodenum or pancreas, thus leading to obstructive jaundice.

Hepatitis ('hepar' means liver and 'ites', inflammation) or liver inflammation is the most common type. The immune system attacks liver cells, which then break down releasing bilirubin. This mixes in the bladder and the patient develops yellowness of the sclera.

► *What are the common causes of hepatitis?*

The most common cause is virus attack. Some 80 virus types can affect the liver and cause jaundice. But many are rare and some specific to certain regions such as Africa, Italy (Europe) and so on. A group of viruses called Hepatotropic viruses is very fond of the liver. Introduced anywhere in the body they find their way to the liver, multiply and live on for years. Six such viruses – A, B, C, D, E and G – are well-defined. While A and E are water-borne, B, C, D and G are parenterally transmitted just like the Human Immunodeficiency Virus (HIV), which causes AIDS, through improperly sterilised needles, shared razors, unprotected sex, homosexual activity and blood and blood products. But Hepatitis B, unlike AIDS, can be prevented and cured. Now effective vaccines are also available.

T.A. HAFEEZ

► *What is the incidence of hepatitis in India?*

We have a national carriage of 4 per cent, or 4.2 million. All carriers are not diseased people. Over 90 per cent of those afflicted with Hepatitis B, when treated, get cured completely. Only 5-10 per cent of the acute cases become chronic carriers. That is, they carry the virus after six months and also develop complications. It is this group that needs attention.

► *Has the development of drugs kept pace with the advances made in understanding the disease?*

For long, the only drug available for treating chronic Hepatitis B was Interferon, a biological response modifier. It is still the sheet anchor of therapy all over the world. But its cure rate is only 25 per cent. In 1992, I combined Riboviron with Interferon to treat chronic Hepatitis B patients. Riboviron, introduced in India in 1990, is a broad-spectrum antiviral and is effective against viruses in the ribonucleic acid (RNA) and the deoxyribonucleic acid (DNA).

This created ripples in the treatment of Hepatitis B. Even those who initially criticised me endorsed the treatment later. Now everyone agrees that there is the need for cocktail therapy.

I am convinced that if a chronic case of Hepatitis B is recognised early – before the liver gets cirrhotic, shrinks

and the patient develops portal hypertension and varices – the progress of the disease can be halted.

► *How serious are the diseases caused by other Hepatitis viruses and can they be cured?*

The D, or the Delta, virus is incomplete in the sense that it cannot survive in the liver without the surface coat of the B virus. So, if we take care of the B virus, D would also be taken care of. In India, the virus is very rare, existing only in some pockets.

The C virus is more common. It has a 1-1.7 per cent spread in the country. There is as yet no vaccine for the C virus as it mutates rapidly and a single vaccine cannot take care of it. Cocktail therapy can probably help.

Whether or not G virus is harmful is still being debated. But what is clear is that if C and G co-exist, the damage to the liver is very high. Not many commercial tests have been done in this field.

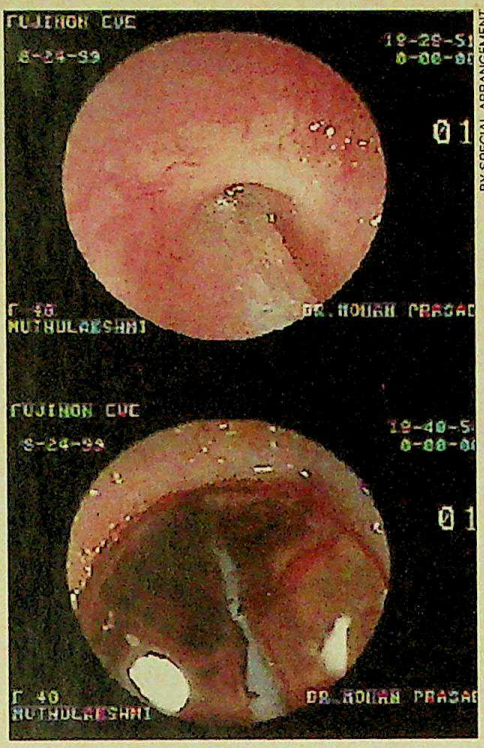
In India, A and E viruses (both water-borne) are the most common causes of jaundice. Fifty per cent of acute jaundice in adults is caused by the E virus. In children, 80 per cent of the cases are because of the A virus. If both attack together, the liver could be severely damaged and it could even lead to death. The A virus is dangerous for the aged and for infants, and the E virus for pregnant mothers. A third of the pregnant mothers affected by jaundice caused by the E virus die, and 50 per cent of the foetuses are also lost.

But Hepatitis A and E are easily preventable. If drinking water is boiled, chlorinated or purified before use, they can be prevented. Ultra-violet filtration or deradiation, and ozone treatment can also destroy the A and E viruses. It is important to use protected water not only for drinking but also in the preparation of chutneys, salads, juices and buttermilk.

The incubation period of these viruses is usually four to six weeks. There is a vaccine for the A virus now. But as it is genetically engineered, it is very expensive – Rs.1,000 for a dose (three doses have to be administered).

► *Why is the vaccine for the Hepatitis A virus expensive?*

The primary reason is that the A virus is not widely prevalent in the West, except in a few countries such as Italy. So, not much money has been invested on research into it. If India manufactures this vaccine, costs can come down dramatically. This is amply demonstrated by the production of the Hepatitis B vaccine by Shanta Biotech and some other companies. The genetically engineered recombinant Hepatitis B vaccine is now available for Rs.40-50 a dose for children.



BY SPECIAL ARRANGEMENT

Balloon dilation of a stricture in the oesophagus.

► *There is fear in the U.S. and Europe that the administration of the Hepatitis B vaccine causes autism and multiple sclerosis in children. Is this fear real?*

The International Viral Hepatitis Research Board has found no evidence to show that there is a relationship between the Hepatitis B vaccine and autism or multiple sclerosis in children. It has categorically stated that at this point of time there is no scientific basis to believe that there is a relationship between the two.

► *What are your other areas of interest?*

I am also working on the causes and treatment of ulcers. For long, it was believed that erratic timing of food intake or spicy/oily food caused peptic ulcers. Thirteen years ago, B. Marshall and Warren from Australia found a spiral bacteria, Helicobacter pylori, to be the cause of 90 to 95 per cent of duodenal ulcers. In the developing world, where its incidence is high, it spreads mainly through polluted water. The use of the Berkefield Candle Filter can remove this bacteria from drinking water. Ozonisation and ultra-violet deradiation can also kill the bacteria. If the bacteria get into the stomach, they stay there for as long as 30 years.

► *How can H. pylori be diagnosed?*

It can be done by using endoscopy and performing a biopsy of the stomach. Two methods are used for this. One is the "rapid urea test". Unfortunately, many endoscopists do not do this.

Other specialised histopathology tests can also identify H. pylori by scanning the stomach tissues. Although there is no vaccine for H. pylori, 95 per cent of the bacteria in an infected person can be eradicated by combining two antibiotics for a week. In six weeks' time, the patient would be completely cured. Thus, endoscopy has various uses.

► *Apart from diagnostics, what are the other uses of endoscopy?*

Endoscopy is commonly used for diagnostics. But it can be used for therapeutic procedures as well. There are over 4,000 endoscopists in India, but only some 50 use it for therapeutic procedures.

► *What are the therapeutic procedures that can be done using endoscopy?*

Several out-patient procedures can be done using an endoscope. For instance, cancer in the foodpipe produces strictures needing a dilatation – balloon or the rigid type. Endoscopy can be used for this. Stents can be put in the foodpipe using endoscopy. Early stages of cancer in the oesophagus can be removed using endoscopy. The method employed is called Endo Mucosal Resection (EMR). It is 100 per cent cure if treated early. Using endoscopy lasers can melt cancers in the foodpipe. Reflux diseases can be treated by chromoscopy using colouring agents such as Lugol's Iodine. Several surgical procedures in the stomach, bile duct and colon can be done using endoscopy. All these and many more can be done using endoscopy without anaesthesia. The only problem is that not many doctors do it as it is not taught in classrooms. Very soon we will have the endoscopy suturing device. This will be a revolution in the treatment and cure of several disorders.

► *How expensive is therapeutic endoscopy?*

It is expensive now. But if more people use it and if the required equipment is produced indigenously, costs will come down. Awareness and education on precautions and treatment methods is important to reduce diseases relating to gastroenterology, including ulcers. The public healthcare system should be strengthened in order to generate this awareness. ■

The Friend Of Tropics

Attempts have been made to replace DDT with organophosphates and other compounds. Though environment - friendly, the toxicity of the new materials can affect health workers handling them

BY T. R. THIYAGA RAJAN

RECENTLY, 92 nations met at Montreal to discuss DDT and other long-lasting chemicals. They decided to have a timetable for negotiations to reach a decision by 2000.

The meeting was sponsored by the UN Programme For The Environment (UNEP). The talks focused on 'the dirty dozen,' the 12 most persistent organic pollutants (Pops) which remain dangerous for years and can be carried along waterways for thousands of kilometers. They range from industrial chemicals, primarily insecticides like DDT, and pollutants created by industrial activity, like dioxin and furan. Should DDT be banned?

DDT (Dichloro Diphenyl Trichlro Ethane), a synthetic organic compound commonly used as an insecticide, was first prepared in 1874 by Othmer Zeidler, a German chemist. Zeider prepared it by the reaction of Chloral with Chlorobenzene in the presence of Sulphric Acid. But it was left to Paul Hermann Muller, a Swiss scientist, to recognise it as an effective insecticide for which he was awarded Nobel Prize in 1939. This highly toxic compound, functioning as a contact poison, kills a variety of insects by disorganising their nervous system.

This pesticide was extensively used by the Allies during World War II in the occupied areas to control the breeding of mosquitoes.

In USA, after the War a vigorous spraying programme was carried out resulting in a perceptible fall in the incidence of Malaria from an alarming number of 2.5 lakh cases per year to a fewer than ten in 1950. This compound not only controlled Malaria, but in combination with new crop strains (such as those developed by Norman Borlaug), it also led to dramatic increase in agricultural production.

DDT as an insecticide has come under scrutiny since the publication of the book *Silent Spring* in 1962 by American marine biologist, Rachel Carson. This book links the use of DDT as an agricultural pesticide to massive ecological disruption. Suspicion grew that DDT by entering the food chain and eventually concentrating in higher animals, caused reproductive dysfunctions such as thin egg shells in some birds. Some insect pests gradually developed DDT resistant strains whose population grew unchecked while their natural predators, such as wasps, were eradicated by spraying. In 1973 DDT was banned by the US government except for use in extreme health emergencies. This was soon followed by Canada, Sweden and Denmark.

But in tropical countries DDT still remains a crucial public health tool against mosquito borne diseases like Malaria, Filaria and Yellow Fever.

The detractors of DDT while agreeing with it's merits point out the damage it can cause on human endocrine system leading to cancer. But this finding is not conclusive. A recent study published in the *New England Journal of Medicine (Vol. 337, P. 1253)* amply disproves this theory.

It is feared that a total ban on DDT will deprive developing countries of a potential public health tool to combat insect borne diseases. Donald Roberts of Health Sciences University, Maryland, cautions saying that a bandoning DDT can result lead more Malaria cases. It is also pointed out that there is a sharp difference between DDT being used for mosquito control and agriculture. For instance, DDT was used on 2.15 lakh sq km of cotton in Guyana during a single growing season. Though several insects developed resistance to DDT, it's effectiveness could be restored partly by using it with certain chlorinated hydrocarbons.

But environmentalists say that a total ban on DDT would force the health workers to explore other alternatives involving lesser risk to control insect borne diseases. "This has to be pursued in all seriousness" says Tryggvi Felixson of Iceland Ministry of Environment. Nordic Governments are insisting upon the ban as many persistent chemicals that evaporate in the South are carried away to North, polluting the atmosphere.

Attempts have already been made to replace DDT with organophosphates and other environment-friendly compounds. Though they fail to pollute the environment, their toxicity is a health hazard for workers them. Says Pushpa Herath of WHO: "Until we have enough alternatives, DDT is the only choice."

It is also suggested that non-chemical methods such as mosquito nets and biological means as predatory fish may be tried to control mosquito breeding. While lauding these methods, health workers claim that these measures cannot be implemented in the field. "Experience has shown that when there is pressure to make changes it yields positive results. We have seen that with the ozone depleting chemicals. We must not have an agreement with the lot of loop holes. There would be no incentive to find alternatives," says Felixson.

Some countries have already discontinued or restricted it's use due to pressure brought in by the foreign aid organisations. As traces of chemicals appeared in agricultural exports, developing countries like Indonesia and Bangladesh have already banned DDT fearing endangerment of their international trade links. Another reason is DDT earmarked for Public Health purposes finds it's way into the farmers,' fields due to flourishing black market trade, in pesticides.

Detractors also point out the serious health hazard that the Arctic women are facing on account of unchecked use of DDT. It had been detected that the DDT concentrations in their breast milk and umbilical cord blood far exceed the recommended safe levels. It has been recorded that the accumulated chemicals get condensed in the Arctic air and fall as rain, moving up the long marine food chain. Thus, Arctic people are exposed to serious health problems.

Health watchers clearly see the writing on the wall. Already several companies have cut down production fearing stoppage of foreign aid. Hence it is viewed that there is no necessity to officially ban DDT. It is also hoped that in the event of a total ban, the UNEP's package may include adequate funds for developing countries to explore alternatives to DDT.

While huge funds are earmarked by developed countries to fight diseases like cancer and AIDS, which are widely prevalent there, scant regard is shown for tropical diseases such as Malaria and Filaria. Health workers who have used DDT as a public health weapon shudder at the prospect of giving up something that has worked successfully for four decades.

(The author is Retired Chief Chemist in National Polio Laboratory, King Institute of Preventive Medicine, Chennai)

Black Stripe Moth Trap

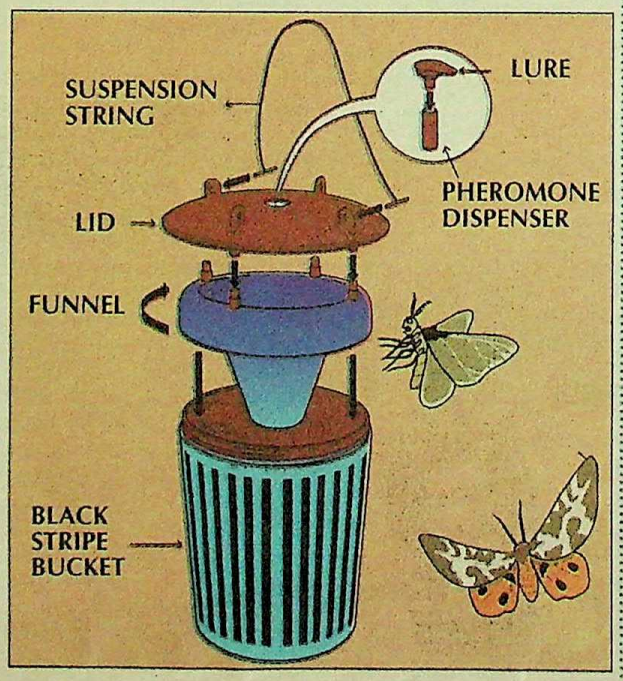
BY S. MOHAN AND K. ASAF ALI

SEVERAL models are used to monitor moth pests in stored foodgrains. A special trap has been developed for *Ephestia cautella* and *Plodia interpunctella*. Called the Black Stripe Moth Trap, it makes use of pheromone and black stripes to attract stored pests.

The trap consists of a black stripe bucket, yellow funnel, greenlid and `suspension string'. Pheromone in the trap attracts *Ephestia sp.* and *Plodia interpuctella*. The black stripes act as a visual attraction to maximize the catch.

The stripes replicates exactly the stripes in wings and dorsal side of the abdomen of the moth. This is the special feature of this trap. These traps can be used in flour mills, bulk food storage areas and food processing areas. There should be at least one trap per 600 cubic meters and should be hung as this is convenient for inspection.

(The authors are from the Dept of Agricultural Entomology, Tamil Nadu Agricultural University, Coimbatore)



Growing Cotton Through Tissue Culture

SCIENTISTS in Delhi have identified for the first time a strain of a popular south Indian cotton variety that can be multiplied through tissue culture. Generally most cotton varieties, including Indian ones, cannot regenerate through tissue culture, except for the "coker" variety which is not economically important, reports PTI.

So far scientists have tried to coax cotton varieties into regenerating through tissue culture by crossing them with "coker" which helps transfer the gene for this trail from coker plants. Though the approach did yield results, it "would be more useful if these varieties could be regenerated without any input from coker," Deepak Pental, who led the team from Delhi University South Campus, said.

The team has identified some genetic types (called genotypes) in the popular south Indian cotton variety "MCU-5" that regenerate profusely through tissue culture in just six months. They selected a few genetic types within the MUC-5 strain for tissue culture.

The scientists grew tiny pieces of hypocotyl – the region below the cotyledon – on a special nutrient medium to which growth regulators, vitamins and salts were added. The tiny pieces called "explants" directly formed embryos, about one third of these embryos sprouted shoots and roots to form small plants that could be grown in pots.

Steps To Combat Asthma

BY DR DWARAKANATH

AN expert group has been formed to tackle the problem of bronchial asthma which is assuming dangerous proportions, especially in developing countries. The group, Global Initiative For Asthma (GINA), constituted by the National Heart Lung and Blood Institute and the World Health Organisation has formulated guidelines for diagnosing, assessment of severity and treatment methods.

Here are a few guidelines:

Signs for suspected asthma

❑ If there are recurrent attacks or even a single attack of breathing, especially while breathing out or cough during night or early morning.

❑ Sleepless nights because of cough or breathlessness.

❑ Breathlessness or cough after physical exercise and during particular seasons of the year.

❑ Cough, wheezing or chest tightness when exposed to dust or irritants in the air.

❑ Repeated attacks of cough and cold which does not abate quickly.

❑ Relief when broncho dilators are used.

Diagnostic difficulties

Children commonly suffer from cough, cold and breathlessness due to various viral, upper respiratory tract infections, sinusitis or adenoids.

But if these conditions persist even after 10 days or the child wakes up from sleep, it can be a suspected case of bronchial asthma.

In infants it is very difficult to diagnose asthma because they have the same symptoms even for acute viral respiratory infections or bronchiolitis which may respond to anti-inflammatory drugs than antibiotics.

Smokers frequently suffer from same symptoms of asthma though they may be having chronic obstructive pulmonary disease. Stopping smoking helps in relieving symptoms.

As it is difficult to conduct pulmonary Function Tests (PFT) in elderly people, they are treated for bronchitis or emphysema though they may be suffering from asthma.

Workers exposed to inhalant chemicals or allergins may develop asthma which could be wrongly diagnosed as chronic bronchitis. Hence, pulmonary function tests should be done before employment and periodically.

In people who get asthma attacks only during certain seasons, cough at night or recurrent respiratory infections, diagnosis becomes difficult.

How to diagnose Bronchial Asthma?

Diagnosis depends on measurement of expiratory flow rate. For this, either a spirometer or peak flow meters are used. Spirometer is to measure Forced Expiratory Volume (FEV) at the end of first second. Peak Flow Meters (PFM) are more popular because it is compact. A patient is made to blowing forcibly into the mouth piece of PFM. The results are compared with FEV and standard readings which depend on height, age, sex and race of people. Bronchial Asthma is diagnosed if:

❑ Peak Expiratory Rate (PER) is greater than 15 per cent of the observed value after inhalation of a short acting bronchodilator or after a course of corticosteroid and bronchodilator.

❑ Variation of peak expiratory flow values more than 20 per cent if not on bronchodilators.

❑ Decrease of 15 per cent or more in PEF after exercise or running.

Severity of Bronchial Asthma

Severity of asthma is grouped into four steps. One is the least severe form called `Intermittent step' and step four is most severe called severe persistent asthma. Steps two and three are mild and moderately persistent asthma respectively. This grouping helps in formulating treatment plan. For instance, if a patient has intermittent symptoms less than once a week or occasional attacks and normal lung functions in between, he comes under step one and needs intermittent quick relief drugs or inhalations only during attacks. But a patient is in step four i.e. having severe persistent asthma, needs multiple daily long term preventive medications high doses of inhaled cortico steroids and long term steroids.

This stepwise approach to asthma not only helps in controlling asthma but also assures uniformity in treatment.

Control of asthma

To know that asthma is under control, we have to assess patients both symptomatically and clinically. A patient should be maintained at the state where the symptoms and attacks of asthma are minimal, no emergency visits to hospital, minimal drugs and full physical activity, Variations of Peak Expiratory Flow less than 20 per cent with a normal Peak Expiratory Flow rate .

If this state is maintained for at least three months the patient steps down to lower category of treatment. Otherwise, the patients' treatment has to be reviewed and factors which aggravate bronchial asthma have to avoided.

What types of medications are available for Asthma?

Asthma patients basically need two types of medications i.e. quick relief and long term preventive medications.

Quick relief medications are used for a short period. They are known as case Beta 2 agonists because they block receptors of Beta type 2. This results in dilatation of bronchial tree relieving symptoms of breathlessness, cough and choking sensation. Beta 2 Agonists are available as tablets, syrups and inhalers and act for about 4 to 6 hours. These should be used for quick relief and not as long term routine medications. If these medications are needed more often it means asthma is aggravating. Inhalers cause less systematic side effects.

For a long term relief, drugs like cortico steroids, sodium chromoglycate or nedocronil sodium are used. These are anti-inflammatory drugs which prevent allergic reactions when used on long term. However, these take long time to act. Hence short acting bronchodilators should be combined with long term medications for better control.

Different forms of therapy are advocated by several groups who practise herbal, homeopathy, acupuncture and ayurveda. As patients of bronchial asthma are likely to try any of these forms, caution should be exercised since there is no research that proves their superiority.

How to manage asthma attacks in the house?

Proper use of medications can help control asthma. One should be able to recognise the aggravating symptoms of asthma like increase in cough, chest discomfort and wheezing during nights. The patients are to be treated with Beta 2 agnosists – inhalers or tablets or syrups. Response is is good if the symptoms subside in four hours. If not, the patient will have to take corticosteroid tablets or syrups at home and see a doctor immediately.

Cold or viral infections can also trigger asthma, but more commonly in children. Hence, early treatment of such conditions are necessary along with treatment with Beta 2 agonistes and or cortico steroids. Immunisation against influenza also reduces the chances of infection.

For most asthmatics, exercise or physical activity can trigger an attack. Unlike other triggering factors, this needs not be avoided because with proper treatment and pre-treatment with Beta 2 agonist, the attack can be prevented. Regular exercise in fact reduces symptoms.

All these require a change in lifestyle, readjusting houses and working environment for a long term prevention or at least to reduce the severity of symptoms and the quantity of medication.

Role of patient education

The key to success of every aspect of asthma is patient education. This also helps patients and parents to understand more about the disease, triggering factors and aggravating symptoms so that prompt action is taken at proper time.

Following points should be stressed during patient education.

❑ To take medication regularly as advised.

❑ To know the different types of drugs given and their basic actions.

How to prevent asthma attacks?

The main goal of asthma control is to prevent attacks. This can be achieved by medications, avoiding triggering factors, patient education and careful monitoring of the patient.

The factors that precipitate attacks of asthma have to be carefully identified and removed from the patients environment. The commonly known triggering factors are domestic mites, tobacco smoke, animal furs, cockroach, pollen and mild allergins. Other factors could be wood burning stoves, respiratory infections and physical activity.

House dust mites or domestic dust mites are small, invisible to normal eyes and are found in mattresses, blankets, rugs soft toys and stuffed furnitures. They breed fast in damp and humid weather. Exposure, especially in childhood to mite, contributes to the development of asthma, which can be avoided by washing bed linens or blankets once a week in hot water and dried thoroughly. Avoid carpets stuffed furniture in bedrooms and wash curtains and soft toys regularly.

Animals such as small rodents, cats and dogs can trigger asthma attack by the allergins present in their fur. This can be avoided by removing them from home or away from sleeping areas. If this is not possible, washing them regularly may help to some extent. Cockroaches can also trigger asthma. Houses should be thoroughly and regularly sprayed with pesticides to get rid of cockroaches. For long term asthma control, a written management plan is issued to patient. This classifies patients to different zones based on their symptoms severity, treatment to be taken and further steps to be taken. Green Zone means patient is having asthma under control with no symptoms or interruption of activities or sleep. If patient continues in green zone for three months, patient can be advised step down treatment.Yellow Zone means mild symptoms of asthma are present and needs to be cautious.

The patient may have to increase medication on consultation with doctor so that patient goes back to green zone.Red Zone means symptoms are more pronounced and asthma is getting out of control. Hence with these symptoms the patient should seek medical help immediately so that his condition reverts back to yellow and then to green zone.

In short, Asthma Management Zone System is the written guidance for long term control of bronchial asthma.

(The author is Deputy Chief Medical Officer at ITI Hospital, Bangalore)

Patient education is vital for asthma. This helps patients and relatives to understand more about the disease, triggering factors and aggravating symptoms so that prompt action can be taken.