DIS

NON-TRANSMISSIBLE HEALTH PROBLEMS

PROBLEM	SOME CAUSES
ACCIDENTS	IGNORANCE — LACK OF CARE — FATIGUE — RISK TAKING — CHANCE
HEART DISEASE	INCORRECT DIET — STRESS — SMOKING — LACK OF EXERCISE
CANCER	[STILL SUBJECT TO MUCH RESEARCH] SOME CERTAINLY CAUSED BY CIGARETTE SMOKING, CHEWING TOBACCO AND LIME, CHEMICALS, EXHAUST GASES, DUST.
BRONCHITIS	TOBACCO SMOKE — INDUSTRIAL CONDITIONS — AIR POLLUTION — RESPIRATORY INFECTIONS.
MENTAL ILLNESS AND SUICIDE	

HOOK WORM

HOOK WORM disease is the infestation of very small worms in the intestines. The worm is called 'hookworm'. A hookworm sticks to the walls of the small intestines with the help of its teeth. There it feeds mainly on human blood.

The hookworm disease by itself very rarely causes death. But it makes its victim lazy and lethargic. Regular loss of blood may produce serious complications in the patient who may thus fall a victim to other diseases easily.

Though the disease effects all age groups, it is common among children. It is prevalent more in areas where people have the habit of going outdoors for toilet or otherwise bare-footed.

Symptoms:

Hookworms, if present in any significant number, produce continuous loss of blood in the human body. This condition is responsible for the commonly observed symptoms like laziness, muscular weakness, dull skin, dry hair and a constant feeling of being hungry.

Children having hookworm are pale and thin. They get tired easily and do not feel like playing. They will not grow as they should. If it is a serious case, the child may have dull eyes and pale gums. Some parts of his body may swell.

The doctor can make sure whether the patient is really suffering from hookworm disease or not through laboratory examination of the patient's stool.

How the Disease spreads?

The actual organisms that cause the disease are the thousands of eggs laid by the female hookworm in the intestines of the person suffering from this disease. A female hookworm can lay up to 15,000 eggs a day. These eggs pass out along with the stools of the patient and are deposited on the soil where the patient defecates. These may be scattered to nearby soil by rains and wind. Where the soil is particularly sandy, moist and warm, these eggs may develop into larvae within 24 hours.

These larvae usually find their way back into the human body through the skin of the bare-foot of the person who goes to defecate in the open or walks bare-foot. The retention of wet soil or mud in the toes greatly favours the larvae's entry into the human body.

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When the larvae bites the skin in an effort to enter the body, it sets up an intense itching and inflammation known as 'ground itch'. The hole made is so small that the victim never knows that a hookworm larvae is entering his body. Once inside it makes its way through the flesh to the blood veins and eventually into the lungs. Here it may produce lung inflammation which is often mistaken for pneumonia.

From the lungs, a hookworm may crawl up into the throat of the victim to rest, but is swallowed back into the small intestines, where finally, it stays and grows into adult hookworm capable of laying eggs.

Sometimes, the larvae may also enter the body through contaminated food and water. But mostly, it enters through the skin. It takes about six weeks for the larvae that have entered the body to become egg-laying adult hookworms. They excrete a poisonous substance which tends to make the tiny blood vessels, to which they are attached, swell up and burst. They move from one place to another in the intestines, having bleeding sores behind. This chain of the hookworm's life may continue unless it is broken.

Treatment:

Regular practice of sanitary measures is the only successful way of getting rid of hookworm disease. This will break the chain of the hookworm's life-cycle. Houses should be fitted with sanitary latrines. Defecation in the open should be avoided. Children should not be allowed to play bare-footed. They should always wear shoes before going out. Thus, when infestation cases, the worms will disappear from the intestines. But their complete eradication may take up to seven years.

The patient must consult the doctor immediately when hookworms are suspected. He should not treat himself.

Drinking water should not be taken from a doubtful source. It should be boiled before drinking. Foods should be protected from contamination.

Remember

* Hookworm enters the body through the bare-foot.
* Hookworm mainly feeds on human blood.
* The eggs of hookworm are passed out along with the stools of the patients. They are hatched when deposited on a sandy, moist and warm soil.
* Do not defecate in the open. Use of sanitary latrines will break the chain of the hookworm's life-cycle.
* Do not go outside bare-foot. Make children wear shoes before going out.
* Get stool examined for hook worm disease.
* Take treatment on doctor's advise only

DIS-1A-3

FOOD-BORNE DISEASES

1. Diarrhoea
2. Dysentry
3. Typhoid
4. Paratyphoid
5. Cholera
6. Food poisoning
7. Tuberculosis
8. Brucellosis
9. Undulant fever
10. Worm infestations, eg. round worms, thread worms, trichandua spiralis etc.
11. Leptospira haemorrhagica
12. Poliomyelitis
13. Lathyrism
14. Epidemic dropsy
15. Ergotism

Food allergy : occurs among some persons due to acquired or inherent, dissynchhracy. Usually the allergic manifestations eg. gastrointestinal upset, uoticuria or asthma are due to protein in the food and therefore foodstuffs which cause allergy are mostly fish, eggs, prawn, milk, cheese etc.

Food sanitation : food may serve as a vehicle in transmission of diseases.

1. Milk - excellent food and ideal culture medium. Dirty milk a major health hazard.

Warm surrounding favour : growth of Bacteria. In Urban areas, where the milk is pooled from various sources before distribution. Contaminated milk samples from a single source may contaminate the complete stock of milk.

Milk borne diseases are :-

1. Those due to salmonellae organism
2. Those due to shigellae organism
3. Those due to staphylocci organism
4. Those due to streptocci eg. sore-throat, scarlet fever.
5. Diphtheria
6. Tuberculosis
7. Poliomyelitis
8. Undulant fever.

Sources of milk borne diseases :-

Dirty udders of cow
Dirty fingers of milker
Dirty utensils and bottles
Flies
Droplet infection from Milkman etc.

Milk sanitation :-

1. Animal should be clean and healthy as certified by veternary doctor.
2. Dairy farm to be clean. 3. Milker - free from illness eg. open T.B., typhoid, sorethroad or staphylococci eg. boils and abscesses + clothing, personal cleanliness and nails. 4. Utensils - for storage and transportation clean + sterilized. 5. Pasteurization - destroys most organisms. Does not destroy spore forming organisms.

Meat and fish - uncovered and left meat. Growth of

1. gangrene causing organisms
2. worm infestations
3. Oysters - typhoid
4. Infection due to fingers and flies

Sanitation :- 1. Animals
2. Inspection of cut meat & fish
3. Rapid transportation
4. Proper storage
5. Correct preservation
6. Adequate cooking

Fruits and vegetables :- If eaten raw - poor culture media

1. Choebic dysentry; 2. Worm infestations - round worms + thread worms. proper washing preferably in the $KMNO_4$. Steaming if necessary. Droppings of rats and mice - leptospire haemorrhagica.

Prevention of dietary infections :-

1. Choice of food -fresh, free from infestations
2. Storeage and transport + Preservation, cool dry place protected from rodents.
3. Kitchen staff - strict hygienic practices free from illness and infections.
4. Water - Boiled
5. Sanitary conditions work place + utensils.
6. Health education
7. Use of effective pesticides.
8. How to use only approved chemicals for preserving or as additives.

FOOD POISONING

Injurious effect of food may be due to different causes and gives use to symptoms of Acute Gastro-Enteritis.

I. Chemical :

a) Antimony in Enamel were in contact with fruits/acids dissolves out
b) Zinc from galvanised articles.
c) As from harmful colouring matter.
d) Pb (lead) from soldered utensils
e) Insecticides in food grains
f) Commercial acids used in manufacture of Bees.

II. Parasites or their Cysts like Tape-worm Cysts

III. Bacteria : 1. Salmonella Group causing Salmonellosis or Acute Gastro Enteritis e.g. a) Enteritidis } from
b) S. Typhimurium } Ducks, eggs & partially cooked meats e.g. Sausages.

These organism are nonsporing and Thermolabile. The symptoms appear after about 8 hours as severe pain, vomiting, diarrhoea and collapse.

2. B. Morgani and Proteus
3. Bact. Flexneri
4. B. Coli
5. Stapphylococcus which may contaminate Food, Milk, Milk Products Cakes, etc., due to Staphylococci from the skin, nose or throat or from the cow's udder if some lesion is present. This is usually due to an Exotoxin which multiplies in the food/milk before ingestion and sometimes known as Toxin Type. or poisoning.
6. Cl. Botulinum giving rise to Botulism. It produces a potent toxin and contaminates, fruits and vegetables. Canned and pickled foods are sometimes the source as Cl. Botulinum is an anaerobic sporing organism. Symptoms mat be immediate or delayed after 12-24 hours and are very serious like nervous disorders or vision and dysphagia to be differentiated from Belladonna poisoning. Paralytic iteus may occur and then failure of the heart and respiration.

IV. Food poisoning may be also caused by poisonous fungi e.g. toadstools mistaken for mushrooms.

Investigations to be carried out in an outbreak of food poisoning.

1. Source of infection. Food, milk, cakes etc. to be determined. Food sample, of suspected food to be taken and examined bacteriologically also aerobic and anaerobic cultures to be made and examined. Stop further consumption of suspected food and sale of the same food/milk.
2. Note time interval. When F.P. is caused by living Infective Bacilli the incubation period is longer since time for the Bacilli to multiply and cause sign. If symptoms appear rapidly, it is due to be preformed Toxin the food e.g., tinned foods.
3. Agglutination tests may be positive with known culture.
4. Examination of Vomit/Faeces in the acute stage for Bacteria.

Prevention of Food Poisoning

1. Prompt refrigeration of sliced meats, pastries, custards and cream fillings to prevent any staphylocci from multiplying.
2. Exclusion from food handling of persons suffering from pyogenic infections of the hand or skin.
3. Education of food handlers, cook etc, in hygienic standards in preparation and storage.
4. Extermination of Rodents from kitchens and stores.
5. Prevention of human carriers e.g. in Salmonella infections.
6. Food should be covered. Left overs avoided.
7. Proper canning and Preservative methods of food.

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ORAL REHYDRATION

CHOLERA, Gastro-enteritis and other diarrhoeal diseases lead to much loss of fluid and salts from the body. This condition is called dehydration and it often leads to death if not treated promptly.

One can easily identify a case of dehydration. The following are the signs:

The patient has extreme thirst, sunken eyes, shrivelled finger-tips. He has also rapid pulse and breathing.

In infants, sinking of the soft spot on the head is a sign of extreme dehydration.

In a case of dehydration, it is necessary to replace the lost fluid and salts from the body as early as possible. This process of replacing the lost fluid is called rehydration. This can be done by giving early the rehydration solution.

The development of this oral glucose electrolyte solution therapy for rehydration marks a major achievement in the treatment of all diarrhoeal diseases. The solution can be prepared with the following ingredients, in amounts indicated against each item.

* Sodium chloride	3.5 gm
* Sodium Bicarbonate	2.5 gm
* Potassium chloride	1.5 gm
* Glucose/anhydrous	20.0 gm
* Drinking water	1 litre

Caution: The solution should not be heated or boiled.

Infants and younger children should be given small quantities of the solution-two to three tea-spoon-fuls by mouth after every five to ten minutes. Older children and adults should be given as much fluid as they like. Infants should also be given their usual diet, such as breast milk, cereals, etc. There is no need to wait for diarrhoea to stop. Normal feeding should be continued. The child should never be starved.

Rehydration solution prepared once should not be used longer than 12 hours. In case of need, the solution should be prepared afresh and this can be kept for use for the next 12 hours only.

If a patient shows such symptoms as suppression of urine, cramps in the joints and loss of consciousness, he should be immediately referred to the nearest health centre or a doctor for advice and treatment.

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REMEMBER

A patient of cholera and other diarrhoeal diseases loses a lot of fluid and salts from his body. This might be a danger to his life. This can be prevented by giving the patient rehydration fluid along with normal feeding.

Rehydration solution should be prepared from items purchased only from registered pharmacists and approved medicine stores. The solution once made out of these items should not be kept for more than 12 hours.

The ingredients used for making the solution can also be had from the Primary Health Centres.

Source: Swasth Hind - August 1979

Smallpox

2.1 Identify cases of fever with rash and report them to the Health Worker (Male)

Smallpox is an infectious disease in which the main symptoms are fever accompanied by a characteristic rash. This rash appears mainly on the face, upper limbs and lower limbs and is scanty on the trunk (see slide S.P. 1)

The rash must be distinguished from that of chickenpox which is mainly found on the trunk (see slide S.P. 2)

Smallpox spreads rapidly from person to person by direct contact and by contact with articles which have been infected by small pox cases.

In India smallpox has been eradicated, but you must be alert and inform the Health Worker (Male) immediately whenever you see any case which resembles smallpox.

2.2 Inform the Health Worker of infants aged zero to one year requiring primary vaccination as follows:

2.2.1 In the intensive area inform the Health Worker (Female)
2.2.2 In the twilight area inform the Health Worker (Male)

Smallpox can be prevented by ensuring that every infant is protected by vaccination as early as possible after birth.

If you come across any infant in your area who has never been vaccinated against smallpox, inform the Health Worker (Male/Female) so that he/she can arrange for giving vaccination.

2.3 Assist the Health Worker (Male/Female) in arranging for primary vaccination

You can assist the Health Worker in arranging for primary vaccination in the following ways:

1. Tell the mothers that their children need to have primary vaccination to protect them against smallpox.
2. Instruct the mothers to bathe their babies before bringing them for vaccination.
3. Collect all unprotected infants at a central place at the time when the Health Worker is to visit the area.
4. Follow any instructions given by the Health Worker during the vaccination.

2.4 Follow up cases who have been given primary vaccination.

You should visit each infant on the day after it has been vaccinated to reassure the mother if the child has fever and to ensure that nothing is applied on the vaccination site. Visit the child again 4 to 9 days after vaccination to see whether the vaccination has been successful. The usual course of a successful vaccination is as follows:

1. Between 3rd to 5th day - Blister forms
2. Between 6th to 9th day - Blister becomes larger and pus forms in blister
3. Between 11th to 12th day - Scab forms
4. Between 14th to 21st day - Scab falls off.

Note: If the vaccination is progressing normally, do not interfere with it. If it is infected or there are any unusual reactions, refer the infant to the [illegible]

2.5 Educate the community about the importance of primary vaccination

In your talks with the people in the community, stress the following points:

1. It is important for every person to be protected against smallpox by vaccination.
2. Vaccination is harmless and practically painless.
3. The vaccination site should be cared for as follows:

   a) Leave it uncovered
   b) Do not wash it for 24 hours
   c) Do not apply anything to it, eg., ointments, herbs, oils, etc.
   d) Prevent the child from scratching the site and make sure that the child's finger nails are cut short.

4. If there is no reaction by the 6th day the vaccination will have to be repeated.

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2. The occurrence of a disease which is normally absent from the area, e.g., if cholera normally does not occur in an area and one case of cholera occurs, then a cholera epidemic is on.

EPIDEMICITY IS, THEREFORE, RELATIVE TO THE USUAL FREQUENCY OF A DISEASE IN THE AREA.

Vigilance against the common communicable and infectious diseases is an essential component of public health, particularly in areas where these infections prevail. It is, therefore, necessary that you keep accurate records of the occurrence of these diseases to be able to know when their incidence increases or decreases.

IT IS ESSENTIAL THAT YOU KNOW THE SIGNS WHICH INDICATE A DISEASE OUTBREAK SO THAT YOU CAN MOBILIZE AS EARLY AS POSSIBLE PREVENTIVE MEASURES TO LIMIT THE SPREAD OF THE DISEASE. IT IS ALSO VITAL THAT YOU KNOW HOW THE PARTICULAR DISEASE SPREADS.

Certain diseases are listed as notifiable, i.e. their occurrence must be immediately notified to the Medical Officer of the Primary Health Centre.

Make sure that you are familiar with the list of notifiable diseases in the State in which you are working.

THE MAIN PURPOSE OF NOTIFICATION IS TO PUT INTO OPERATION AS SOON AS POSSIBLE MEASURES TO PREVENT THE DISEASE FROM SPREADING.

7.1 CHOLERA

Cholera is a notifiable disease and as soon as a case is detected it must be notified immediately to the Medical Officer of the Primary Health Centre.

7.1.1 IDENTIFICATION

Cholera is a serious acute intestinal disease. Look for the following signs and symptoms:

i. Sudden onset of profuse watery stool (rice water stool).
ii. Vomiting
iii. Rapid dehydration (loss of elasticity of the skin).
iv. No fever
v. Collapse.

REMEMBER THAT BOTH THE STOOL AND THE VOMIT ARE INFECTIOUS AND MAY TRANSMIT THE DISEASE.

7.2 SMALL-POX

Smallpox in India is under control but vigilance must be maintained to detect any new cases. The disease is characterized by a skin rash and has been described in section 18.2.2.

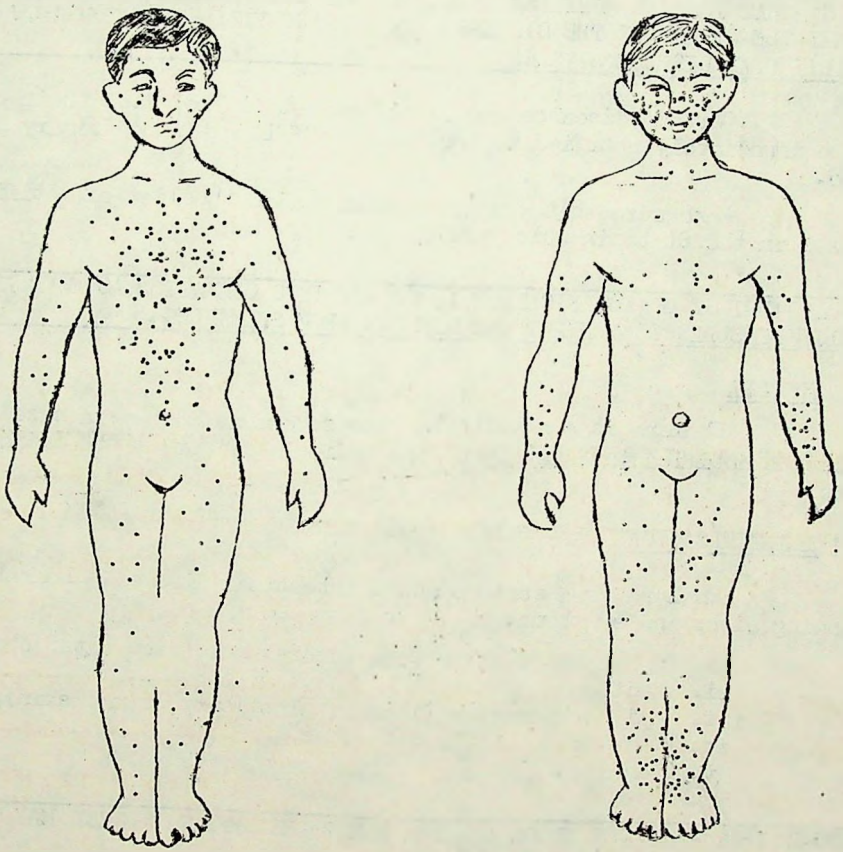
According to current standing instructions, every case of suspected smallpox must be seen by senior experienced Government and World Health Organization staff.

7.2.1 IDENTIFICATION

The patient develops a characteristic rash on the skin. The skin rash is preceded by signs and symptoms which resemble an attack of [illegible]

When the skin rash appears after two to four days, it is identified by the following characteristics:

i. The rash is more abundant on the face and limbsthan on the trunk, i.e. the rash affects mostely the uncovered parts (see fig. 7.1a and b).
ii. Fever.
iii. Sore throat.
iv. Backache and body ache.
v. Frontal headache.
vi. Restlessness and delirium



Chickenpox rash Smallpox rash

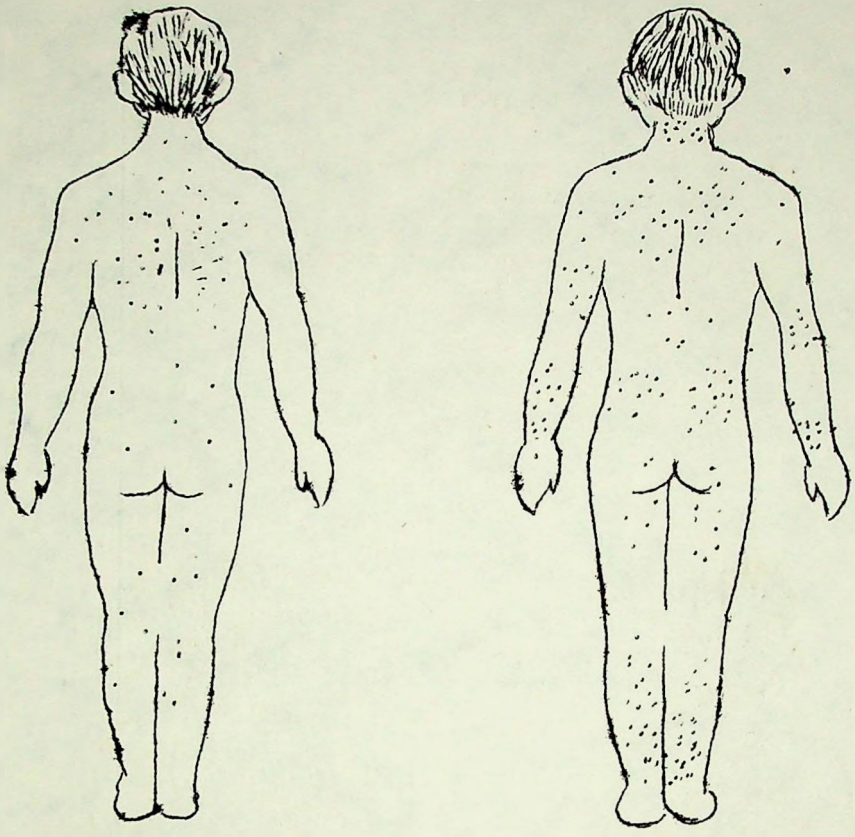
Fig: 7.1a: Smallpox rash and chickenpox rash

REMEMBER THAT THE DISEASE IS TRANSMITTED BY CLOSE CONTACT WITH THE RESPIRATORY DISCHARGES OF PATIENTS AND THROUGH MATERIALS WHICH HAVE BEEN CONTAMINATED BY PUS AND SCABS FROM THE SKIN LESIONS.

7.2.2 CONTROL MEASURES

As soon as you see a patient with a rash suspected to be smallpox take the following steps:

i. Report the suspected case immediately to the Medical Officer (IHC)
ii. Inform your Supervisor immediately.
iii. Isolate the patient. If the patient is seen in his home,



Chickenpox rash Smallpox rash

Fig. 7.11: Smallpox rash and chickenpox rash

GET THE COOPERATION OF THE COMMUNITY LEADERS TO HELP YOU. On NO ACCOUNTS MUST ANYBODY ENTER OR LEAVE THE HOUSE AND THE PATIENT MUST BE CONFINED TO THE HOUSE.

iv. With the help of the Health Worker (Female) and your supervisor vaccinate all the village community.

v. Assist in the disinfection of all the materials which have been in contact with the patient.

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FOLLOW THE INSTRUCTIONS OF THE DOCTOR AND PARTICIPATE FULLY WITH THE CONTAINMENT TEAM TO LIMIT THE SPREAD OF THE DISEASE.

vi. With the help of the Health Worker (Female), carry out a health education programme to increase awareness about the incidence of smallpox in the community.

At the end of Volume II of this Manual you will find a card showing a baby with smallpox rash. Show this card to people in the community and ask if there is any case like this in the village.

THE FIGHT TO ERADICATE SMALLPOX IS STILL GOING ON AND HAS REACHED ITS FINAL STAGES.
PUBLICIZE WIDELY THE INCENTIVE OF Rs.1,000 WHICH THE GOVERNMENT PAYS TO ANYBODY REPORTING A CASE OF SMALLPOX TO THE AUTHORITIES. THIS INCENTIVE IS CONSIDERED IMPORTANT IN THE FINAL EFFORT TO ERADICATE SMALLPOX IN INDIA.

Contd/18.

7.3 PLAGUE:

Plague is an infectious disease transmitted to human beings through the bite of a flea which has been infected by a plague-infected rat or other rodent. In rural areas, wild rodents transmit the infection to each other through flea bites and, therefore, keep a focus of plague infection alive. These infective rodents may pass the infection on to the domestic rats which live in or near houses. From them the infection spreads to human beings.

PLAGUE IS TRANSMITTED BY THE BITE OF AN INFECTED FLEA, HANDLING OF TISSUES OR FLUS OF INFECTED RATS, AND BY INFECTED DROPLETS OR SPUTUM FROM PATIENTS SUFFERING FROM PNEUMONIC PLAGUE.

7.3.1 IDENTIFICATION

The sings and symptoms of plague are both local and general. Look for the following.

i. Swelling of the glands in the groin or armpits.
ii. Small skin haemorrhages which look like red spots
iii. Fever.
iv. Restlessness, mental confusion and, in some cases coma.
v. Staggering gait which makes the patinet walk like a drunken person.

VERY OFTEN THESE SYMPTOMS, ESPECIALLY IN THE EARLY STAGES, ARE MISSED AND THE PATIENT IS TREATED FOR A GLAND INFECTION. THEREFORE, ENQUIRE AS TO WHETHER OTHER CASES WITH SIMILAR SYMPTOMS FROM THE SAME AREA HAVE OCCURED. IF SO, PLAGUE MUST BE SUSPECTED.

vi. Signs of pneumonia develop as the disease progresses.

WHEN PNEUMONIA DEVELOPS THE SPREAD OF THE DISEASE IS RAPID AND SEVERAL CASES OCCUR IN A SHORT TIME WITH SIGNS AND SYMPTOMS OF PNEUMONIA.

The confirmation of plague can be made only by laboratory investigations. It is, therefore, very important to confirm the presence of plague early in order to take emergency measures and start treatment. Early treatment reduces the mortality rate, which is very high in pneumonic cases.

7.3.2 CONTROL MEASURES

As soon as you see a patient suspected to be suffering from plague, proceed as follows:

i. Notify the Medical Officer (IHC) immediately.
ii. Inform your supervisor.
iii. Isolate the patient. The isolation must be very strict in cases of pneumonia and no person should be allowed near the patient until the doctor sees him and gives further instructions.
iv. Collect all sputum and other discharges in containers, if possible in a disinfectant solution.
v. Clothing which has been in contact with the patient should be disinfected.
vi. Educate the community and tell them how the disease is spread, particularly stressing the importance of the flea in transmission of the disease.

CARRY OUT ALL THE INSTRUCTIONS GIVEN BY THE DOCTOR AND HELP YOUR SUPERVISOR IN CONTAINING THE SPREAD OF PLAGUE.

vii. Usually the area is closed for travellers and people are not allowed to leave or enter the area for about a week from the last confirmed case.

viii. Immunization with plague vaccine should be carried out to contain the infection and protect the local community.
ix. Measures to control the rat population are carried out.
x. Destruction of fleas by spraying with DDT is an important control measure.

BOTH YOU AND THE HEALTH WORKER (FEMALE) HAVE A ROLE TO PLAY PARTICULARLY IN HEALTH EDUCATION, IMMUNIZATION, RAT CONTROL, AND DDT SPRAYING OPERATIONS.

REMEMBER, IF AN UNUSUALLY LARGE NUMBER OF DEAD RATS ARE SEEN AROUND HUMAN HABITATIONS, YOU SHOULD NOT ALLOW PEOPLE TO TOUCH THEM. REPORT THE FACT TO YOUR SUPERVISOR WHO WILL ARRANGE FOR THE DEAD RATS TO BE EXAMINED FOR PLAGUE.

7.4 POLIOMYELITIS (INFANTILE PARALYSIS)

Poliomyelitis affects mainly children but adults of any age get the disease. The virus which causes the disease is transmitted by direct contact with the throat secretions of infected persons, and possibly by the contamination of food with infected faeces.

OVERCROWDING AND BAD SANITATION ARE, THEREFORE, THE MAIN FACTORS IN THE SPREAD OF POLIOMYELITIS.

7.4.1 IDENTIFICATION

It is difficult to detect the disease in its early stages because the signs and symptoms resemble those of an attack of influenza. However, when the paralysis sets in and the child is unable to move the affected limb freely, medical advice is usually sought. When the disease affects the upper part of the spinal cord, the patient is unable to breathe and unless quick treatment is given in a hospital, death results.

The disease is identified by the following signs and symptoms.

i. Paralysis of a limb (arm or leg), usually on one side only.
ii. The paralysis is accompanied by wasting of the muscles so that the limb looks thinner and is colder than the other one.
iii. Headache.
iv. Some stiffness of the neck but not complete rigidity.
v. Fever and slight sore throat.

THE SIGNS AND SYMPTOMS MAY APPEAR IN ISOLATED CASES OR A NUMBER OF CASES MAY APPEAR AT SHORT INTERVALS.

7.4.2 CONTROL MEASURES

When you see a child with paralysis of a limb and symptoms which resemble a cold, poliomyelitis should be suspected. Proceed as follows:

i. Send the child to the Primary Health Centre without delay.
ii. Keep a very close watch on the other children who have been in contact with the child; especially school children.
iii. Inform your supervisor.
iv. Inform the school teacher that the child is suspected to have poliomyelitis and request him to inform you immediately if any child is absent from school because of sickness.

IT IS NOT USUAL TO CLOSE THE SCHOOLS UNLESS THE EPIDEMIC IS A SEVERE ONE. IT IS EASIER TO WATCH THE CHILDREN AT SCHOOL AND DETECT EARLY SIGNS OF DISEASE THAN TO LET THEM MIX AND PLAY TOGETHER AWAY FROM SCHOOL WHERE CLOSE SURVEILLANCE IS NOT POSSIBLE.

CHAPTER 3

Communicable Diseases

Communicable diseases are those which are transmitted from man to man or animal to man directly, through infected materials or through insects. Some of the common communicable diseases include cholera, typhoid, smallpox, malaria, filariasis, leprosy, tuberculosis, measles, common cold, trachoma and sexually transmitted diseases.

3.1 Inform the Health Worker(Male) immediately an epidemic occurs in his/her area

When a disease occurs in an area where it does not normally occur or when there are more than the usual number of cases, an epidemic is established.

Be alert to an increase in the number of cases in your area with the following signs and symptoms:

1. Diarrhoea, vomitting, jaundice and passing worms
2. Fever with or without rigors
3. Rash with fever
4. Cough and cold
5. Discharging eyes
6. Discoluration of skin with loss of sensation
7. Stiffness of neck
8. Lockjaw
9. Paralysis or weakness of limbs in children
10. Sore on the genital organs with or without discharge

Report epidemics immediately to the Health Worker(Male) so that the necessary action can be taken to prevent spread of the disease.

3.2- Take immediate precautions to limit the spread of disease

While waiting for the Health Worker(Male) to arrive you must yourself take certain measures to limit the spread of disease.

These include the following:

Signs/Symptoms	Precautions to be taken
3.2.1 Diarrhoea, vomiting, jaundice and passing worms	1. Where sanitary latrines are not available see that the stool & vomit are buried so that flies do not settle on them. 2. See that any soiled clothing or linen of the patient is washed, boiled and dried in the sun 3. Tell the community members to boil water and milk before drinking 4. Chlorinate all drinking water sources 5. Those caring for the patient should not handle the food of the family and should wash and dry their hands after caring for the patient 6. See that all food is covered to protect it from flies 7. Hands should be washed after defaecation and before handling food 8. See that all eating utensils especially those used by the

3.2.2.	Fever with or without rigors	1. Isolate the patient
3.2.3	Rash with fever	1. Isolate the patient 2. Keep the patient in bed in a darkened room 3. See that any dressings which have been in contact with the patient are burned or buried 4. See that soiled clothing or linen of the patient is washed, boiled and dried in the sun
3.2.3	Cough and cold	1. Isolate the patient especially from young children 2. Teach people not to spit or clear the nose indiscriminately 3. Teach people to cover the nose when sneezing or coughing
3.2.5	Discharging eyes	1. Ensure that no one else uses the patient's towel, handkerchief or surma stick 2. Teach people not to allow flies to settle on the eyes and face
3.2.6	Discolouration of skin with loss	1. Isolate the patient especially from children
3.2.7	Stiffness of neck	1. Isolate the patient
3.2.8	Lock jaw	1. Keep the patient in bed in a quiet place
3.2.9	Paralysis or weakness of limbs in children	1. Keep the patient in bed 2. Where sanitary latrines do not exist, see that the stool is buried so that flies do not settle on it.
3.2.10	Sore on the genital organs with or without discharge	1. Tell the patient to avoid sexual intercourse until cured

Start treatment of those signs and symptoms which are within your competence to treat. See Chapter 11 for details.

3.3. Educate the community about the prevention and control of communicable diseases

There are certain steps which can be taken in order to prevent communicable diseases from occurring. You should educate the community about the need for taking the following measures:

1. Immunization against tuberculosis, diphtheria, whoopin cough, tetanus, poliomyelitis, smallpox, cholera and typhoid
2. Proper disposal of solid waste, liquid waste and refuse
3. Safe drinking water
4. Latrine construction
5. Food hygiene

6. Control of mosquitoes, flies and other insects
7. Control of rats
8. Destruction of stray dogs
9. Prevention of overcrowding
10. Personal hygiene and clean habits

In addition you should tell the community to inform you immediately there is a case with any of the signs or symptoms mentioned. You should teach them what speci ic precautions they should take to control the spread of disease.

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DIS-183

INSURING ONE ANOTHER AGAINST DISEASE.
THE RAHA EXPERIENCE.

The Need

India has the highest mortality rate (1 39/1000) among all Asian countries, and the infant mortality rate is still around 129/1000. Although there is a significant improvement in the health situation, accompanied by a substantial growth in manpower and instiutions, the inequalities between geographical areas, social classes and between urban and rural populations remain unacceptable,

The Tribals are one of the most neglected rural populations, and serious sickness does not only mean an almost unimaginable degree of human suffering, but also economic ruin since they'll often have to sell or mortgage cattle and fields in a belated, mostly ineffective effort to save their relative.

The underlying causes of this situation need to be analysed, but this is not our purpose here. Rather than curse the darkness, let us light a candle.

Can the poor ensure themselves?

From our past experience in RAHA, we are inclined to answer this question with a clear "YES",(if the scheme is built on THREE PILLARS. These three basic prerequisites can be listed as follows:

1- A great number of people must take part.

This is easy to understand. A burden which will crush a few, can be carried more lightly by many.

2- There must be a great stress on prevention.

All effective means must be used, and everybody's cooperation enlisted to prevent people from falling sick.(60% of diseases are preventable). Every group that takes part, must be actively engaged in preventive and protective measures under the guidance of the nurse and village Health Promotors.

3- A spirit of solidarity should be the main motive.

This last element is often over-looked, but it is as essential as the previous two. If everybody tries to get back his money's worth, irrespective of his medical need, the whole scheme is bound to fail. Every group that can develop a minimum of mutual trust and an attitude of service, should be able to do it. A spirit of solidarity is not the monopoly of Christians, although it may imply christian values.

How is it done in RAHA(Raigarh Ambikapur Health Association)?

RAHA was started in 1969 but remained rather dormant. From 1974 onwards, it develped itself into a community based, preventive-oriented health service.

It serves a population of predominantly tribal people in Surguja and Raigarh districts of Eastern Madhya Pradesh. It tries to coordinate the work of 3 base-hospitals and 47 rural Health Centres (2-8 Bed facilities) staffed by trained nurses-midwives.

The most characteristic development has been the Village Health Promotors Training Programme. During the past ten years, VHPs have been trained for more than 1000 villages, serving a population of approximately 400,000 people. They were selected by the villages, and work as volunteers. Every health centre has two to four supervisors who act as liasion between health centre and VHP, play a supportive role and also assist with protective and educational health activities(immunizations, mother-child clinics, etc.)

The nurses conduct monthly follow-up meetings for all the VHP of their catchment area.

(For further details, kindly refer to the report of 1983 "Tribal Community Health")

The Medical Insurance Scheme of RAHA(M.I.S.)

Historical Development.

It is important to note that the M.I.S. began only after a very intensive compaign towards community health with stress on education and prevention. More than 5 years of training village Health Promotors together with motivation and orientation of the nursing staff preceded the start of the M.I.S.

Even then we made the initial mistake of focussing on insurance for hospitalisation, since that was the most crucial problem. There was very little or no response because nobody wanted to be hospitalised.

A proposal for " local treatment" insurance scheme got a better response. Our ability to "double" the contributions of the people was a strong incentive to both health staff and people.

In the beginning however people did not have the slightest idea what "insurance" could mean for them, and how it had to function. Therefore the membership fee was kept extremely low.

After three years of operation, most people have began to see the tremendous benefits(they were regularly kept informed), and now they also begin to see the practical requirements to come to a self-supporting scheme.

The fees could gradually be raised, and have now been linked with the value of "rice", so that there is no longer need to change every year.

How does the M.I.S. function at present?

The first thing to understand is that we work in a two-tier system. The "local insurance" fund at health centre level is called the Samaritan Fund.

Purpose:- to finance all preventive, protective and curative expenses of the health centre, and
- to foster early treatment, independent of economic condition (when the patient does not have to pay at the time of treatment he/she will be inclined to come earlier)

Collection:

1. Chiefly from membership fees
   - once a year to be paid two months in advance

i.e.before 31st January when on 1st April MIS starts(late-comers pay double)

- through the village health promotors who record the names of all the members, and forward them to the nurse in cash or kind. the value is fixed, but they can pay in any form
- per person

unborn children are insured together with the mother. To avoid abuse, the whole family should become members. When there are more than six persons ina nuclear family-(parents,children,grand-parents)concessions can be given to those in excess of six.

2. from incidental other sources
donations, campaigns, fairs, lotteries etc,
3. Registration fees: even the members pay a nominal Rs.0.50 to avoid abuse.

Amount of membership fee

In principle, each health centre can decide for themselves, in practice, they all want a common directive.
Repeatedly changing the fee is disturbing and not favoured.
Therefore the fee is fixed in relation to a common market commodity e.g. the value of two kilos of rice per person per year.
This allows for minor local variations, fosters early collection and will show a yearly increase in real value.

N.B. To ease the psychological parting, every family that this year take part in the scheme, can receive a small bottle of iodine free of cost(which in turn can prevent a lot of infected wounds)

Eligibility

Those who belong to the economically weaker section, irrespective of caste or religion, but who can be trusted to join in a spirit of solidarity, and are ready to take part in the proposed preventive measures. The presence of an active VHP is therefore essential.

Management of the Fund.

The fund are kept at health centre level, but the members are regularly informed through the VHP of expenses incurred/income received.

For the first few years, RAHA has been able to double every rupee that is collected by fees.

If the fund is insufficient to last a whole year, restrictions can be agreed upn (EXCLUDE tonics, + vitamins or part /full payment for injections)

The Samaritan Fund is not just an insurance schemes. It acts as a catalyser for community participation. It is the change-over from the old system of health care where everyone pays for himself and consequently was limited to those who can pay, to a self-reliant community health care system which includes the poor because each one pays for the other, and the others pay for him.

In the old system, there was no end to the line of sick and only those who had enough, could afford to pay for our services while the

poor either got ruined by these services or had to be satisfied with lesser services. Now through the Samaritan Fund we see the sick diminish in number and the poor can benefit equally because all share th burden. It is an implicit invitation towards concern for one another and gives our health institutions the opportunity to serve those for whom they originally started.

Central Fund/Hospital Referral Scheme.

This forms the second tier, because without this the M.I.S. would not be complete, the top of the broad-based pyramid would be missing Part of the money collected at the health centre level (Samaritan Fund) is paid into the Central Fund from which under certain conditions the major share of the hospital bills of members can be paid.

Conditions for referral:

1. Only those who have paid their contribution to the Samaritan Fund, two months earlier, can benefit from the scheme.
2. Besides being a member, there must be an active VHP in the village who regularly attends the follow-up meetings and has to fill in a form showing the care that has been taken to prevent the disease, if applicable.
3. The health centre has to have a sufficiently qualified and experienced nurse who effectively takes care of both preventive and curative needs.

Method of Referral:

Normally a sick person first reports to the VHP who, when the need arises, informs the nurse or sends the patient to the health centre. Depending on the case, the nurse has then to decide whether the patient needs to be referred to the doctor.

The patient comes to the OPD of the hospital, with a RAHA referral form on which the nurse has written the essential data of the case + treatment given. The patient also comes with Rs.100/- in hand. In case the patient is too poor to collect this amount, the nurse who refers has to pay or advance the money from Samaritan Fund.

If treated in OPD only, the patient pays up to Rs.100/-, the balance above this is paid by the Central Fund.

If admitted by the doctor, the patient pays also only Rs.100/- as his share of the bill. RAHA pays the rest up to Rs.1000/-

No individual acquires the right to be referred or to be admitted. And only the doctor decides when and how long the patient shall be admitted. The patient arranges for his own food and shall be accompanied by a companion who sees to this and other needs.

Limitations:

No case, resulting from a criminal action(abortion, fight)should be given a RAHA referral form.

Individuals already covered by another Insurance Scheme(v.g.Govt. servants) should apply to that scheme and not to RAHA.

No case, older than one month, should be given a RAHA referral form.
(T.B. Patients are covered by another supplementary programme)
N.B. These regulations are not yet perfect, as anyone will realise but they provide a framework which is sufficient to operate.

Can the M.I.S. become self-supporting?

- at the health centre level
  with a competent nurse, who enjoys the confidence of the people there is no real problem if the patients pay individually.
  Our question is: can it be done if people pay collectively?
  The answer, based on our experience in RAHA, is yes, if you build on the three pillars mentioned earlier.

The real cost of treatment for the whole community can be kept low, if people take an active part in prevention

If they report early, or are detected early

If treatment is started without delay

If nurses and people are gradually educated towards a low-cost and rational drug therapy

If all protective measures(immunizations, etc)are used

If herbal remedies/inexpensive home-remedies are used judiciously

- at the hospital level
  here I can give accurate figure of the last three years(see appendix) although the last few months, we have seen a rather sharp decline in the number of patients referred(due to a growing awareness among doctors and nurses), we can rightly assume to get about 500 referral cases per year(in the 3 base-hospitals) at an average cost of Rs.400/- per patient. This means we need a yearly collection of Rs.2,00,000/- at the present rate of contribution from the Samaritan Fund (Rs.2/- per person), we therefore aim at 100.000 members.
  Given the number of VHPs(approx.1400), we need only an average of 71 members to be recruited per VHP(or 12 families).
  As time passes, people begin to realise the enormous benefits and numbers are increasing quite satisfactorily.
  Here again, very much depends on
- the understanding and the cooperation of the doctors and hospital authorities
- the smooth relations between hospital and health centre staff
- the increased diagnostic skills of the health centre staff which makes early referral possible.
- whether the transport expenses to the hospital can be subsidized.

THE RAHA INFRASTRUCTURE

		RAIGARH DISTRICT	SURGUJA DISTRICT	TOTAL
Village Health Promotors		983	444	1,427
VHP Supervisors		59	21	80
Reg.Nurses, RM		31	5	36
Aux.Nurses, M		16	19	35
Dioc.Health Coordinators		2	2	4
Hospitals	Kunkuri	100 Beds	Ambikapur 120	3
	Raigarh JMJ	50 "		
Population covered by RAHA		300,000	100,000	400,000
Total Population of the district		1,250,000	1,250,000	2,500,000

MEDICAL INSURANCE SCHEME

Membership total: 1980/1 - 2000
1982 - 10000
1983 - 37000
1984 - 45000

RAHA patients referred

1981 - 300
1982 - 551
1983 - 711
1984 - 594 2156 Total

BILLS PAID BY RAHA

	HC-KUNKURI	HC-AMBIKAPUR	RAIGARH JMJ
1981 RS:	50,487.95	26,477.25	15,081.00
1982	1,17,076.40	38,694.75	27,336.00
1983	1,76,904.05	79,889.65	29,780.00
1984	1,33,365.50	34,293.29	16,493.00
TOTAL:	4,77,833.90	1,79,354.94	88,690.00

D.S-1A-10

SRI CHINNASWAMY MUDALIAR MEMORIAL TRUST

ENDOWMENT LECTURE
ON

11TH MARCH 1990

"HEART DISEASE - AN OVERVIEW"

BY

DR.V.PARAMESHVARA*
BANGALORE

Mr President, Mr. V.C.Ramachandran, Ladies and Gentleman,

I appreciate the significant honour that the trustees of Sri Chinnaswamy Mudaliar Memorial Trust have bestowed on me by inviting me to deliver the endowment lecture this evening, I am proud of the fact that I belong to the band of illustrious orators who have preceded me.

The Late Sri Chinnaswamy Mudaliar was a distinguished son of the soil, who has contributed tremendously towards construction, growth and development of Bangalore. Lofty and prestigious buildings bear testimony to his vision and statemanship as well as his involvement in the health and welfare of the people. Hence, I thought it appropriate to speak on the subject which has become number one killer in all age groups. The subject is "Heart Disease - An Overview".

India has the dubious distinction of having the disease and health problems of both developed and developing countries. The case in point is the modern epidemic of heart diseases. Rheumatic fever and rheumatic heart disease are major causes of mortality and morbidity especially in young people. It has been estimated that rheumatic fever is the most common cause of heart disease in the 5-30 years age group. Its prevalence amongst children between 5-15 years is in the range of 6 per thousand with regional variations. It is common knowledge that rheumatic heart disease is typically associated with poverty, poor housing and overcrowding and the disease has declined throughout the century in industrialised societies. The prevention of rheumatic fever is certainly possible by early effective treatment, which makes it one of the most preventable cardiovascular diseases. The Government should commit itself by appointing a National Programme Co-ordinator for rheumatic heart disease and also make necessary budgetary provisions.

Ischaemic heart disease (Heart attacks) is emerging as the most important heart disease in recent times. Unchecked, the epidemic is threatening to engulf the whole society in the very near future resulting in incalculable damage. It is estimated that by the turn of the century, life expectancy of an average Indian will be in the range of 64 years and no doubt, at the rate of demographic transition it could assume an alarming public health dimension. It is feared that if present conditions persist, every second person born alive, will die from heart disease. The disturbing trend is younger age groups are being affected by coronary artery disease (Heart Attack).

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Economic advance and changing life styles appear to be primordial causes. The sheer size of the problem, the early onset and insiduous development of narrowing of arteries, provide sufficiently strong reasons to mandate the taking of immediate steps towards prevention.

The prevention of Ischaemic Heart Disease (Heart Attack) should be directed towards

1. **Population Strategy** : For altering the life style and environmental characteristics.
2. **High Risk Strategy** : For bringing preventive care to individuals at special risk.
3. **Secondary Prevention** : For averting recurrences and progression of disease in those already affected.
4. **Primordial Prevention** : Preventing the emergence of predisposing conditions in regions in which they have not appeared.

The problem has to be seriously tackled by adoption of healthier life style and environment, screening to identify the disease in its early stages, bringing preventive care to individuals at special risk and secondary prevention in known cases.

The Government responsible for the health of the public, should straight away garner adequate information on current life styles and prepare time bound action goals. It is imperative to formulate a national plan for prevention and control of heart diseases and also to establish appropriate communication and co-ordination with other departments, particularly those dealing with food policy, education and public information.

Government and national medical and voluntary associations should declare a joint committment towards a tobacco smoke free society.

The whole problem has to be tackled on a war footing and an approach that emphasises the intervention to change the whole community by health education and matters such as nutrition and eating patterns, low fat dairy products, changed diet in institutions, prohibition of smoking, physical activity at work and leisure, and special training of health personnel.

Use of mass media in health education is an important cost effective method to enhance community participation. Mass media experts learned in behaviourial sciences and health communication skills must be included in this programme.

The need of the hour is better doctors than more doctors, better health education than more education, better health care than more health care delivery, a political will, involvement of non-governmental organisations, a dedicated profession and a motivated community.

" PREVENT DISASTER PROTECT COMMUNITY"

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DIS-1.A.11

more than two hours previously. The management of young children is more difficult—most episodes are poison scares rather than true poisonings. Rather than give young children charcoal immediately on presentation, we suggest confining it to the few who develop symptoms—in a dose sufficient to increase elimination of the drug.

Repeated doses of oral activated charcoal have not yet been shown to reduce morbidity and mortality. Further studies are required to establish its place and the dose to be given. Until these data are available, severely poisoned adults should be given 150-200 g through a nasogastric tube over 4-8 hours with the aims of achieving a maximum reduction in elimination half life and an improvement in the clinical state. The total dose given is probably more important than the frequency of dosing.

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The cervical spine in rheumatoid arthritis

Needs careful assessment

Rheumatoid arthritis commonly affects the cervical spine, causing several well defined deformities.[1] Damage to the cervical spine from rheumatoid arthritis has been noted in 30% to 46% of necropsy studies and is second in frequency only to that seen in the metatarsophalangeal joints.[2] One in four inpatients with rheumatoid arthritis and between 17% and 86% of all patients with this disease have radiological evidence of instability of the cervical spine.[3-6]

These high rates reflect the anatomy of the cervical spine and the dynamic forces that act on it. Each of the apophysial and ligamentous articulations of the cervical spine is susceptible to the same inflammatory changes as those in peripheral joints of patients with rheumatoid arthritis.[2] Furthermore, the cervical spine is constrained between a somewhat rigid thoracic spine and a skull weighing 6 kg; movement of the head, which has been estimated to occur around 600 times each hour, adds to the forces on the articulations.[7]

Any segment of the cervical spine may be affected by the rheumatoid inflammatory process, but destructive changes are most prominent at the occipitoatlantoaxial junction. Atlantoaxial subluxation is the most common deformity and is due to destruction and resultant laxity of the transverse ligament. This allows the atlas to move forward relative to the odontoid process of the axis when the neck is flexed. In radiographs this is seen as a widening of more than 3 mm in the space between the anterior arch of C1 and the odontoid. The corresponding reduction in the space posteriorly restricts the canal available for the spinal cord. By contrast, posterior subluxation of the atlas is infrequent and is seen only in the presence of severe erosion and dislocation of the odontoid.[8]

Recent studies using magnetic resonance imaging in patients with atlantoaxial subluxation have shown an inflammatory mass of granulation tissue around the odontoid arising from the synovial lining of the articulations. This periodontoid mass is not visible in patients who have had surgical fusion of the first two cervical vertebrae or in whom deformity has progressed to that of atlantoaxial impaction (see below).[9,10] The bulging of this mass may further reduce the space available for the spinal cord and cause neurological deficits in patients with only a moderate degree of atlantoaxial subluxation.

When the disease affects one of the occipitoatlantoaxial articulations (termed lateral mass) it may produce the syndrome of non-reducible rotational tilt of the head, the main clinical features of which are occipital pain, tender points in

the neck, and tilting of the head towards the affected side.[11] If both sides are affected collapse of the lateral masses allows the skull to descend on to the cervical spine and the odontoid to enter the foramen magnum. This deformity has been termed cranial settling, superior migration of the odontoid, or atlantoaxial impaction and is seen almost exclusively in association with atlantoaxial subluxation.[6 8 12] Subaxial subluxation is a late development; it often affects several vertebrae, leading to a "stepladder" deformity. Extensive rheumatoid disease of the cervical spine results, then, in a combined deformity of atlantoaxial subluxation—subaxial subluxation and atlantoaxial impaction—a devastating complication and a truly formidable therapeutic challenge.

Deformities of the cervical spine are seen most often in patients with rheumatoid arthritis of more than 10 years' duration. They are usually associated with severe destructive peripheral arthritis, rheumatoid nodules, a high titre of rheumatoid factor, and treatment with corticosteroids.[6 11] Progression of the deformity is unpredictable in a given patient, but follow up for five to 10 years has shown worsening of the instability in 16% to 41% of the patients.[6 8 12] These percentages may be too low: with progression of the deformity to atlantoaxial impaction the magnitude of the atlantoaxial subluxation may seem on radiography to be reduced, giving the false impression radiologically of improvement.[12]

Many patients with rheumatoid disease of the cervical spine remain asymptomatic for years, but they are at risk of a range of neurological complications and even sudden death from medullary compression. Neurological abnormalities may be subtle and difficult to establish in the presence of deforming arthritis, muscular atrophy, and the neuropathy that may be associated with rheumatoid arthritis. Patients may complain of intractable pain in the neck or the back of the head. They may have symptoms of vertebrobasilar insufficiency with vertigo or drop attacks and may have signs of myelopathy.[13 14] Myelopathy, once it develops, is usually rapidly progressive. In patients with subaxial subluxation myelopathy may occur with only slight subluxation because of the narrower diameter of the spinal canal below the axis. Profound and complex neurological deficits may be found in patients with the combined deformity of atlantoaxial subluxation—subaxial subluxation—atlantoaxial impaction. Atlantoaxial subluxation with subluxation of less than 9 mm carries the least risk of neurological damage, while atlantoaxial subluxation of more than 9 mm, atlantoaxial impaction, subaxial subluxation, non-reducible rotational tilt of the head, and combined deformities are all associated with a higher risk of neurological deficit.[8 15]

Plain radiographs of the cervical spine in flexion and extension will allow recognition of atlantoaxial subluxation and subaxial subluxation. In patients with atlantoaxial impaction, however, odontoid erosion and osteoporosis may make plain radiographs inadequate for assessing the extent of cranial settling and resultant penetration of the odontoid into the foramen magnum. Various measurements have been advocated to define the extent of cranial settling. McGregor's line, which assesses the protrusion of the odontoid process above the foramen magnum, is widely used in clinical practice. Because of its superior contrast capabilities magnetic resonance imaging is the current first choice technique for assessing instability of the cervical spine.[16 17]

Patients with a minor degree of atlantoaxial subluxation or with subaxial subluxation need treatment only with a soft cervical collar—which provides symptomatic relief, acts as a reminder to patient and doctor, and may provide some degree of protection from trauma. In the presence of intractable cervical pain, neurological deficits, or myelopathy, or combinations of these, the recommended procedures are halo stabilisation and surgical arthrodesis. The place of surgery in the early stages of instability of the cervical spine is less certain, nor is there any consensus on whether progression can be retarded by early surgery. In a retrospective study of 110 patients with rheumatoid arthritis who had surgical treatment we found recurrence of their cervical instability after a mean interval of nine years in 5·5% of patients with atlantoaxial subluxation who required only atlantoaxial fusion —but a 36% recurrence rate after a mean interval of 2·6 years in patients with atlantoaxial subluxation and atlantoaxial impaction who required fusion from the occiput to C3. No patient with atlantoaxial subluxation and fusion of C1 and C2 progressed to develop atlantoaxial impaction.[14 18]

Many patients with substantial deformities remain asymptomatic, but they are at increased risk of neurological damage with the passage of time. They are also at risk if they need surgery or induction of anaesthesia for any other reason. In one recent study 60% of patients with rheumatoid arthritis having total hip or knee replacements had radiological evidence of instability of their cervical spine, and nearly half of these had no symptoms referrable to their necks.[19] Patients with rheumatoid arthritis undergoing any major surgical procedure should be assessed by having radiographs taken of the cervical spine in flexion and extension. Indeed, all patients with rheumatoid disease of the necks, even though asymptomatic, should be followed up carefully for evidence of neurological deficit, and all should undergo periodic radiographic monitoring.

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a location not approachable from other techniques. Destructive procedures yield best results for cancer pain, but our feelings are that they are contraindicated for benign generated pain, and stimulation techniques should be reserved for these patients.

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CHAPTER

18

Stimulation of the Peripheral Nervous System for Pain Control

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INTRODUCTION

It has now been 10 years since electrical stimulation of the peripheral nervous system was reintroduced into medicine for the control of pain (12, 28, 29, 37). In that period of time our understanding of the anatomy and physiology of nociception has undergone dramatic change. The separation of pain (nociception), suffering, and pain behavior has occurred, and our understanding that human pain is a complex issue with physical, psychological, and social aspects is developing slowly. It is impossible to discuss the therapy of pain without considering the multiplicity of factors which may influence the complaint (36, 38). In order to present this summary of afferent stimulation for pain control, it is necessary to place the stimulation procedures in appropriate historical context and to discuss current concepts which are important in understanding human pain. Then it is possible to collate the many publications dealing with pain control through transcutaneous electrical stimulation and to place the techniques for stimulation of the peripheral nervous system appropriately in the therapeutic armamentarium of the physician interested in treating pain.

Stimulation-induced analgesia was a reality in classical times, and the description of the use of naturally occurring electrical sources, the electric fishes for pain treatment, is well documented (32). It was appreciated centuries ago that electrification of acupuncture needles seemed to improve the results in pain control. Benjamin Franklin was an enthusiast for the value of electricity in medicine and described the effective treatment of trigeminal neuralgia with electrical stimulation. This medical literature over a long period of time is a curious mixture of fact and fancy, valid scientific observation, and quackery (42). Throughout the latter half of the 19th century, there were many electrical stimulators on the market which were freely available to both the medical profession and the lay public. During this time, it was generally assumed that the electricity had some special properties, and only rarely is there any

indication that it was the stimulation of the nervous system which was beneficial. This, in spite of the fact that Althaus (2) in 1856 had clearly described sequential paresthesia, hypalgesia, analgesia, and anesthesia occurring with electrical stimulation of major nerve trunks. Anecdotal reports correlating nerve stimulation, analgesia, and pain control appeared but were not collated or evaluated in any meaningful way. The rise of surgery, neuroanatomy, and pharmacology, coupled with the application of the scientific method to medicine, combined to virtually eliminate the use of electrical stimulation of the nervous system for any purpose in the early 20th century. One major observation was made when it was determined that the application of a large electrode around the upper abdomen seemed to decrease postoperative discomfort and ileus in abdominal surgery, but this was erroneously attributed to warming the liver. The 1965 publication of the gate theory of pain perception rekindled interest in pain as both a laboratory and a clinical problem (43). By that time, only one American manufacturer was continuing to make an electrical stimulator-massage device for sale on the open market.

It would be nice to be able to say that those stimulated by the Melzack-Wall theory began an orderly progression of research investigating the cutaneous application of stimulation and then progressing centrally with more elaborate and potentially dangerous applications. In fact, the clinical research centered on the production of an implantable dorsal column stimulator was based somewhat loosely on the concept that stimulation of this collection of large afferents would produce the desired effect without serious concern for the mechanism of action. The fact that stimulation of the spinal cord was related to the published gate theory in the most nebulous way did not stop the surge of enthusiasm for spinal cord stimulation (30). The primary emphasis for the development of cutaneously applied electrical stimulation as a therapeutic tool came from the failure of spinal cord stimulation to relieve many patients with chronic pain (31). The original applications of spinal cord stimulators were of the phase 2 type, in which a wide variety of painful states were treated with a single modality of therapy and then analyzed retrospectively with regard to pain control. Little was known about the chronic pain neurosis from which the majority of these patients probably suffered (35). It was the failure of pain control in large numbers of such patients that led to a search for a way to predict efficacy of the implanted stimulators. Shealy first employed the commercially available device (the Electreat stimulator) and was probably the first to recognize that some patients found these devices to be adequate therapy and did not need to progress to implantable stimulators for pain control. The unpredictable nature of the stimulation provided by this device, its erratic wave form which could not be studied, and the lack of parameter control led Long

and Hagfors (30) to design and test a stimulator utilizing a modified square wave pulse width and controllable amplitude and frequency. This device went into clinical testing in 1971. At approximately the same time, the Medtronics Company began to provide a complicated transcutaneous stimulator for testing purposes. Both prototypes were utilized in survey studies (phase 2) (Fig. 18.1). Long presented initial data at the organizational meeting of the International Society for the Study of Pain in 1973. With the availability of several models of devices, a number of other investigators began study, and a series of survey reports supported efficacy in pain control (Fig. 18.2).

These initial studies were all of the phase 2 type. They represented broad surveys of an unselected group of patients referred to specialists for pain control. In general, little attempt was made to modify drug utilization or influence behavior. Although the studies were organized in a prospective fashion, the analysis of subgroups was carried out retrospectively. In 1975, Long and Hagfors reviewed the current literature and surveyed a group of physicians interested in chronic pain who used transcutaneous electrical stimulation for their opinion of efficacy. The results were remarkably consistent. Approximately one-third of this unselected group of patients achieved satisfactory pain control, a sur-

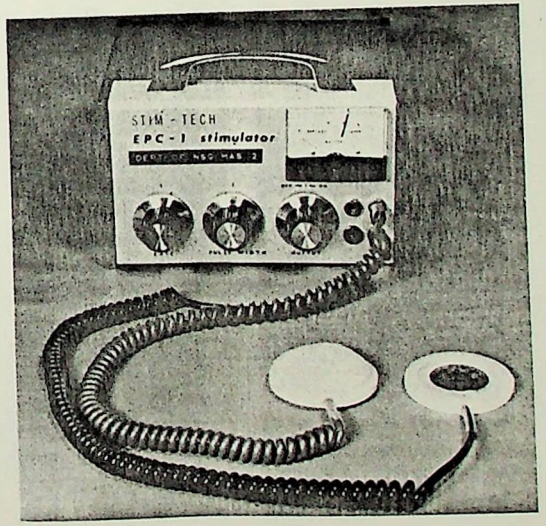

FIG. 18.1. A prototype stimulator. Vintage 1972 with which the original studies were done.

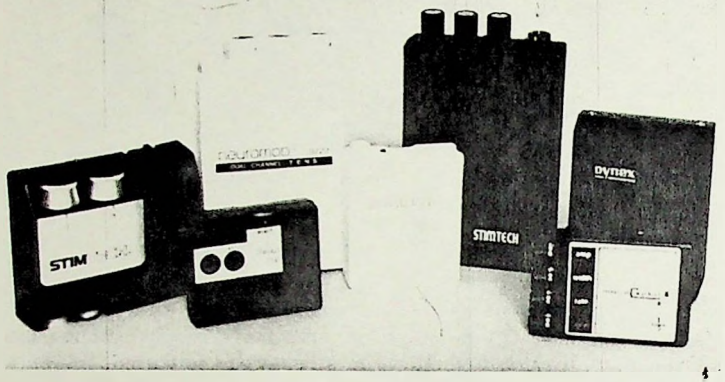

FIG. 18.2. A collection of some of the more commonly used modern stimulators, illustrating the significant improvements in engineering design.

prising figure in view of the fact that the patients treated were generally intractable for any other form of therapy. Furthermore, some groups of patients seemed to do significantly better than others. Those with peripheral nerve injuries responded much more frequently. Other patients were never improved. Those with central nervous system injury or major psychological problems were generally not helped by the technique (30).

CONTROLLED INVESTIGATIONS WITH TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION (TENS)

Following the demonstration that electrical stimulation might have a beneficial effect upon pain in these survey studies, a number of individuals have undertaken controlled investigation of the modality. The largest controlled series contains 300 patients treated at the University of Minnesota or Johns Hopkins between 1973 and 1978 (34). The initial evaluation compared transcutaneous stimulation with sham therapy and subliminal stimulation. Patients actually using the stimulator achieved pain relief significantly more frequently than those receiving sham or subliminal stimulation (who responded at an expected placebo rate). A prospective analysis of the response of patients to stimulation with regard to diagnosis was then undertaken. Patients with peripheral nerve injuries of various kinds responded more than 70% of the time. Patients with relatively acute musculoskeletal syndromes and postherpetic neuralgia also benefited regularly, although not as predictably. About one-third of patients with chronic back pain from the failed back syndrome were

helped, irrespective of the cause of the disability. The use of stimulation was beneficial in the treatment of chronic pain in these selected groups of patients in a situation where most had exhausted virtually every other form of pain therapy. Some patients were found not to benefit at all. Those without obvious physical problems who were thought to have concurrent psychological difficulties were not helped for more than a short period and frequently claimed to be worsened by the technique. They were extremely intolerant of the stimulation. Patients with metabolic peripheral neuropathies characterized by hyperesthesia or serious sensory loss were generally not helped. Central pain states such as spinal cord injury or thalamic syndrome did not benefit.

Over this same period of time, a large number of controlled studies clearly demonstrated the value of electrical stimulation to be greater than that expected from nonspecific factors in pain control (so-called placebo). Stimulation was investigated with peripheral nerve injury, as contrasted with acupuncture and placebo, and was investigated in an inpatient and an outpatient in a physical medicine setting. All reported studies have demonstrated greater than placebo responses and variability of response between diagnostic groups (1, 3, 23, 46, 51, 60, 62). The results in peripheral nerve injury are spectacularly good, and many other forms of chronic pain are benefited (10, 39). Considering the fact that most of these patients have exhausted other forms of pain control and that the techniques have been employed without strict attention to related factors, such as drug misuse, anxiety, or depression, it is probable that transcutaneous electrical stimulation represents the single most effective physical entity yet introduced in the management of chronic pain (11).

RESEARCH AND IMPROVEMENTS IN THE TECHNIQUE

Since the late 1970s, when phase 3-controlled studies demonstrated the specific value of transcutaneous electrical stimulation for pain control, there have been a large number of studies detailing technical advances and the exploration of parameter changes which may make the technique more effective (9, 41, 52, 59). The original stimulation techniques used spike or modified square wave pulses with pulse widths in frequencies ranging from approximately 40 to several hundred and in voltages which were quite variable but were sufficient to provide long-lasting discernible stimulation. The devices were all battery powered. The original stimulating machines were large, but successive improvements led to rapid miniaturization. Solid state circuitry has led to even more impressive advances, and small programmable devices are now available. The original manufacturers were Stimulation Technology (see Fig. 18.1) and Medtronics Incorporated. Since the design and manufac-

turing concepts are relatively simple, many manufacturers rapidly entered the field, and today there is a tremendous diversity from which to choose (see Fig. 18.2). In general there is no evidence that one device works better than another, and the important factors in choosing a stimulator of the conventional type are (a) price, (b) the reliability and stature of the company, (c) the quality of the manufacturer, (d) available software, and (e) ease of service support. The initial enthusiasm which brought large numbers of manufacturers into the field has now subsided, and a smaller number of reliable devices now dominate.

Major advances have occurred in the software. Appropriate electrodes were an early difficult problem. The original devices utilized simple electrified stainless steel plates covered by plastic and sponge to provide stimulation. Now several natural and artificial adhesives which are hypoallergenic and reusable have been developed to make the devices increasingly user-friendly. These electrodes have reduced skin irritation, improved contact and stimulation, and made life much simpler because of their ease of application. However, they have the disadvantage of significantly increasing the cost.

Important new research has been carried out in the modification of parameters to provide better pain relief. It has been suggested that low frequency trains or bursts of stimulation may be more useful. There is evidence that patients who do not respond to conventional parameters of stimulation may be benefited by this low frequency burst mode. The reverse is also true, but at present it is not possible to accurately characterize patients who would respond to one form or the other most predictably. Otherwise, the majority of recent studies relate to clinical usefulness of transcutaneous electrical stimulation in a wide variety of pain control situations. The greatest emphasis has been placed upon the use of stimulation techniques in the alleviation of acute musculoskeletal syndromes, although chronic pain has also been studied further (5, 8, 20, 21, 26, 27, 44, 55, 61, 65). There has been much discussion of optimum techniques for general use (7, 13, 24, 45, 64). More recently, some have explored stimulation of specific nerves, particularly the sympathetic system for therapeutic blockade affects. Such techniques are not in widespread use and remain the province of individual investigators interested in specific pain states. For example, Jenkner and Schuhfried (24) have described temporary electrical sympathectomy from blockade of the stellate ganglion for both diagnostic and therapeutic purposes (24).

TRANSCUTANEOUS STIMULATION FOR POSTOPERATIVE PAIN

Shortly after the introduction of stimulation techniques for chronic pain, Hymes *et al.* began to explore the possibility of achieving relief from incisional pain with electrical stimulation. Their preliminary observations were reported in 1973. Because of the difficulty in assessing the

degree of postoperative pain, these investigators initially ignored pain control and studied other more measurable factors in the postoperative period. In a survey study, they demonstrated decrease in ileus, decrease in atelectasis, improved pulmonary function, decreased intensive care stay, and decreased hospital stay in a series of patients undergoing abdominal and thoracic operations. A number of uncontrolled studies appeared, substantiating these observations and indicating that there was a reduction in the need for narcotics in these same patients. Even though those are not controlled studies, it appeared possible to conclude from them that postoperative pain is controlled by electrical stimulation. The reports available for review indicate that patients achieve satisfactory pain control which satisfied them and those caring for them while using significantly smaller amounts of narcotics than patients who were not treated with electrical stimulation. A few controlled studies have appeared (1, 8, 47, 48, 56, 63). Those studies consistently demonstrate major reductions in narcotics use when postoperative electrical stimulation is utilized. Pain control is satisfactory. Long and Solomon (58) have also shown that transcutaneous stimulation is not useful in patients who were using narcotics routinely before surgery. However, the same group of patients denied significant pain relief with any dose of postoperative narcotic utilized.

Postoperative pain is a difficult phenomenon to study. There is good evidence indicating that for the same operation carried out by the same surgeon, the experience will be quite different from patient to patient. Thirty to forty percent will complain of severe pain, an equal number will have moderate pain, and a smaller percentage (20%) will virtually have no pain at all and have little need for narcotics. For this reason it is very difficult to quantify the pain experience in any reasonable way. Applying the criterion of satisfactory pain control is more productive, although it does not allow the comparisons of degrees of pain relief. When pain relief is considered as the goal, electrical stimulation of the area surrounding the incision will be satisfactory in the majority of the patients and will markedly reduce the need for postoperative narcotics. There is excellent evidence that postoperative complications of intraabdominal and thoracic surgery can be substantially reduced as well. The effective use of stimulation for postoperative analgesia will not be realized, in spite of its apparent advantages over many current techniques for postoperative analgesia, until an accessible system of delivery of this service is available to surgeons.

PRACTICAL USE OF TRANSCUTANEOUS STIMULATION (Tables 18.1–18.4)

Pain As A Medical Problem

It is not possible to utilize transcutaneous electrical stimulation effectively for pain control without understanding the complaint of pain and

TABLE 18.1

Evaluation of Patients for TENS Use

1. Accurate physical diagnosis. You can't just treat a pain.
2. Careful psychosocial evaluation. At least half of patients who arrive at the doctor's office complaining of pain have primarily psychiatric problems.
3. Correction of drug misuse or abuse.
4. Treatment of associated problems such as depression.
5. A thorough trial of TENS by trained personnel.

TABLE 18.2

Clinical Situations in Which Use of TENS Rarely Helps

TENS rarely helps in the following situations:

1. Psychosomatic pain
2. Drug addiction
3. Situations in which secondary gain is important
4. Metabolic neuropathies
5. Spinal injury
6. Pain of central origin

TABLE 18.3

Clinical Situations Warranting the Use of TENS

The uses of TENS are (in order of efficacy):

1. Acute, musculoskeletal injury
2. Acute postoperative pain
3. Phantom, stump, and nerve injury pain
4. Chronic pain of musculoskeletal injury
5. Chronic pain in the multiply-operated low back patient
6. Chronic pain of other causes
   a. Visceral
   b. Sympathetic dystrophies
   c. Postherpetic neuralgia

TABLE 18.4

Effective Use of TENS

1. Thorough trial (30–60 min) by experienced personnel.
2. Patient education in device use.
3. Reasonable number of return visits for reevaluation and reeducation (1 or 2).
4. One month trial at home using a prescribed schedule.
5. The decision for purchase and long term use should be between physician and patient, *NOT* between TENS therapist and patient.

the various factors which may influence both the complaint and the success of therapy. While it is not possible in this discussion to elaborate the psychodynamics of pain and the multitude of factors which are important, it is necessary to provide a background against which the physician can choose appropriate pain therapy. The indiscriminant treatment of pain as a symptom is likely to fail more often than it succeeds. If an entity such as electrical stimulation is applied without selection to a large number of patients, simply treating the complaint of pain, then the physician will be disappointed with the technique. It is common for doctors confronted with this new technique to send a few patients for trials of stimulation. Those patients are usually the most recalcitrant of their practice and the most likely to suffer from a major psychiatric problem. Failure of the technique to relieve the pain is interpreted by the physician to mean that it is valueless for pain control when the error has been in patient selection. Transcutaneous stimulation has been available for 10 years and is now a proven technique for the treatment of chronic pain. It is not ubiquitous in its action, nor is it a panacea. It is a useful adjunct in pain therapy whose success depends upon accurate diagnosis and the selection of appropriate patients for trials of treatment (4). In order to do this effectively it is important to understand pain as a complex problem and to have a reasonable way to approach the patient who comes complaining of disabling pain.

Over the years, we have evolved a practical scheme for evaluating patients with chronic pain. This scheme is based on extensive studies of more than 2000 patients. This is not the place to present the data upon which we base these concepts. However, it is important to summarize them because they allow a framework within which all forms of pain therapy may be employed (36, 38).

Pain as a Symptom of Disordered Thinking

There are a small number of patients who present to the physician in whom the pain is purely a psychiatric problem. The pain exists in the mind of the patient, even though it is described in very real, often florid, terms. Typical patient diagnoses in this group include conversion hysteria, depression, paranoid schizophrenia, and manic depressive disease. Conversion hysteria and masked depression are the most likely diagnoses to be missed in our experience. The key to the conversion hysteric is often the multiplicity of organ systems involved in the complaints without evidence for significant demonstrable disease. Masked depression often presents with intractable headache or back pain which cannot be modified by any therapy until the depression is recognized and treated. It is obvious that if transcutaneous stimulation is utilized in such patients in lieu of appropriate psychiatric therapy, it will fail. Most of these patients will claim to be aggravated by the treatment.

The Magnified Pain Syndrome

There is another group of patients most important for the physician to recognize. Many patients, particularly those complaining of low back pain, have a typical syndrome characterized by the lack of any major

physical abnormalities, complaints of disability which are beyond any physical findings, a fixation upon physical abnormalities, denial of any psychosocial problems, and a fixation upon the pain, which easily becomes a true neurosis. There is usually a history of psychosocial dysfunction which antedates the injury, and the complex is frequently associated with drug-seeking behavior and a desire for disability. Since many of these patients suffer from apparent musculoskeletal abnormalities, transcutaneous stimulation is reasonable to try but most will not respond to any therapies without attention to the entire symptom complex. It is reasonable to treat whatever physical abnormalities can be identified, but the most important approach to such patients is behavioral, and any treatment which neglects the behavioral aspects will undoubtedly fail.

Pain of Physical Origin

The other large group of patients are those we have termed "objective." Such individuals have clear-cut physical reasons for pain, normal premorbid personalities and, generally, the psychosocial influences are otherwise negligible. It is important to recognize that depression and anxiety over the chronic disability are very real parts of the chronic pain syndrome for all patients and are likely to be present in this group. Attempting to treat pain in the presence of unresolved depression is futile, and even in this well-demarcated group of patients, it is necessary to treat depression and anxiety effectively before relying upon any modality of pain therapy. These patients are the ones that have a reasonable chance of responding to physical techniques for pain control. Patients in the other two large groups are really candidates for psychotherapy, and techniques such as electrical stimulation are at best minor adjuncts in their management.

The first step in effective use of any pain therapy is an accurate diagnosis with a decision about the physical modalities should be utilized. An important part of this diagnosis is assessment of psychosocial factors and the understanding that for large numbers of patients, therapy for the complain of pain is useless unless appropriate psychiatric and behavioral therapies occur concomitantly. The successful therapy of pain depends upon a clear-cut diagnosis of its cause; equally clear-cut differentiation of the sensation and suffering purely dependent upon nociception from suffering; and pain behavior modified by personalities, psychological, or social factors. Electrical stimulation is a technique to treat pain and will not influence drug addiction, personality disturbance, personal problems, or the desire for secondary gain (25, 53, 54). Failure to differentiate pain of an ongoing nociceptive input from suffering and behavior induced by these other factors will lead to failure of any physical

modality of pain therapy, including transcutaneous stimulation. The key to successful use of this therapy is an adequate diagnosis and application of the technique to those patients who can be expected to benefit from it. If the complaint of pain is accepted at face value and pain therapies are uncritically applied, then both patient and physician are likely to be disappointed with the result.

Even recognizing the complexity of the complain of pain, it is another matter to evaluate all of its factors. When the syndrome is severe enough or has gone on long enough, a multidisciplinary approach is mandatory to assess all the psychosocial components of the pain syndrome and treat them before proceeding to trials of therapy for the pain (16).

There is now abundant evidence for those situations which are most likely to respond. Incisional pain will be well controlled anywhere the electrodes can be placed to surround the incision. Adequate stimulators and electrodes are available. Since all evidence suggests that pain could be alleviated, postoperative side effects reduced, hospital stay shortened, and nursing time saved, it is difficult to understand why electrical stimulation has not achieved more prominence in incisional pain control. The major difficulty is that no easily accessible method of delivery has evolved in most places. In a few hospitals where interested individuals have established the technique, it is used extensively, but it is not a system which has been freely available to most patients. This pain control method certainly deserves further exploration and in all likelihood wider utilization for postoperative pain (58).

The studies involving acute musculoskeletal pains and the use of transcutaneous stimulation in physical therapy programs are less controlled than those which define its value in postoperative pain and in several chronic pain states (50). Nevertheless, these reports suggest that transcutaneous stimulation can play an important role in pain relief from soft tissue injury. It has been used extensively for athletic injuries and applied to acute low back and neck pain syndromes. Its effectiveness in all of these injuries is often dramatic, and transcutaneous stimulation is now a regularly employed modality in physical therapy units. Again, the lack of a simple way for the physician to prescribe stimulation in these acute situations has limited the effectiveness of the therapy.

Transcutaneous stimulation still finds its greatest use in chronic pain states (4, 34). It is most effective in situations in which the pain is clearly of peripheral nerve injury origin. Neuroma pain in stump and phantom limb, at least early in its development, and virtually all types of traumatic nerve injuries will respond to peripheral stimulation. The stimulation must be delivered proximal to the site of injury, in the area surrounding the denervated part, and hyperesthetic areas must be assiduously avoided. It has been my personal experience that pains of these types will fre-

quently relent after long periods of successful control by stimulation. After one year, many such pains will disappear. The minor reflex sympathetic dystrophies also will respond. In general, it is best to place electrodes over injured nerve trunks proximal to the site of injury or over the plexus to the involved limb. The success in pain of this kind is well documented.

Patients with chronic back pain, uncomplicated by multiple operations, are also benefited in a significant percentage. This varies from report to report but, in general, such patients find stimulation a useful adjunct in an aggressive nonoperative program. When coupled with the usual factors of weight control, exercise, and modification of activities, stimulation is very effective in providing analgesia, particularly for the occasional more severe flare-up. The patient suffering from chronic back and leg pain secondary to multiple operations represents a more difficult problem. The new diagnostic studies available now allow identification of many more correctable abnormalities than in the past have been assumed to exist. When no surgical correction is possible, then electrical stimulation plays an important part in the conservative management. Stimulation provides pain relief and reduction in muscle spasm in a significant number of these patients. The figure has been constant at about one-third in a 10-year experience (11). Considering the fact that these patients rarely have adequate therapy with anything but an aggressive multidisciplinary approach, the number that can be successfully treated with transcutaneous stimulation becomes significant. Most of those patients use their stimulators indefinitely.

Abdominal pain from visceral disease responds predictably. The pain of chronic pancreatitis has been especially treatable. Stimulation takes place posteriorly over the splanchnic outflow and in the celiac area. Most patients respond rapidly (32).

It is equally important to be cognizant of those patients who have little or no chance of benefiting from afferent stimulation. Patients with major psychological or psychiatric components to their complaint will not be helped (48). They may demonstrate short-term response, but that is usually no more than a few days, and is unusual. Patients with these psychosomatic factors often claim to be worsened by the stimulation or find the added sensory input intolerable. It is important to remember that patients with hyperesthesia are temporarily worsened by stimulation. The general experience with patients suffering from pain of central nervous system origin has also been poor. There is no evidence that patients with pain of spinal cord injury or thalamic syndrome are benefited from stimulation. The pain of metabolic peripheral neuropathies usually will not respond, although some reports indicate successful therapy. There has been no consistency in the reports of the use of stimulation

for migraine headache, although an occasional investigator finds the technique useful. Tension headache, on the other hand, will respond promptly to paracervical muscular stimulation.

Stimulation is an adjunct in the overall management of the patient complaining of chronic pain, and like any such technique its use is optimized by trained personnel, patient education, and adequate support for both the physician and the patient. It is not a technique that is likely to be used personally by a busy neurosurgeon. The physician's initial involvement is in diagnosis and the appropriate selection of patients for trials of stimulation. Unless the physician is a pain specialist or physical therapist, it is unlikely that he/she will personally use the device or supervise the trial. The techniques can be utilized by nurses, physical therapists, or trained technicians who are experienced in stimulation use. The first step is the appropriate application of stimulating electrodes in areas which are most likely to relieve the particular complaint of pain (Fig. 18.3*A–D*). This is very important; all too frequently the patient is simply sent to a supplier of the device and instructed only through the manufacturer's advertising material. Stimulating electrodes are not properly placed, and the patient does not know how to use the machine. The results of such an approach are obvious. Appropriately, the first meeting with the patient includes the accurate placement of electrodes for the most likely successful relief of pain and then an educational session with the patient to demonstrate the use of the device. This should include a schedule by which the patients will try stimulation. These sessions should be repeated once or twice to be certain that the patient understands the use of stimulation. Then, the patient can use the stimulator while going about a normal daily routine so as to accurately ascertain its value. In general, it requires several weeks to 1 month to be certain of the effect. Many patients are enthusiastic about pain control in the first few days, only to discover that it is less effective as their enthusiasm wanes. There is little excuse for repetitive returns, and there is no reason at all why a patient has to come to a physician's office or some other facility for regular treatments. These devices can be used at home effectively, and it is much less expensive to do so. If pain relief persists after an adequate trial, the patient can be instructed to buy the device. Here again, the physician's assessment becomes important. Purchase of one of these devices should occur only when adequate trials suggest both to the patient and to the doctor that the stimulator will be of real long-term use in pain control. This decision should not be made by manufacturers or distributors of the hardware or software, and it certainly should not be made without a trial of stimulation adequate to establish efficacy. Even though stimulation has become a commonly used entity, it is unfortunate that few facilities for its appropriate application

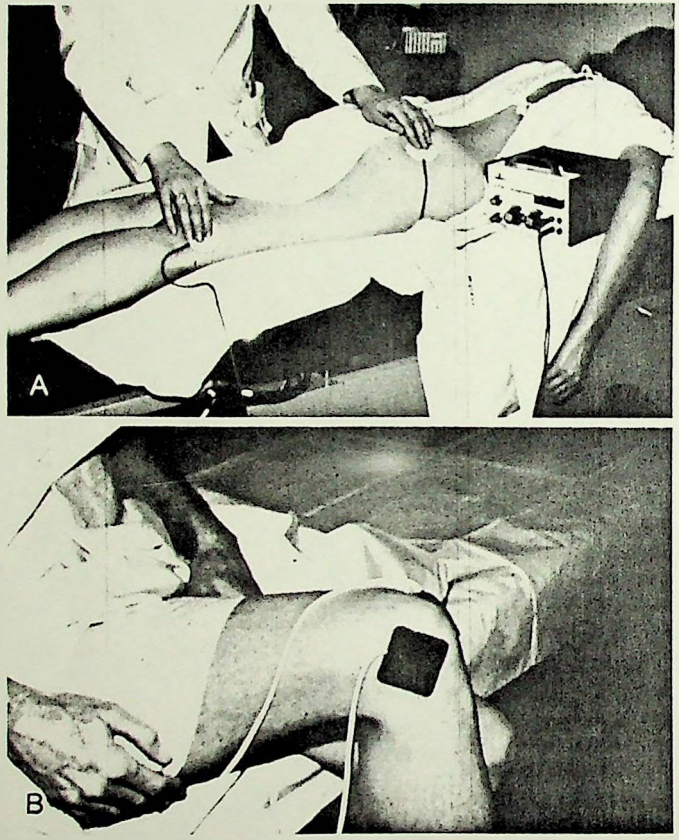

FIG. 18.3. (*A*) Trial stimulation of the sciatic nerve in a patient with sciatica. (*B*) Stimulation across an arthritic knee. (*C*) Stimulation across the brachial plexus in a patient suffering from brachial plexus injury following mastectomy. (*D*) Stimulation across the wrist in a patient with pain of median nerve injury.

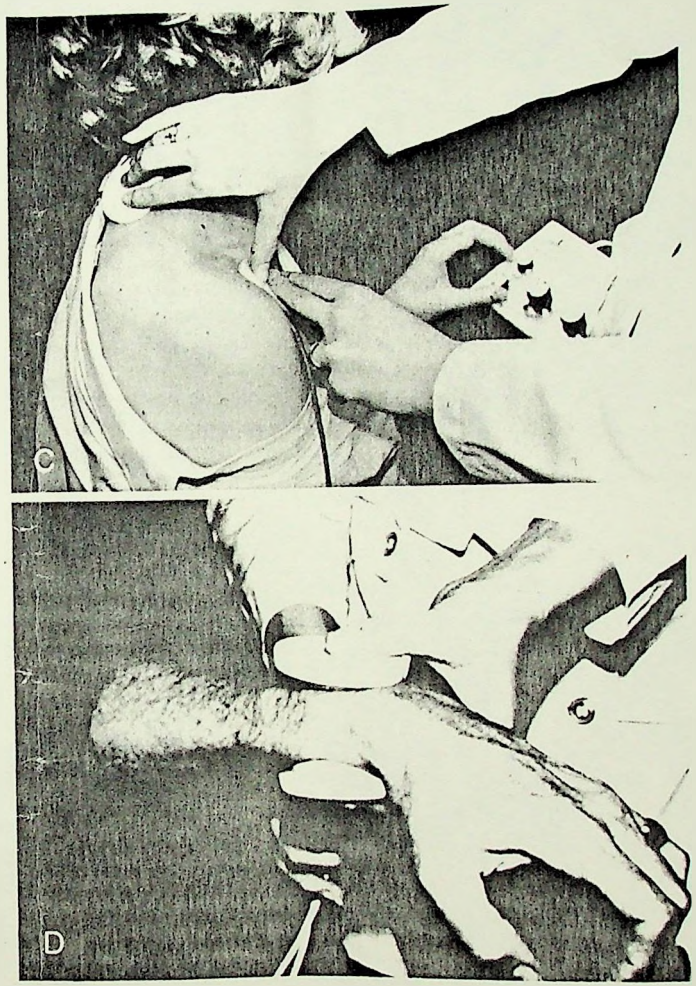

FIG. 18.3*C* and *D*

exist, except under the direction of a relatively small number of pain specialists. This makes it difficult for the average physician to refer a patient for a trial of stimulation. The availability of responsible programs which will provide this service to physicians and their patients will undoubtedly increase the general utilization of stimulation (7).

Optimal use of electrical stimulation for pain control requires that the physician establish a reasonable diagnosis for an ongoing nociceptive input that is likely to respond to this modality. Pain of peripheral nerve injury origin and musculoskeletal pain problems are most favorable for treatment. If the patient's complaint is complicated by important psychosocial factors, then stimulation is likely to be useful only if these factors are approached at least simultaneously with pain therapy. The majority of such patients do not need any physical modality but will respond best to behavioral techniques. The stimulation must be applied by well trained personnel who will educate the patient and service his needs during therapeutic trials. Long-term support for maintenance of hardware and supply of software is required. When these goals are met, electrical stimulation is a valuable adjunct for pain control. When it is applied uncritically to the complaint of pain, it is unlikely to benefit significant numbers of patients (33, 40).

MECHANISM OF ACTION

Given our current understanding of the pathways and mechanisms wherein nociception is appreciated as pain, there are four general possibilities for a locus of action for electrical stimulation of the nervous system. The first is in the periphery at the receptor level. It is possible that the stimulation changes the sensitivity of the receptor (14, 51, 56). There is no evidence for this, and the mechanism remains a source of speculation. Another possibility is blockade of transmission of impulses along nerve trunks. Campbell and Long (11, 44, 66) have suggested that there is some evidence for a potassium block induced by stimulation. The experimental evidence for this is far from solid. A third site of potential inhibition is the dorsal horn. The stimulation activates nonpain-carrying fibers exclusively, at least as much as can be measured by the perception of the patient. (It is very important that stimulation not cause pain if it is to be effective.) Inhibition at the dorsal horn level by interactions between large and small fibers is certainly possible, but supporting evidence is not yet available. A fourth possibility is activation of the descending inhibitory system. This is quite possible and could occur at any level, and there are no data which help us to accept or reject this hypothesis (6).

The relationships of electrical stimulation to the opioid system have been studied. Sjolund and Ericksson (57) were the first to suggest that

low frequency burst stimulation effects were reversible by naloxone administration. While the studies to date do not prove this sensitivity with certainty, they certainly are suggestive that some forms of electrical stimulation are reversible by naloxone. Freeman *et al.* (15) have clearly demonstrated that traditional stimulation is not, and they have also shown that stimulation of the spinal cord and peripheral nerves by the available implantable stimulators is not influenced by naloxone either. These data prove that the stimulation techniques which have traditionally been utilized for pain relief are not dependent upon an opioid system. No information is available concerning the anatomical pathways or the neural transmitters which may be involved.

COMPLICATIONS

The complications reported with electrical stimulation have been minor and very few. Skin irritation from the tape, gels, or electrodes is probably the most common. Burns can occur but are rare. The usual problem is contact dermatitis. Generalized allergies have developed but are extremely uncommon. Occasionally, hyperpigmentation will occur under sites of chronic electrode placement, and neovascularization of the skin may also rarely occur. There have been no instances of serious injury of any kind, and no effects upon other body organs have been reported. The devices should not be used in the proximity of demand pacemakers but can be employed elsewhere in the body in these patients. It is probably wise to monitor the patient in the hospital for a few days when the devices are to be used in this circumstance. As a precaution, it has been recommended that they not be used in patients with serious cardiac irregularities. There is no evidence that they would have any effect upon the heart and, in fact, stimulation has been used successfully to treat angina. They should not be used over the eyes. The question of pain relief followed by resumption of activity with reinjury is very real (19). This is particularly possible in acute athletic injuries, but no such instance has been reported. The technique is basically free of side effects which are of significance.

SUMMARY

Transcutaneous stimulation is a proven effective way to relieve pain. Its optimal use requires an accurate patient diagnosis. Treatment of pain as a symptom only is likely to fail. There must be a careful psychosocial evaluation, for the majority of patients who come to the doctor complaining of pain have major psychological, social, or behavioral factors that are most important in the genesis of the complaint. Drug abuse must be corrected. Related symptoms, such as anxiety and depression, must be treated. Then, a thorough trail of transcutaneous stimulation is manda-

tory. A desultory use will undoubtedly lead to failure. This trial must begin with patient education by experienced personnel. Then the electrodes must be properly applied, and there must be a regular follow-up of stimulation to be certain the patient is utilizing it correctly. The patient must be supported through an adequate trial which should extend over 2–4 weeks before purchase of the device is contemplated. Furthermore, all related nursing and physician personnel must be educated in the proper use of the technique. The uninformed professional who denigrates the therapy is a very effective deterrent to appropriate use. In this situation, transcutaneous electrical stimulation will be of great value in the treatment of acute musculoskeletal injury and acute postoperative pain. It will be effective in the treatment of peripheral nerve injury pain, chronic musculoskeletal abnormalities, chronic pain in the patient who has undergone multiple operations upon the low back and neck, visceral pain, some of the reflex sympathetic dystrophies, and postherpetic neuralgia. Stimulation will not help a complaint which is psychosomatic in origin. It will not influence drug addiction. It is not likely to be useful in any situation where secondary gain is important. The metabolic neuropathies, pain of spinal cord injury, and pain from cerebrovascular accident will not respond frequently enough to warrant more than hopeful trials.

The technique is inexpensive, places the patient in control of his own pain, and has no known serious side effects. Its widespread application awaits the development of reasonable systems to provide this service to physicians and patients. Stimulation-induced analgesia deserves a place in the armamentarium of every physician dealing with the complaint of pain.

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more than two hours previously. The management of young children is more difficult—most episodes are poison scares rather than true poisonings. Rather than give young children charcoal immediately on presentation, we suggest confining it to the few who develop symptoms—in a dose sufficient to increase elimination of the drug.

Repeated doses of oral activated charcoal have not yet been shown to reduce morbidity and mortality. Further studies are required to establish its place and the dose to be given. Until these data are available, severely poisoned adults should be given 150-200 g through a nasogastric tube over 4-8 hours with the aims of achieving a maximum reduction in elimination half life and an improvement in the clinical state. The total dose given is probably more important than the frequency of dosing.

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The cervical spine in rheumatoid arthritis

Needs careful assessment

Rheumatoid arthritis commonly affects the cervical spine, causing several well defined deformities.[1] Damage to the cervical spine from rheumatoid arthritis has been noted in 30% to 46% of necropsy studies and is second in frequency only to that seen in the metatarsophalangeal joints.[2,3] One in four inpatients with rheumatoid arthritis and between 17% and 86% of all patients with this disease have radiological evidence of instability of the cervical spine.[4-6]

These high rates reflect the anatomy of the cervical spine and the dynamic forces that act on it. Each of the apophysial and ligamentous articulations of the cervical spine is susceptible to the same inflammatory changes as those in peripheral joints of patients with rheumatoid arthritis.[7] Furthermore, the cervical spine is constrained between a somewhat rigid thoracic spine and a skull weighing 6 kg; movement of the head, which has been estimated to occur around 600 times each hour, adds to the forces on the articulations.[7]

Any segment of the cervical spine may be affected by the rheumatoid inflammatory process, but destructive changes are most prominent at the occipitoatlantoaxial junction. Atlantoaxial subluxation is the most common deformity and is due to destruction and resultant laxity of the transverse ligament. This allows the atlas to move forward relative to the odontoid process of the axis when the neck is flexed. In radiographs this is seen as a widening of more than 3 mm in the space between the anterior arch of C1 and the odontoid. The corresponding reduction in the space posteriorly restricts the canal available for the spinal cord. By contrast, posterior subluxation of the atlas is infrequent and is seen only in the presence of severe erosion and dislocation of the odontoid.[8]

Recent studies using magnetic resonance imaging in patients with atlantoaxial subluxation have shown an inflammatory mass of granulation tissue around the odontoid arising from the synovial lining of the articulations. This periodontoid mass is not visible in patients who have had surgical fusion of the first two cervical vertebrae or in whom deformity has progressed to that of atlantoaxial impaction (see below).[9,10] The bulging of this mass may further reduce the space available for the spinal cord and cause neurological deficits in patients with only a moderate degree of atlantoaxial subluxation.

When the disease affects one of the occipitoatlantoaxial articulations (termed lateral mass) it may produce the syndrome of non-reducible rotational tilt of the head, the main clinical features of which are occipital pain, tender points in

the neck, and tilting of the head towards the affected side.[11] If both sides are affected collapse of the lateral masses allows the skull to descend on to the cervical spine and the odontoid to enter the foramen magnum. This deformity has been termed cranial settling, superior migration of the odontoid, or atlantoaxial impaction and is seen almost exclusively in association with atlantoaxial subluxation.[6 8 12] Subaxial subluxation is a late development; it often affects several vertebrae, leading to a "stepladder" deformity. Extensive rheumatoid disease of the cervical spine results, then, in a combined deformity of atlantoaxial subluxation—subaxial subluxation and atlantoaxial impaction—a devastating complication and a truly formidable therapeutic challenge.

Deformities of the cervical spine are seen most often in patients with rheumatoid arthritis of more than 10 years' duration. They are usually associated with severe destructive peripheral arthritis, rheumatoid nodules, a high titre of rheumatoid factor, and treatment with corticosteroids.[6 11] Progression of the deformity is unpredictable in a given patient, but follow up for five to 10 years has shown worsening of the instability in 16% to 41% of the patients.[6 8 12] These percentages may be too low: with progression of the deformity to atlantoaxial impaction the magnitude of the atlantoaxial subluxation may seem on radiography to be reduced, giving the false impression radiologically of improvement.[12]

Many patients with rheumatoid disease of the cervical spine remain asymptomatic for years, but they are at risk of a range of neurological complications and even sudden death from medullary compression. Neurological abnormalities may be subtle and difficult to establish in the presence of deforming arthritis, muscular atrophy, and the neuropathy that may be associated with rheumatoid arthritis. Patients may complain of intractable pain in the neck or the back of the head. They may have symptoms of vertebrobasilar insufficiency with vertigo or drop attacks and may have signs of myelopathy.[13 14] Myelopathy, once it develops, is usually rapidly progressive. In patients with subaxial subluxation myelopathy may occur with only slight subluxation because of the narrower diameter of the spinal canal below the axis. Profound and complex neurological deficits may be found in patients with the combined deformity of atlantoaxial subluxation—subaxial subluxation—atlantoaxial impaction. Atlantoaxial subluxation with subluxation of less than 9 mm carries the least risk of neurological damage, while atlantoaxial subluxation of more than 9 mm, atlantoaxial impaction, subaxial subluxation, non-reducible rotational tilt of the head, and combined deformities are all associated with a higher risk of neurological deficit.[8 15]

Plain radiographs of the cervical spine in flexion and extension will allow recognition of atlantoaxial subluxation and subaxial subluxation. In patients with atlantoaxial impaction, however, odontoid erosion and osteoporosis may make plain radiographs inadequate for assessing the extent of cranial settling and resultant penetration of the odontoid into the foramen magnum. Various measurements have been advocated to define the extent of cranial settling. McGregor's line, which assesses the protrusion of the odontoid process above the foramen magnum, is widely used in clinical practice. Because of its superior contrast capabilities magnetic resonance imaging is the current first choice technique for assessing instability of the cervical spine.[16 17]

Patients with a minor degree of atlantoaxial subluxation or with subaxial subluxation need treatment only with a soft cervical collar—which provides symptomatic relief, acts as a reminder to patient and doctor, and may provide some degree of protection from trauma. In the presence of intractable cervical pain, neurological deficits, or myelopathy, or combinations of these, the recommended procedures are halo stabilisation and surgical arthrodesis. The place of surgery in the early stages of instability of the cervical spine is less certain, nor is there any consensus on whether progression can be retarded by early surgery. In a retrospective study of 110 patients with rheumatoid arthritis who had surgical treatment we found recurrence of their cervical instability after a mean interval of nine years in 5·5% of patients with atlantoaxial subluxation who required only atlantoaxial fusion —but a 36% recurrence rate after a mean interval of 2·6 years in patients with atlantoaxial subluxation and atlantoaxial impaction who required fusion from the occiput to C3. No patient with atlantoaxial subluxation and fusion of C1 and C2 progressed to develop atlantoaxial impaction.[14 18]

Many patients with substantial deformities remain asymptomatic, but they are at increased risk of neurological damage with the passage of time. They are also at risk if they need surgery or induction of anaesthesia for any other reason. In one recent study 60% of patients with rheumatoid arthritis having total hip or knee replacements had radiological evidence of instability of their cervical spine, and nearly half of these had no symptoms referrable to their necks.[19] Patients with rheumatoid arthritis undergoing any major surgical procedure should be assessed by having radiographs taken of the cervical spine in flexion and extension. Indeed, all patients with rheumatoid disease of the necks, even though asymptomatic, should be followed up carefully for evidence of neurological deficit, and all should undergo periodic radiographic monitoring.

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(FROM BMJ VOL 306 9 JANUARY 1993 70 M)

Diabetes and its diagnosis

Health Committee
Lok Vidnyan Sanghatana, Maharashtra

What is diabetes? Why does it occur?

When sugar in our blood can not be utilised adequately by the cells in the body, the level of blood sugar increases beyond the normal limit and this is diabetes. In diabetes, not only the sugar in blood but the metabolism of carbohydrates, proteins and fats gets disturbed and a complex disease process sets in.

The food we eat ultimately gets converted in our body into a sugar called glucose. This glucose provides the energy required for the bodily functions. A hormone called "insulin" is essential for the utilisation of the glucose by the cells. Insulin is produced by some special cells in the organ called "pancreas". In diabetes, this insulin is either not produced in adequate quantities or does not get utilised properly, due to which the cells cannot convert the glucose into energy. Instead, it keeps accumulating in the blood. This results in an increased level of glucose in blood.

The exact reason as to why insulin is not produced or utilised properly is not known. To some extent diabetes is hereditary. However, obesity, lack of exercise and improper food habits are known to interfere in the action of insulin. This is seen from the fact that Indians settled abroad with affluent, sedentary, Western lifestyle show a higher ~~percentage~~ prevalence of diabetes.

What are the ill effects of diabetes?

If the level of sugar in blood increases beyond normal limits, it has several ill effects. The fine blood vessels called "capillaries" get damaged due to high blood sugar. This may cause blindness due to bleeding from the capillaries in the retina of the eye. Diabetes increases the chances of cataract or glaucoma (increase in the pressure in the eye-ball).

Diabetes also affects the capillaries in the kidneys, heart, skin or nerves. It also increases the proportion of fatty substances in the blood which in turn leads to a condition called arteriosclerosis (hardening of blood vessels called the arteries). A fatty substance called cholesterol gets deposited on the inner lining of the arteries and thus increasing the resistance to the flow of blood through the arteries. This causes high blood pressure.

Increased blood sugar increases the chances of infection as the sugar itself is a good nutrient medium for the infectious agents to grow. Therefore, the chances of fungal infection of external genital organs, or of urine infection or T.B. are more in diabetic persons. The prevalence of impotence in diabetic men is higher. In diabetic women, if the disease is not kept under check during pregnancy, the chances of still birth or of a deformed foetus are more. In fact a diabetic women should keep diabetes under control before she conceives.

When should diabetes be suspected?

Diabetes usually occurs after the age of 30 or 40 years. There is the other, more severe form of diabetes in which the production of insulin is almost stopped. This can happen in childhood or in young age also. In this juvenile diabetes, the patient becomes weak, despite eating frequently. The person remains ever hungry because despite having lot of sugar in the blood, the body cells cannot utilise it (i.e. they remain starved). The muscles and fats in the body get broken down into sugar in an attempt to provide sugar to the cells. However, it is of no use due to lack of insulin. This break-down of muscles, fat causes loss in weight, tiredness and excess sugar gets filtered down into the urine. The sugar in the urine also absorbs more water from the blood into the urine. This results in frequent urination and persistent thirst. If this is not attended to, the young diabetic falls seriously ill and may develop coma.

The symptoms of diabetes in older age are relatively minor. These patients have frequent hunger and thirst, frequent urination and tiredness as in juvenile diabetes but in a milder form. The patient has to get up at night for urination. Delayed healing of wounds, tendency to develop pus, itching due to fungal infection, ~~and~~ premature dimness of vision, feeling of tingling, numbness in hands and feet, impotency in men and unsuccessful pregnancy in women.... all these symptoms can be due to diabetes. If close blood relatives are/were diabetic, one should be more suspicious about these symptoms. However, absence of these symptoms does not necessarily mean absence of diabetes. Nearly 50% of diabetics do not show these symptoms. Diabetes can be diagnosed with certainty only through blood test. Therefore after the age of forty years, blood sugar should be tested at least once in five years.

How is diabetes diagnosed?

As mentioned above, diabetes can be correctly diagnosed only by blood sugar test. Test for only urine sugar does not give a certain diagnosis. This is because in some cases urine may show sugar with the blood sugar being normal or in some case blood sugar may be high but sugar may be absent in urine.

The blood sugar ~~may~~ is to be examined after the person remains without food, drink (except water) for 10-12 hours. This is called as the "fasting" sugar. If it is more than 140 mg per 100 ml of blood it means the person has diabetes. However, if it is less than 140 mg it does not necessarily indicate absence of diabetes. Therefore in such persons, a test called post-glucose blood sugar is done. In this test the person is asked to take 75 gram glucose in water after fasting for 10-12 hours. The patient is advised not to take anything except water, not to smoke or to exert for two hours after taking glucose. Exactly after 2 hours, another blood sample is taken to test the blood sugar level. If the person has diabetes then the blood sugar level ~~in the blood~~ would be more than 200 mg/100 ml of blood two hours after the glucose meal. Some time instead of giving glucose, the patient is asked to have normal food and the blood is tested two hours after. The test performed by giving glucose is more reliable for correct diagnosis because we are giving measured quantity of glucose. If the sugar level is found to be 115 to 140 mg in fasting sample or between 140 and 200 mg in the sample after glucose/food the diagnosis becomes uncertain. Under such circumstances further testing or frequent testing or testing after controlling diet and doing exercises may be done on the advise of the doctor.

Besides blood sugar what other tests are required?

If diabetes is confirmed by blood-sugar test a thorough physical examination of the patient including weight, blood pressure, examination of the heart, blood vessels, nervous system, retina is carried out because diabetes can affect these systems. Any problem of kidney function can be detected by simple urine examination. If any problem is detected in this urine-examination, further test can be done to know its extent and severity. Urine sugar is also tested. Although, test for blood sugar is must for the diagnosis of diabetes, once it is diagnosed urine can be tested every week at home by the patient himself/herself to get a rough idea as to whether the disease is under control or not.

Initially the urine is also tested for the presence of substances called "ketones". The presence of ketones indicates that the person is suffering from severe diabetes. This calls for immediate treatment. Once diabetes is detected tests for blood fats (lipids) and electrocardiogram of the heart are also done. Diabetes is known to induce abnormal changes in blood-fat and in the ECG. These tests are therefore helpful to decide ~~off~~ the line of treatment.

Treatment of Diabetes

Health Committee
Lok Vidnyan Sanghatana, Maharashtra

What is the treatment for diabetes? Can it be cured?

Diabetes can not be completely cured and once acquired it stays for life. However, with appropriate exercise, diet control and drugs it can be kept under check. Mild diabetes can be kept under control without taking medicines with the help of proper diet and exercise alone. In addition to diet control and exercise, some patients have to take antidiabetic drugs. In some, only tablets are needed, but in some patients, insulin injection is to be taken daily.

Diet: Indian, especially Maharashtrian traditional balanced diet is mainly based on cereals and pulses. There is no qualitative change required in this diet for a diabetic person. One has to avoid sugar, consume only very limited quantities of oil, butter, ghee, etc., take only limited quantities of food to maintain weight within prescribed limits. The formula for prescribed limit of weight is - height in cms minus one hundred = weight in kg. If your weight is more than this limit, with the help of following simple guidelines change your diet to reduce weight.

To reduce 1 kg weight in a month through dieting, one will have to reduce 7200 calories from diet (about 240 calories per day). One hundred calories are provided by the following food items and accordingly the calories can be controlled by appropriate reduction in consumption. Cooked rice - 1 cup - 30 grams of dry rice); one chapati of 6" diameter (made out of 30 grams of wheat); one sada dosa; two idlis; two slices of bread; one cup (katori) liquid dal (in the form of sambar, amti, etc.); one egg; one banana; 2.5 spoons of butter, 2 spoons of oil or ghee.

The following can be consumed in liberal quantities as they have low calories e.g. carrot, cucumber, onion, tomato, lavaki (dudhi bhopla), radish, clear vegetable soup, etc.

The following should be taken only in moderate quantities: (The calories contained in the oil used for cooking have not been taken into account. Oil should be used to the minimum). Vegetables like cabbage, cauliflower, ladies finger, brinjal, etc. Fruits such as papaya, sweet lime, oranges, figs, etc. Fluids such as butter milk and milk without cream, tea without sugar, fish, lean chicken, etc.

The following should be taken in very small quantities: potato, sweet potato, yam, sago, green peas, guava, banana, mango, custard apple, etc.

The following should be avoided as far as possible: Sugar, gud or jaggery, honey, sweets, ice-cream, jams, pastries, cream, ghee, butter, fried foods, oily pickles, groundnuts, almonds, cashew. They are calorie-rich substances.

Alcohol also has calories like sugar. It should be avoided. This is particularly so when the diabetes is severe and not under control or when other complications are present. Eat a little at a time, four times a day so that the sugar level in blood does not rise suddenly.

Use only limited quantity of oil for cooking. Daily 20 grams of oil per head i.e. about 0.6 kg per head per month is the limit. Other fats particularly animal fats such as ghee, butter should be avoided as they are much more likely to increase cholesterol in diabetics. Home made ghee may be used but it should be restricted (only one tea spoon in a day (10 grams).

Diabetes may also result in other problems relating to heart and kidneys. If this happens, this would need further diet control, which should be done under appropriate medical advice. There is a general misconception that diabetic should not eat rice. As a matter of fact rice has less calories as compared to chapati or roti (weight for weight). However, since rice has less fibre content its digestion is quicker which may increase blood sugar. There is no harm in eating limited amount of rice.

Exercise: Exercise reduces the amount of insulin required to control blood sugar. With the help of proper diet and exercises it is possible for some diabetics either to avoid use of drugs altogether or reduce its dose. Therefore diabetics must do some light exercises. Many diabetics are overweight and exercise helps to reduce it. Walking is the simplest and the safest exercise. One should walk briskly for at least half hour to stay fit, and for longer time to reduce weight. To reduce 1 kg in a month one has to burn 7200 calories through exercise. Keeping in mind the need to reduce weight to the desired level within 3-6 months, the diet and exercises have to be planned.

Types of activities and calorie consumption per minute: Cleaning household utensils, mopping, gardening, painting - 2 to 5 cal; works like masonary - 4 to 5 cal; brisk walk, climbing stairs, cycling, tennis, carpentry - 5 to 10 cal.

In fact exercises are of three types and the exercise programme preferably should include all the three. Please refer to the separate booklet by the Health Committee on this subject. Diabetics should consult doctor before starting these exercises, and also observe the following precautions.

A patient taking insulin should eat 25 to 30 gms of carbohydrates (half a banana or guava, half cup milk or butter milk) before exercise. Otherwise exercise may induce sudden drop in sugar level causing giddiness. If the person also has hypertension or heart ailment, only such exercises which can be easily tolerated should be done in consultation with the doctor. However there is no need to consult a specialist if one chooses only brisk walk as exercise. If there is a problem in the retina of the eye, exercises which include jerky movements and lifting of weights should be avoided since they may trigger bursting of already weakened capillaries. Exercise causes quicker absorption of insulin injected on arm or thigh. If the time of exercise coincides with that of the injection, the injection may be taken under the skin on the abdomen.

Medicines: The prescribed medicines should be taken at the right time in correct dosages and should not be changed without medical advice. Diabetes cannot be cured and therefore diet control should always be properly observed. In some cases insulin is needed only for temporary reasons such as during stress like pregnancy or during illness. Once the stress is over, insulin can be replaced with tablets.

Diabetes may cause symptoms such as tingling and numbness or burning or pains in hands and feet. Sometimes `B' vitamin is prescribed to treat these symptoms but its utility has not yet been scientifically proved. However, due control on the blood sugar can reduce these problems in some patients.

How to know whether diabetes is under control or not?

Symptoms of diabetes recede as diabetes is controlled. The true indication of control of diabetes is level of blood sugar which to some extent can be gauged by testing urine sugar. Urine sugar can be tested at home but it is not very dependable. Its utility for a particular patient is determined by the doctor depending upon factors like the severity of diabetes and patient's attitude.

If the patient does not have to take insulin it is good to test the blood and urine sugar every month (however, if the patient cannot afford it, then at least every three months) to know whether diabetes is under control. If the patient is on insulin then more frequent testing for blood-sugar is required depending upon the severity of the disease.

The usual blood sugar test indicates whether the sugar has increased beyond normal limits on that particular day. A test called 'Glycosylated Hemoglobin' shows the control on blood sugar during the preceding 3 months. Since this test is more expensive costing about Rs. 150 to 200, doctors do not advice it to all the diabetics. However, if the doctor suspects that the patient does not take the necessary precautions related to food, exercise and medicines, this test becomes quite necessary.

In the initial stages of the treatment when there is a need to reduce weight, the patient should keep a note of his/her weight every 15 days for a few months and if the progress is satisfactory, a monthly check-up weight is enough.

Once in a year it is necessary to do a general physical check up and examination of eyes, urine, blood cholesterol, ECG as diabetes increases the possibility of certain problems in the eyes, kidneys, and the heart. These diseases are symptomless in the initial stages and hence they aggravate silently. These tests are therefore needed to detect these diseases at an early stage. This is more so for certain retinal problems which otherwise could cause blindness. This blindness can be prevented by timely check up and treatment of the disorder at an early stage.

What other care is required?

Any increase or recurrence of symptoms of diabetes should be reported to the doctor and be properly treated. Any infection should also not be ignored and a prompt, proper treatment for that infection is a must, as any infection can quickly become serious in a diabetic person.

Specific time table for for food and medicines should be strictly observed. An excess dose of insulin or a powerful oral drug in relation to diet may cause a sudden drop in the level of blood sugar. If this happens, the patient may get profuse sweating, giddiness, uneasiness and even unconsciousness. In this situation 4-5 spoonful of sugar should be taken. A patient on insulin or powerful oral anti-diabetic drugs should carry a small plastic packet of sugar (about 5 spoonful) in his/her packet. Patients taking insulin should carry an identity card with a drug chart indicating the drug used and dosages.

Diabetics should avoid all forms of tobacco as it increases risk of heart diseases. They should avoid walking barefoot, should cut their nails properly and do proper early dressing for all kinds of injuries including small cuts as diabetics are more prone to get their wounds infected fast and develop a septic.

DIS-JAN 15

NATIONAL CAMPAIGN

ON

DUST RELATED LUNG DISEASES

UPDATE

Dear Friends,

It was not long ago that you received the last update. During this period two major tragedies struck our nation; one was the earthquake in Maharashtra and the other, fire at New Kenda Colliery at Asansol, West Bengal. We had human loss in both of this, but only difference is, whereas earthquake was natural and unpredictable, accident in New Kenda Colliery was man-made and predictable. Who ever works or gets opportunity to visit coal mines of our country knows in what hazardous conditions miners work in them. Many significant and important safety operations are neglected. The inquiry reports of Chasnala and Mahabir collieries accidents had already named management for failure in safety standards. Past records also show that there has been twenty five percent increase in the accidents in the coal mines in the country. But this astonishing fact has not opened the eyes of either the managements or the policy makers. We express our deep sorrow and solidarity with the Trade Unions of Asansol and the family members of those who have sacrificed their life in this race of blind industrialisation.

1. A significant development for NCDRLD, is the announcement of shifting its secretariat to Baroda. If you remember in the last National Convention which we had in Delhi in December 1992, it was said to shift secretariat rotation wise so that issues and problems can be highlighted. This will also give this campaign a true national character.

2.On December 17, 1993, a meeting of the active partners of NCDRLD was convened at Baroda. 25 participants from Trade Unions, Non-Governmental Organisations, Government and Research Institutes and activists attended this meeting. Mr. Jagdish Patel introduced the agenda to the participants, which was followed by the presentation of the report of last year's activities of the Campaign by Mr. Harsh Jaitli. Most of the activities are reported in the previous update.

3.Mr.H.P. Mishra of MKSS Mandal, Ahmedabad discussed at length about the movement for compensation and prevention of byssinosis, which is going on in Ahmedabad. While appreciating the efforts made by ESI Scheme of Gujarat for identifying cases of Byssinosis, he criticised ESI Corporation for delaying the process of disability assessment and compensation payment to the sufferers. According to Mr. Mishra - workers are waiting for their turn to get compensation. He also expressed the need for spreading the same type of movement in the areas like Bombay and Kanpur, where textile industry is concentrated.

4. Mr. Jagdish Patel and Mr. Aman Vyas shared their experiences of working with Silicotic workers at Khambhat (Gujarat). Mentioning about the difficulties faced by them he said that, in Khambh at the employers pressurised the sympathisers of the movement not to adopt safe technology. The employers lobby used Government officials in their efforts to stop safety measures. The Co-operative employees were dragged to the court on flimsy charges, for which court fined them. " We had our limitation of providing defense to the sympathetic employers. These were the problem faced by activists in early studies" he said. One of the Semi-Government organisations has developed the safe-technology upon request by the campaign but this machine still requires some modifications in design before it becomes acceptable to polishers of different shapes. The machine now prepared, is useful to only one for type of shape. It was also felt that the polishing work is spreading to the other new regions where the campaign activities are needed to be initiated. Last year, these agate workers were given coverage of life insurance under scheme for rural workers by Life Insurance Corporation. JANPATH, an Ahmedabad based NGO worked as nodal agency. Its representative Aman Vyas informed that among 900 workers covered under this insurance scheme, 25 had already died of silicosis. The LIC felt that this death rate is very high and so they hiked the premium rates. Which was double of the original rate of Rs.15. JANPATH is busy negotiating. Ms. Rani Advani of CERC, Ahmedabad, offered her help for litigation against LIC, on this issue.

Secretariat :- PTRC : 63, Snehsmruti Society, (Behind Nutan Maheshwar Society)
Shubanpura, Baroda - 390 007, Gujarat

5. Com. Rajkumar Singh, AITUC, Baroda, expressed a need to form a local committee on ESI in Baroda to look after the problems. During discussion ESI was frequently attacked by the participants. So, in order to clarify the stand Dr. Charulata Shah, Dy. Director, ESI Scheme, Gujarat State, gave details of the working of ESI. She informed that the most of the criticism of ESIS is for monitory problem, which is handled by ESI Corporation. She informed delegates that the ESI Scheme gets money from corporation. The amount, the scheme gets from corporation is too less. The State Government spent Rs.4-5 crore in excess and the State Government is now asking the Corporation for more money.

6.Dr. J.R. Parikh of NIOH gave details of his recent research on Byssinosis. His research is now focused on prevention part and not on identification. He has reached to the conclusion that, it is the leafy part of the cotton-plant which is responsible for the disease. He further informed that if better quality of cotton is used and process of cotton-picking is modified, the dust level can be reduced. He also explained the design of the training programme on Byssinosis. He mentioned about the administrative problems during the training of the first batch, which was taken care of by the arrival of the subsequent batch. All the members supported this strategy, and requested the secretariat to circulate a copy of his lecture.

7.In the later half of the meeting a budget and activity plan was charted out. It was unanimously agreed to raise funds for NC-DRLD, through contribution. Major activities included publication and distribution of four updates every year. In order to motivate and support activities on dust related lung diseases, traveling was also suggested.

All these activities, along with other expenses like postage-photostat and other administrative assistance would amount to Rs.1,00,000. This does not include salary of the Co-ordinator and office rent. So need was felt to raise fund through appeal. Accordingly we are sending you herewith an appeal for the contribution and look forward for your active involvement.

Role of Secretariat was also discussed. It was clear from the discussion that partners find the role of Secretariat as a major linking force. Secretariat will not only publish Updates, but also disseminate, information and create a data bank with the help of PRIA and other Research Organisations. It will also assess the needs of the partners and link them with the available facility.

The meeting concluded with a note of thanks for the AITUC, Baroda who, provided its office for this meeting.

We look forward to your continued support and involvement in the campaign and your suggestion for making it more effective.

With best wishes

Yours sincerely

Harsh Jaitli

APPEAL FOR SUPPORT

National Campaign on Dust Related Lung Diseases was initiated in 1990. PRIA, Delhi nurtured the campaign since its inception by providing secretarial support. The major activities undertaken by the NCDRLD during that period were National convention, poster competition, and research studies and popular publications. The campaign has achieved very positive results so far in some areas. Yet it has to go a long way to achieve its objectives. During all these years, Trade Union and NGOs, doctors, lawyers and Government institutions have become partners in our struggle to achieve dust free working environment. The campaign needs to be strengthened further. You have been involved in the campaign activities for quite sometime, now this update comes to you with an appeal for your contribution.

Jagdish Patel, PTRC, 63, Snehsmruti Society, (Behind Nutan Maheshwar Society)
Shubanpura, Baroda - 390 007, Gujarat

The cheque/draft should be in favour of NC-DRLD A/C

DIS-1A.16

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TRANSMISSION OF KYASANUR FOREST DISEASE VIRUS BY *HÆMAPHYSALIS TURTURIS*, *HÆMAPHYSALIS PAPUANA KINNEARI* AND *HÆMAPHYSALIS MINUTA*.

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Out of 14 species of ticks of the genus *Hæmaphysalis* found in the Kyasanur Forest disease (KFD) epidemic area (Trapido *et al.*, in press), isolations of KFD virus have been made from seven : *H. spinigera*, *H. turturis*, *H. papuana kinneari*, *H. leporidarum*, *H. kyasanurensis*, *H. minuta* and *H. wellingtoni* in that order of frequency (Virus Research Centre, unpublished data). Of the above, laboratory transmission of KFD virus has so far been reported for *H. spinigera* (Varma *et al.*,

The present communication deals with the studies on experimental transstadial transmission of KFD virus by *H. turturis*, *H. papuana kinneari* and *H. minuta* and transmission of the virus by bite to susceptible animals. These three species were selected because they have been successfully reared in the laboratory. Attempts to rear other species have not yet been successful.

MATERIALS AND METHODS.

GH1338 strain of KFD virus, originally isolated from a mixed pool of larvæ and nymphs of *Haemaphysalis sp.* collected in the Kyasanur Forest, was employed throughout. It had undergone three intracerebral adult mouse passages and five intramuscular passages of infected blood in one-day old white leghorn chicks.

All of the species of ticks in this study were obtained by 'flag dragging' or direct picking from leaves in the forest of Hillemarur, Shimoga District, Mysore State, India. *H. turturis* and *H. papuana kinneari* were collected as unfed adults and *H. minuta* as unfed nymphs and subsequently reared in the laboratory. After laying the adult females were tested individually for presence of virus as described below. Progenies from ticks found uninfected were employed in these experiments.

The ticks were infected as 10 to 15-day old larvæ on two-day-old white leghorn chicks which had been inoculated intramuscularly 24 hours before with approximately 10^3 LD_{50} of KFD virus. Some of the freshly engorged larvæ were crushed and the suspensions inoculated into mice to assure that they had ingested virus while feeding ; the remaining fed larvæ were kept in desiccators maintained

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at 25°C. to 28°C. and 90 to 95 per cent relative humidity (R.H.) and allowed to moult into nymphs.

When the nymphs were ready for feeding, some were tested for the presence of virus. The remaining nymphs, in pools of five, were released on one-day-old chicks to determine their ability to transmit virus by bite. Most of the nymphs released on chicks attached within 24 hours and became fully engorged in three to four days. Some, which did not attach, died very soon and were discarded. Blood from the chicks was drawn into heparin on the third day and on the fifth day after the release of the nymphs, and was tested for the presence of circulating virus. Suspensions of the fed nymphs from each chick were also tested for virus.

Other nymphs, in groups of 20 to 30, were fed on one-day-old chicks. These fed nymphs were not tested for virus but kept at 25°C. to 28°C. and 90 to 95 per cent R.H. for moulting into adults. The chicks were bled into heparin on the fourth day after the release of the nymphs and the bloods were tested for circulating virus.

Only the adults of *H. minuta* were fed on one-day-old chicks to determine the transmission of virus by bite since the adults of the other two species of ticks did not feed on any KFD susceptible laboratory animal. Because of an acute shortage of mice it was not possible to test for circulating virus, therefore, the chicks on which adults of *H. minuta* had fed were bled 22 days after the release of ticks and their serum tested for the presence of antibodies. The adults of *H. papuana kinneari* and *H. turturis* were processed and inoculated into adult mice in order to detect the presence of virus.

Suspensions of ticks were prepared by grinding larvæ and nymphs in 1·0 ml. to 1·5 ml. and adult ticks in 2·0 ml. of 0·75 per cent bovine albumin in phosphate saline (BAPS), pH 7·2, containing 1,000 units of penicillin and 1·0 mg. of streptomycin per ml. The suspensions were centrifuged at 2,000 r.p.m. for ten minutes. All search for and titrations of virus were done in adult mice inoculated intracerebrally. Titres of virus have been expressed as LD_{50} per 0·03 ml.

Serological confirmation of the virus present in tick suspensions and chick bloods was made sufficiently often at all stages of the experiments to provide reasonable assurance that the agent being transmitted was in fact KFD virus. The virus was identified by complement-fixation tests employing a crude 10 per cent suspension of infected mouse brain as antigen. Immunity in chicks was tested by hæmagglutination inhibition (HI) technique (Clarke and Casals, 1955).

RESULTS.

A. *Hæmaphysalis turturis*

(*i*) *Trans-stadial transmission of virus.*—The titres of the circulating virus on the fourth PI day of the four donor chicks fed on by larval tick batches 65, 66, 67 and 68 were $10^{5\cdot3} \geqslant 10^{5\cdot1} \geqslant 10^{5\cdot5}$ and $10^{5\cdot0}$ LD_{50} for adult mice, respectively (Table I). Suspensions prepared from each batch consisting of five fed individual

larvæ and three pools of five larvæ each, were tested for virus soon after dropping from the host. All were found to contain virus.

TABLE I.

Trans-stadial transmission of KFD virus in H. turturis.

Batch number.	Circulating virus titre of donor chicks on 4th PI day.	Fed larvæ: 5 larvæ in a pool.	Fed larvæ: Individual larvæ.	Unfed nymphs: 5 nymphs in a pool.	Unfed nymphs: Individual nymphs.	Unfed adults: Individual females.	Unfed adults: Individual males.
65	$10^{5.0}$ LD_{50}	3/ 3*	5/ 5	3/ 3	5/ 5	11/15	10/15
66	$10^{5.4}$ LD_{50}	3/ 3	5/ 5	3/ 3	5/ 5		
67	$10^{3.4}$ LD_{50}	3/ 3		3/ 3	5/ 5		
68	$10^{3.0}$ LD_{50}	3/ 3	5/ 5	3/ 3	4/ 5	9/15	5/15
Total		12/12	15/15	12/12	19/20	20/30	15/30

* Numerators are the number positive for KFD virus; denominators are the number tested.

Similarly, suspensions prepared from each batch consisting of five unfed individual nymphs and three pools of five nymphs each were tested for the presence of virus 13 to 17 days after moulting and 25 to 30 days after the termination of the infective feed as larvæ. KFD virus was isolated from all the individuals and all of the pools of the first three batches. In batch 68 all three pools and four out of five individual nymphs were positive for KFD.

Twenty to 30 days after the emergence of the adults, 90 to 95 days after the end of the infected blood feed in the larval stage, the ticks from only two batches, 65 and 68, were tested for the presence of virus. KFD virus was isolated from ten out of 15 males and from 11 out of 15 females of batch 65, and from five out of 15 males and from nine out of 15 females of batch 68.

(*ii*) *Transmission of virus by infected nymphs to chicks.*—Unfed nymphs in groups of five from batches 65 and 68 were released on nine one-day old chicks (Table II). Transmission of virus by bite was accomplished in all cases; fed nymphs from each were also shown to contain virus following inoculation into adult mice. In a second series, groups of 20 to 40 nymphs from both batches were released on nine one-day old chicks. KFD virus was isolated from the bloods of all the chicks.

B. *Hæmaphysalis papuana kinneari:*

(*i*) *Trans-stadial transmission of virus.*—Larvæ from batches 112, 113, 114 and 115 were released on four infected chicks. The titres of the circulating virus on the fourth PI day of these four chicks were $10^{5.0}$, $10^{5.2}$, $10^{4.7}$ and $10^{3.8}$ LD_{50} for adult mice respectively. Suspensions prepared from each batch consisting of five fed individual larvæ and three pools of five fed larvæ each were tested in adult mice for the presence of virus. All of the individuals and pools of freshly engorged larvæ in batches 112, 113 and 114 and four out of five individual larvæ and two out of three pools in batch 115 were found to contain virus (Table III).

TABLE II.

Transmission of KFD virus to chicks by bite of infected nymphs of H. turturis.

Batch number 65: Chick number.	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs.*	Transmission results.	Batch number 68: Chick number.	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs.*	Transmission
454	5	3	+	+	458	5	4	+	+
455	5	4	+	+	459	5	3	+	+
456	5	5	+	+	460	5	3	+	+
457	5	4	+	+	461	5	2	+	+
529	20	7	NT	+	462	5	2	+	+
530	20	12	NT	+	533	30	18	NT	
531	20	18	NT	+	534	30	10	NT	
532	20	8	NT	+	535	30	28	NT	
					536	30	28	NT	
					537	40	38	NT	+

* + = Virus detected. − = Virus not detected. NT = Not tested.

TABLE III.

Trans-stadial transmission of KFD virus in H. papuana kinneari.

Batch number.	Circulating virus titre of donor chicks on 4th PI day.	Fed larvæ: 5 larvæ in a pool.	Fed larvæ: Individual larvæ.	Unfed nymphs: 5 nymphs in a pool.	Unfed nymphs: Individual nymphs.	Unfed adults: Individual females.	Unfed adults: Individual male
112	$10^{5.0}$ LD_{50}	3/ 3*	5/ 5	2/ 3	4/ 5		
113	$10^{5.2}$ LD_{50}	3/ 3	5/ 5	1/ 3	2/ 5	0/15	0/1
114	$10^{4.7}$ LD_{50}	3/ 3	5/ 5	0/ 3	0/ 5	0/15	0/1
115	$10^{3.8}$ LD_{50}	2/ 3	4/ 5	0/ 3	0/ 5	0/15	0/1
Total		11/12	19/20	3/12	6/20	0/45	0/4

* Numerators are the number positive for KFD virus, denominators are the number tested.

From among the nymphs which emerged from the remaining larvæ, suspe sions were prepared from each batch consisting of five unfed individual nymphs an three pools of five nymphs each and were processed for virus isolation 15 to 22 da after moulting and 33 to 35 days after finishing their larval feed. KFD virus w detected in four out of five individual nymphs and two out of three pools of nymp from batch 112 and two out of five individual nymphs and one out of three poo of batch 113. No virus was detected from either individual nymphs or pools nymphs of the other two batches.

No virus was isolated from the unfed adults emerging from the three batch 113, 114 and 115, when individual suspensions of 15 males and 15 females fr each batch were inoculated into adult mice 78 to 80 days after the termination

TABLE IV.

Transmission of KFD virus to chicks by bite of infected nymphs of Hæmaphysalis papuana kinneari.

Batch number 112: Chick number	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs*.	Transmission results.	Batch number 113: Chick number	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs*.	Transmission results.	Batch number 114: Chick number	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs*.	Transmission results.	Batch number 115: Chick number	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs*.	Transmission results.
621	5	3	+	+	626	5	5	+	+	631	5	2	+	+	636	5	2	–	–
622	5	2	+	+	627	5	5	+	–	632	5	4	–	–	637	5	4	–	–
623	5	5	+	+	628	5	5	+	–	633	5	5	–	–	638	5	1	–	–
624	5	5	+	+	629	5	4	–	–	634	5	4	–	–	639	5	5	–	–
625	5	4	+	+	630	5	4	–	–	635	5	4	–	–	640	5	3	–	–
					658	30	18	NT	NT	663	30	22	NT	NT	653	30	26	NT	–
					659	30	22	NT	NT	664	30	24	NT	NT	654	30	22	NT	NT
					660	30	19	NT	–	665	30	23	NT	NT	655	30	20	NT	+
					661	30	27	NT	+	666	30	26	NT	NT	656	30	24	NT	–
					662	30	22	NT	–	667	30	22	NT	NT	657	30	21	NT	–

* + = Virus detected. – = Virus not detected. NT = Not tested.

their larval feed and 12 to 17 days after moulting. No adult could be obtained from batch 112 in this part of the experiment as very few nymphs were obtained and all of them were used in the earlier part of the experiment.

(*ii*) *Transmission of virus by infected nymphs to chicks.*—Unfed nymphs in groups of five were released on 20 one-day-old chicks. Transmission of KFD virus was successful with all the five groups of batch 112 (Table IV). In batch 113, although three out of five pools of fed nymphs were found infected, transmission could be shown by only one group. There was no transmission of virus by batches 114 and 115 and no virus was isolated when suspensions of the fed nymphs were tested in mice. In the second series of experiments when 15 groups of unfed nymphs each consisting of 30 nymphs from batches 113, 114 and 115 (five groups from each batch) were released on 15 chicks, transmission by bite was accomplished in three instances in batch 113 and once only in batches 114 and 115.

C. *Hæmaphysalis minuta* :

(*i*) *Trans-stadial transmission of virus.*—Two batches of larvæ (69 and 70) were fed separately on two infected chicks. The titres of the circulating virus on the fourth PI day of these two chicks were $10^{4.5}$ and $10^{4.2}$ LD_{50} in adult mice respectively. Suspensions prepared from both batches consisting of five fed individual larvæ and three pools of five fed larvæ each were processed soon after feeding, and all were found to contain virus (Table V).

TABLE V.

Trans-stadial transmission of KFD virus in H. minuta.

Batch number.	Circulating virus titre of donor chicks on 4th PI day	Fed larvae: 5 larvæ in a pool.	Fed larvae: Individual larvæ.	Unfed nymphs: 5 nymphs in a pool.	Unfed nymphs: Individual nymphs.	Unfed adults: Individual females.	Unfed adults: Individual males.
69	$10^{4.5}$ LD_{50}	3/3*	5/ 5	2/3	2/ 5	5/10	7/10
70	$10^{4.2}$ LD_{50}	3/3	5/ 5	3/3	1/ 5	0/10	0/10
Total		6/6	10/10	5/6	3/10	7/20	7/20

* Numerators are the number positive for KFD virus, denominators are the number tested.

Similarly, suspensions prepared from both batches consisting of five unfed individual nymphs and three pools of five nymphs each were tested for virus 28 to 30 days after the end of their infective feed as larvæ and 10 to 20 days after moulting. KFD virus was isolated from two out of five individual nymphs and two out of three pools of nymphs from batch 69 and from one out of five individual nymphs and from all of the three pools of batch 70.

The unfed adults (10 males and 10 females from each batch) were processed for the isolation of virus 80 to 82 days after the end of their infective feed as larvæ and 16 to 22 days after moulting. In the first batch five out of ten females and seven out of ten males were infected ; in the second batch only two out of ten females contained virus.

(*ii*) *Transmission of virus by infected nymphs and adults to chicks.*—In the first experiment nine groups of five nymphs each representing the two batches were released on nine chicks. Transmission was successful in two instances, once with each batch (Table VI). In the second experiment, groups of 30 nymphs from both batches were released on ten chicks. KFD virus was isolated from the bloods of all the ten chicks.

TABLE VI.

Transmission of KFD virus to chicks by bite of infected nymphs of H. minuta.

Batch number 69:					Batch number 70:				
Chick number.	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs*.	Transmission results.	Chick number.	Number of nymphs released.	Number of nymphs fed.	Presence of virus in fed nymphs.	Transmission results.
382	5	3	—	—	387	5	5	+	+
383	5	3	—	—	388	5	5	+	+
384	5	5	—	—	389	5	5	+	+
385	5	5	—	—	391	5	5	+	+
386	5	4	+	+	398	30	19	NT	+
392	30	24	NT	+	399	30	26	NT	+
393	30	24	NT	+	401	30	29	NT	+
394	30	18	NT	+	402	30	28	NT	+
395	30	26	NT	+					
396	30	19	NT	+					
397	30	27	NT	+					

* + = Virus detected, — = Virus not detected, NT = Not tested.

One male and one female tick were released on each of the 14 chicks which were bled on the 22nd day after exposure; no attempt was made to isolate virus in the bloods of the chicks. Three out of seven chicks fed on by ticks of batch 69 and one out of seven fed on by ticks of batch 70 had HI antibodies to KFD virus (titres — 7, 3, 3 and 3 tubes).

DISCUSSION.

It is evident from these results that larvæ of *H. turturis*, *H. papuana kinneari* and *H. minuta* became infected by feeding on viremic chicks and in the nymphal stage transmitted the virus by bite to other susceptible chicks. *H. papuana kinneari* appeared to be less susceptible to KFD virus than the other two species. Although three out of four batches of *H. papuana kinneari* larvæ were fed on chicks with the same or even higher titres of circulating virus than those on which *H. turturis* and *H. minuta* larvæ were fed, trans-stadial transmission was observed in only 25 per cent of the nymphs tested individually, in 30 per cent of the tested tick pools, and in none of the adults. Moreover, only some of the infected nymphs transmitted the virus to the susceptible chicks.

It was observed that when the infected nymphs were still feeding on the chicks, the host chicks began to circulate virus in their blood, thus giving the nymphs a chance to ingest some more virus. Due to this a higher rate of infection was expected in the adults as compared to the nymphs. Contrary to expectations the rate of infection in adults was found to be lower in the case of *H. turturis* and *H. papuana kinneari* and more or less the same as in the nymphs in the case of *H. minuta*. At the present time there is no explanation for this behaviour of the virus in ticks.

SUMMARY.

Transmission of Kyasanur Forest disease virus by *H. turturis*, *H. papuana kinneari* and *H. minuta* was studied. All the three species became infected in the larval stage by feeding on viremic chicks and in the nymphal stage transmitted the virus by bite to other susceptible chicks.

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is prevalent should have a laboratory designated to carry out vir isolation, rapid detection of antigen, and serological procedures su as CF, HI, IFA, and ELISA tests.

Serological surveys of populations should be carried out in 4 g groups: 0–9, 10–19, 20–29, and 30 years.

Vector population. Vector surveillance is necessary for t following reasons:

(*a*) to obtain information on the distribution of vectors;
(*b*) to determine their ecology, their absolute and relative dens and their susceptibility to insecticides;
(*c*) to demonstrate the presence of virus in the vectors.

The virus is maintained in nature by transtadial and transovar transmission within the tick population and by horizont transmission between tick vectors and various domestic and w animals. Man is infected by the accidental bite of an infected ixod tick. *Hyalomma* spp. ticks remain the major vector of the vir Studies should be carried out on this vector particularly on p populations from areas from which the disease has been report earlier and also from areas where *Hyalomma* ticks are abundant virus isolations have been made from them.

3.1.9 *Prevention and control*

Control measures are mainly aimed at reducing the popula of tick vectors. This is particularly necessary when new areas of l are developed for agricultural, industrial, or housing schem Labour forces working in such areas are at a high risk of develop Crimean-Congo haemorrhagic fever. A suitable acaricide shoul selected and applied prior to the development of such tick-infe areas. HCH has been found to be a suitable acaricide, but other also available. Tick repellents are also of value and should be by workers involved in clearing forest and digging ground construction programmes.

Control of nosocomial spread. Once a clinical diagnosi Crimean-Congo haemorrhagic fever virus infection with blee manifestations has been made, the patient concerned should moved to the isolation area of the hospital. Transporting a bleed patient is hazardous and may spread the disease, causin nosocomial outbreak. The syringes, needles, and other materials used on an infected patient should be thoroughly disinfected by heat or chemicals after use. Special barrier nursing should be practised. The fatality rate is especially high among patients who have caught the infection through nosocomial spread. When an outbreak has been confirmed, a thorough clinical, virological, serological, and entomological survey should be undertaken to assess the extent of the epidemic area and the source of the index case.

Immunoprophylaxis. In Bulgaria, an inactivated mouse-brain vaccine has been used to immunize workers. However, no standardized vaccine for Crimean-Congo haemorrhagic fever is available; efforts should be made to develop such a vaccine for use among persons involved in the investigation of disease as well as health personnel and those occupationally exposed to the disease.

3.1.10 *Research needs and recommendations*

(1) Trials of specific immune plasma and/or antiviral drugs should be carried out in areas where Crimean-Congo haemorrhagic fever is common, when such areas are located.

(2) Modern molecular biology techniques should be used to develop a candidate vaccine for Crimean-Congo haemorrhagic fever.

(3) Research on tick biology, using modern biological techniques, should be encouraged.

3.2 Kyasanur Forest disease

3.2.1 *Historical background*

Early in 1957 there were reports of fatal epizootics in wild monkeys in forested areas of Shimoga district, Karnataka (formerly Mysore) State in India, associated with outbreaks of disease in villagers who lived in and around the forest fringe. Local inhabitants called the affliction "monkey disease" because of the known association with dead monkeys. The disease was later named after the locality—Kyasanur Forest—from where the virus was first isolated.

3.2.2 *Etiology*

Kyasanur Forest disease virus is a member of the *Flavivirus* genus of the Togaviridae family. It is antigenically related to other tick-borne flaviviruses, particularly the viruses causing Far Eastern tick-borne encephalitis, and Omsk haemorrhagic fever.

3.2.3 *Epidemiology*

The virus has a complex life-cycle involving a wide variety of tick species, particularly *Haemaphysalis spinigera* in its nymphal stage. Man is an incidental host and plays no part in virus transmission. Small mammals, particularly porcupines, squirrels, and rats are the main reservoirs of the virus. Birds and bats are less important hosts. The silent enzootic situation was perhaps dramatically altered by man's need for more land, both for grazing and other agricultural purposes. Cattle were put to graze around the forest and this provided *Haemaphysalis* ticks with a new and plentiful source of blood meals, which in turn resulted in a population explosion among the ticks. Cattle are very important in maintaining tick populations but play no part in virus maintenance. The ticks feed on other mammalian species such as monkeys, which show marked viraemia and an illness from which they may die. The monkeys are recognized as amplifying hosts for the virus. Seasonal epidemics of Kyasanur Forest disease have been associated with epizootics in monkeys, the most important being the black-faced langur (*Presbytis entellus*) and the South Indian bonnet macaque (*Macaca radiata*).

The disease is restricted to four districts (Shimoga, North Kanara, South Kanara, and Chikamagaloor) in Kurnataka State, India. The epidemic patterns indicate irregular and unpredictable spread of the virus since its recognition in 1957. Earlier, the disease was found to be limited mainly to an area around the original focus covering about 800 km². Newer foci have since been recognized covering over 6000 km². The latest outbreak during 1982 83 seems to have been the largest. A part of the Nidle forest of some 4 hectares was clear-felled by the forest department to make room for a cashew plantation. The labour force brought from the neighbouring areas and some villagers noticed a few dead monkeys in the forest by late October and the first cases of the disease in man were reported from December 1982 onwards.

3.2.4 *Clinical and clinicopathological diagnosis*

The incubation period is estimated to be between 3 and 8 days. The disease appears with a sudden onset of fever, headache, and severe myalgia, with prostration in some patients. The acute phase lasts about 2 weeks. Gastrointestinal disturbances and haemorrhages occur in severe cases. There is no abdominal pain except in patients in whom there is gastrointestinal bleeding. The liver is not palpable but the tip of the spleen may be felt, most often in patients with generalized lymphadenopathy. Bronchiolar involvement occurs in some patients and results in a persistent cough and abnormal physical signs in the lungs. In some patients (with blood-tinged sputum) serious signs of lung involvement have been recorded that were considered to be the precursors of pneumonia.

There is a diphasic disease course in a number of patients. The second phase is characterized by mild meningoencephalitis after an afebrile period of 7 21 days. It is manifested by a return of fever, severe headache followed by neck stiffness, mental disturbance, coarse tremors, giddiness, and abnormal reflexes.

Convalescence is generally prolonged lasting for up to 4 weeks. The case-fatality rate has been estimated to be around 5 10%.

The virus is extremely infectious and therefore is hazardous for laboratory workers; more than 100 persons are known to have suffered from laboratory-acquired infections. The clinical features are not significantly different from those described for natural infections except that the disease is generally milder and, to date, no case has proved fatal.

Limited haematological and biochemical investigations have been carried out, particularly during the early years. Leukopenia has been found to be an almost constant feature of the disease, mediated in all probability through antileukocyte antibodies. Thrombocytopenia of variable degree has also been found to be an important feature. Thromboagglutinins have been found in the circulation of the majority of patients. In contrast to the findings in haemorrhagic fevers from the USSR and the Korean peninsula, there is no evidence of gross capillary damage apart from those incidental to thrombocytopenia.

Albuminuria appears in most cases during the acute febrile stage. Granular casts have been occasionally observed. In most cases, the cerebrospinal fluid is clear with no increase in cells or alterations in

proteins, chlorides, or sugar. In patients developing the second ph
with meningeal signs, there is an increase in both cells and protei

The histopathological findings from 3 fatal cases in man show
similarities to the observations reported for other haemorrha
fevers. It was concluded that the histopathological changes, in bo
man and monkeys, appear to be mild in comparison with the sever
of the clinical illness. The haemorrhagic phenomena that were s
in some cases of the clinical disease were not adequately explain
by the histological appearance of the organs or blood vess
examined.

3.2.5 *Etiological diagnosis*

Unlike many other arboviruses, the Kyasanur Forest dis
virus has a prolonged viraemia of about 10 days or more. Of the
collected during the first 8 days of illness about 95% contained
virus. The serological response of man to infection with this v
was studied in 104 fever patients, all of whom had demonstra
viraemia; the patterns of serological response were not unlike th
seen with other flavivirus infections. The response depended up
whether the current infection was the first encounter with a flaviv
(primary) or whether there had been a previous infection with
or more flaviviruses (secondary). For secondary infections, spe
serodiagnosis was difficult. Both the neutralization test and the a
gel diffusion test are specific; the latter is also useful in the field.
agar gel diffusion test is less sensitive than the neutralization test.
primary infections, haemagglutination inhibition tests or sin
radial haemolysis tests seem to be satisfactory.

3.2.6 *Treatment*

There is no specific treatment for Kyasanur Forest dise
Supportive and symptomatic treatment should be provided, such
analgesics for myalgia and headache, intravenous fluids
dehydration, and blood transfusions if there is haemorrha
During convalescence, rest and an adequate diet seem to be
major requirements. It is not necessary to isolate the patients.

3.2.7 *Surveillance*

A surveillance system has been set up by the Karnataka s
government that records the number of cases and deaths suspe

of being due to Kyanasur Forest disease. Isolation of the virus from
sera taken during the acute phase is sometimes carried out, but only
on a small number of suspected cases.

The majority of human infections, with the exception of those
acquired in the laboratory are transmitted exclusively in and around
the forest. During forest clearing operations, rodents and monkeys
with their tick ectoparasites migrate to other areas, thus enlarging
the focus of natural infection. Attempts should be made to detect the
virus or antigen in ticks and a serological survey of rodents should
be carried out in virgin forest areas that are to be cleared.

3.2.8 *Prevention and control*

On forest tracks used by man, the number of ticks can be
controlled by spraying with acaricides. This, and the application of
effective repellents for the personal protection of the forest
labourers, may be implemented in restricted areas where monkey
deaths occur.

It is proposed that the population at risk should be immunized
with inactivated-killed Kyasanur Forest disease vaccine prepared
with chick embryo fibroblast.

3.3 Omsk haemorrhagic fever

3.3.1 *Historical background*

Omsk haemorrhagic fever is an acute febrile disease. Its
characteristic features are: viral etiology, natural focality,
haemorrhagic symptoms in patients, and a relatively benign course.
Cases of the disease were first reported in 1944–45, although there
is some evidence that similar cases appeared also in 1941–43, in rural
areas north of Omsk (Western Siberia). In 1945 and 1946 during two
outbreaks of Omsk haemorrhagic fever there were more than 200
and 600 cases, respectively, and in subsequent years, clinical,
epidemiological, and etiological features of the disease were studied
by a group of medical specialists from Omsk and Moscow. As a
result of these studies it was concluded that Omsk haemorrhagic
fever is a specific viral haemorrhagic disease different from other
known viral haemorrhagic fevers.

Following an infective tick bite, the incubation period is of the order of 7–12 days. The illness begins abruptly with fever, chills, malaise, irritability, headache, and severe pain in the limbs and loins followed by anorexia, nausea, vomiting, and abdominal pain. Fever is continuous but may be remittent and sometimes biphasic, resolving by crisis or lysis after 8 days. The face and neck are flushed and oedematous, the conjunctivae and pharynx are injected, and there is oedema of the soft palate. The mouth is dry and the breath has a foul odour. Patients are depressed and somnolent. In most cases a fine petechial rash begins on the trunk and then covers the entire body. The liver is enlarged in about 50% of cases but the respiratory system is unaffected. A haemorrhagic enanthema appears on the soft palate and uvula early in the illness and other bleeding manifestations, including haematemesis and melaena, appear on about the fourth or fifth day in over 75% of patients. Leukopenia and severe thrombocytopenia are common. Large purpuric areas caused by subcutaneous extravasation of blood occur at times. Bleeding occurs in descending order of frequency from the nose, gums, buccal mucosa, stomach, uterus, intestines, and lungs. Gastric and nasal haemorrhages often lead to death. Involvement of the central nervous system is seen in 10–25% of cases and usually indicates a poor prognosis; it includes neck rigidity, excitation, and coma. The mortality rate is often as high as 30–50%, usually due to shock, secondary blood loss, or intercurrent infection. This severe disease is in sharp contrast to the pattern of disease in Africa, where haemorrhagic phenomena and death are only rarely reported. A recent report [a] described an unusual outbreak of haemorrhagic fever in Rawalpindi District, Pakistan where the index case, a farmer, was admitted to hospital with haematemesis and melaena. A laparotomy was performed following which four members of the operating team became ill and two of them died. A virus similar to Crimean haemorrhagic fever virus was isolated.

KYASANUR FOREST DISEASE

This disease is caused by a flavivirus which, like Omsk haemorrhagic fever virus, is antigenically related to the tick-borne encephalitis complex but only rarely causes disease involving the central nervous system. The virus was first isolated in Mysore State, India, in 1957 and human infections, which still occur, are limited to villages surrounding Kyasanur Forest. The virus is now known to be widely distributed in India but human infections do not occur outside Mysore.

After an infectious tick bite, there is an incubation period of 3–7 days before the sudden onset of fever, frontal headache, severe myalgia, and prostration. This is quickly followed by nausea, vomiting, confusion, and restlessness. The conjunctivae are injected and the palate is suffused and often covered with maculopapular haemorrhagic spots. A generalized lymphadenopathy has been noted and many patients have bronchiolar involvement. The fever generally lasts 5–12 days and sometimes follows a biphasic course; a mild meningo-encephalitis occasionally occurs during the second phase. Epistaxis, haematemesis, haemoptysis, melaena, and bleeding gums are common and sometimes there may be uterine bleeding. Albuminuria, leukopenia, and thrombocytopenia are usual findings. A proportion of patients may die, usually 8–12 days after the onset of illness, developing coma or bronchopneumonia prior to death. The majority of patients, however, make an uneventful and complete recovery.

The virus is transmitted by *Haemaphysalis* ticks, especially *H. spinigera*, and is maintained in small mammals. In Mysore State, the silent enzootic situation was drastically altered by man's need for more grazing land. Cattle were put to graze around the forest and provided the *Haemaphysalis* tick with a new and plentiful source of blood meals, which produced a population explosion among the ticks. The abundant ticks fed on other mammalian species such as monkeys, and these became infected with Kyasanur Forest disease virus and developed marked viraemia and an illness from which they died. It was noted in 1957 that human infection was preceded by illness and death in forest-dwelling *Langur* and *Macacus* monkeys, which acted as amplifiers of the virus.

OMSK HAEMORRHAGIC FEVER

An epidemic of Omsk haemorrhagic fever occurred in Omsk and Novosibirsk Oblasts of Siberia between 1945 and 1948. The virus was transmitted by the tick *Dermacentor pictus* and by contact with infected muskrats *(Ondatra zibethica)*. Most of the more recent cases of disease in man appear to have been acquired through direct contact with muskrats. Most infections originate in the northern forest–steppe–lake belt of western Siberia, which contains much wet grassland and swamp.

Following an incubation period of 3–7 days, the illness begins abruptly with fever (which often follows a biphasic course), headache, vomiting, and diarrhoea. An enanthema of the palate, sometimes haemorrhagic, generalized lymphadenopathy, and meningism are common findings. Epistaxis, haematemesis, melaena, and uterine bleeding may occur, accompanied by a marked leukopenia, thrombocytopenia, and albuminuria. The central nervous system is rarely involved. The case fatality rate is low (0.5–3%). Convalescence may be prolonged but there are no sequelae.

The precise epidemiology of Omsk haemorrhagic fever is still unknown. There exists a biological cycle of unknown complexity, which may involve rodents and ticks. Muskrats, which were introduced into the region some 60 years ago for hunting purposes, are sometimes infected and are capable of transmitting the virus by direct contact.

JUNIN, LASSA, AND MACHUPO VIRUSES

All three of these viruses are members of the arenavirus taxon, a name derived from the inclusion-like dense particles seen by electron microscopy that give the virion an appearance of having been sprinkled with sand. The three viruses have rodents as their natural hosts and reservoirs in which they induce a persistent infection: the rodent suffers no ill effects and develops no immune response, although during its lifetime the animal continues to excrete virus, particularly in the urine. The rodents are presumably infected at birth.

[a] *Weekly epidemiological record*, 51: 301-308 (1976).

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dengue virus etiology. A significant and welcome feature of all these epidemics was the total absence of the "shock syndrome".

Studies on the distribution and habits of the mosquito vector of dengue, viz. *Aedes aegypti*, are being continued at the Virus Research Centre, Poona and the National Institute of Communicable Diseases, Delhi. A new finding made last year, i. e. the habit of the species to breed in tree holes, in South India, was confirmed by further studies. A study conducted at Bangalore yielded some pockets in the city where *A. aegypti* were found in large numbers and dengue viruses were isolated from them. This is a finding of considerable significance since this disease had not been reported in Bangalore.

Kyasanur Forest Disease (KFD)

This tick-borne virus disease first recognised in 1957, continued to pose a serious problem although still restricted to a small area in Shimoga District, Mysore State. The last two years witnessed the largest incidence of human cases so far reported. A collaborative venture of vaccination was undertaken by the Virus Research Centre, Poona, and the Department of Health, Government of Mysore. Killed KFD vaccine was produced at the VRC and was administered to a section of the population, at risk, for a field trial. Efforts such as these and experiments for controlling ticks have been intensified but serious attention is still being devoted to the more basic problem, viz. that of understanding the dynamics of the natural cycle. The role of certain species of bats which have been found to have antibodies to this virus, not only in the KFD epidemic area but also well outside the limits, is being studied.

Japanese Encephalitis (JE) & West Nile (WN) Virus Infection

Unlike the dengue viruses, JE virus infects several extrahuman hosts and thus has a complex natural cycle. Pigs are now known to be good indicators of the prevalance of the virus in any given locality. As the vector mosquito, *Culex tritaeniorhynchus*, has a predilection to breed in paddy fields, it is more than likely that the changing ecological patterns brought about by the large irrigation projects undertaken in our country will affect the distribution of this mosquito, and hence of the virus that it carries. Intensive comparative studies have, therefore, been started in irrigated and non-irrigated areas in Andhra Pradesh with particular reference to the Nagarjunasagar Project.

As revealed by serological surveys, West Nile virus is perhaps the most prevalent among the arboviruses in this country. The ICMR Virus Unit at N.I.C.D., Delhi has been entrusted with the work of determining the nature of illness it produces in man.

Sandfly Fever

Sandfly fever was hitherto well known to occur in the arid regions of West Pakistan and Middle East countries. Its occurrence in India was thought to be doubtful. However, the Sandfly fever virus was isolated in India in 1967 on two occasions from febrile cases at Aurangabad (Maharashtra). The follow up studies have resulted in several more isolations of this virus both from sandflies and from sera of febrile patients indicating activity of the virus in this area. As a result of this significant finding, epidemiological studies have been initiated at these areas.

ENTEROVIRUSES

Work on entero-viruses is being mainly conducted at the Enterovirus Research Unit at Bombay; Pasteur Institute, Coonoor; the Enteroviruses Unit at the Christian Medical College, Vellore and the Virus Unit at the K. G. Medical College, Lucknow.

At the Pasteur Institute, Coonoor, large batches of bulk monovalent oral vaccine (Sabin) for the three types of polio virus have been prepared and checked for the absence of SV_{40} virus. These are being tested for neurovirulence at the National Control Laboratory of the National Institute of Communicable Diseases, Delhi, before release for public use.

Vigilance for polio cases in Bombay city has been maintained and no change was noticed in the epidemiological patterns of the disease. A collaborative project has been undertaken with the Directorate of Public Health and B. J. Medical College, Poona, to study four rural areas in Poona District to determine (i) the entero-virus pattern in the child population; (ii) the extent of natural immunity against polio-virus in the local community, and (iii) the efficacy of the oral polio vaccine in this rural community.

During 1969-70, 84 cases of paralytic illness were detected among the children vaccinated against polio in Greater Bombay. From 52 cases studied, ten strains of polio type 1 and seven strains of polio type 2 were isolated. This has highlighted the need to review the lack of efficacy of the vaccine which may be due to loss of potency at the time of administration or interference by other wild enteroviruses prevalent in the community. Preliminary results of a study have

Park & Park : Text book of Preventive Social med. Ed. 12.

Control of JE

(a) VECTOR CONTROL : The vector mosquito (s) of JE are widely scattered and not easily amenable to control. An effective way to deal with them is to resort to aerial or ground fogging with ultra-low-volume (ULV) insecticides (e.g.,malathion, fenitrothion). All the villages reporting cases should be brought under indoor residual spray. The spraying should cover the vegetation around the houses, breeding sites and animal shelters in the affected villages. Uninfected villages falling within 2 to 3 km radius of the infected villages should also receive spraying as a preventive measure. Villages within the proximity of infected villages should be kept under surveillance. The use of mosquito nets should be advocated.

(b) VACCINATION : Vaccination of population at risk has been recommended.A killed "mouse -brain" vaccine is available. For primary immunization, 2 doses of 1 ml each (0.5 ml for children under the age of 3 years) should be administered subcutaneously at an interval of 7-14 days. A booster injection of 1 ml should be given after a few months (before one year) in order to develop full protection. Protective immunity develops in about a month's time after the second dose. Revaccinations may be given after 3 years. The vaccine is best used in the inter-epidemic period. It should be offerec to the most vulnerable and high-risk groups *(8, 9)*.

3. Kyasanur Forest Disease

Kyasanur Forest Disease (KFD) is a febrile disease associated with haemorrhages caused by an arbovirus flavirirus and transmitted to man by bite of infective ticks.

History

KFD was first recognized in 1957 in Shimoga district of Karnataka State in South India. Local inhabitants called the disease "monkey disease" because of its association with dead monkeys. The disease was later named after the locality — Kyasanur Forest — from where the virus was first isolated.

Problem statement

Earlier the disease was found to be limited mainly to an area around the original focus (Shimoga district) covering about 800 sq. km. Newer foci have since been recognized. The disease is now restricted to four districts (Shimoga, North Kanara, South Kanara and Chikamagaloor) in Karnataka State in India covering over 6000 sq. km *(10)*. Serological surveys in different parts of India revealed antibodies to KFD or a closely related virus in humans and animals, particularly in cattle in Kutch and Saurashtra *(11)*.

According to recent reports, the disease continues to be active in its endemic foci. The latest outbreak during 1983-1984 seems to be the largest with 2167 cases and 69 deaths, as against 571 cases and 15 deaths during 81. The Karnataka Government has established a surveillance system which monitors the occurrence of KFD in humans and mortality in monkeys in known epidemic, as well as neighbouring areas. Deaths of monkeys are considered as heralders of this disease in endemic areas.

Epidemiological features

(a) Agent : The agent KFD virus is a member of group B togaviruses (flaviviruses). It is antigenically related to other tick-borne flaviviruses, particularly the Far Eastern tick-borne encephalitis and Omsk haemorrhagic fever. Unlike in many other arbovirus infections, KFD has a prolonged viraemia in man for about 10 days or more.

(b) Natural hosts and reservoirs

Small mammals particularly rats and squirrels are the main reservoirs of the virus *(10)*. Birds and bats are less important hosts. The monkeys are recognized as *amplifing* hosts for the virus. However, they are not effective maintenance hosts because most of [illegible] FD infection. Cattle provide *Haemaphysalis* ticks with a plentiful source of blood meals, which in turn leads to a pop[illegible] explosion among the ticks. Thus cattle are very impor[illegible] maintaining tick populations but play no part in virus main[illegible] *(10)*. Man is an incidental or dead-end host and plays no part [illegible] transmission.

(c) Vectors

The virus has a complex life cycle involving a wide variet[illegible] species. At least 15 species of hard ticks of the genus *Haema*[illegible] particularly *H. spinigera* and *H. turtura* are known to tran[illegible] disease. KFD has also been isolated from soft ticks *(12)*. The[illegible] number of human and monkey infections occur during drie[illegible] particularly from January to June. This period coincides with[illegible] nymphal activity of ticks.

(d) Host factors

(i) *Age :* Majority of cases affected were between 20 and [illegible] (ii) *Sex:* Attack rate was greater in males than in fem[illegible] *Occupation*. The attacked people were mostly cultivators wh[illegible] forests accompanying their cattle or cutting wood (iv) *Human* [illegible] The epidemic period correlates well with the period of greates[illegible] activity in the forest, i.e., from January until the onset of rains[illegible]

(e) Mode of transmission

The transmission cycle involves mainly monkeys and [illegible] disease is transmitted by bite of infective ticks, especially [illegible] stages. There is no evidence of man to man transmission.

(f) Incubation period

Estimated to be between 3 and 8 days

Clinical features

The disease appears with a sudden onset of fever, hea[illegible] severe myalgia, with prostration in some patients.The act[illegible] lasts about 2 weeks. Gastrointestinal disturbances and haem[illegible] from nose, gums, stomach and intestine may occur in seve[illegible]

In a number of cases, there is a second phase characteris[illegible] meningoencephalitis after an afebrile period of 7 to 21 [illegible] manifested by a return of fever, severe headache followed [illegible] stiffness, coarse tremors, abnormal reflexes and mental distu[illegible] The case fatality rate has been estimated to be 5 to 10 per [illegible]

Diagnosis is established only after detecting the presenc[illegible] virus in the blood and/or serological evidence.

Control

(a) Control of ticks : Since KFD is a tick-borne disease, [illegible] ticks should be undertaken. For control of ticks in forests, sp[illegible] can be made by power equipment or by aircraft-mounted equip[illegible] dispense carbaryl, fenthion, naled or propoxur at 2.24 kg [illegible] ingredient per hectare *(13)*. The spraying must be carried ou[illegible] spots", i.e., in areas where monkey deaths have been report[illegible] 50 m around the spot of the monkey deaths, besides the ende[illegible] Since the heavy tick population in the forest areas is attribu[illegible] to the free roaming cattle, restriction of cattle movement is th[illegible] bring about a reduction in vector population *(5)*. *(b) Vaccina*[illegible] population at risk should be immunized with killed KFD [illegible] *(c) Personal protection :* Protection of individuals exposed to [illegible] of infection by adequate clothing and insect repellents [illegible] dimethylphthalate (DMP, DEET) should be encouraged. The[illegible] examine their bodies at the end of each day for ticks and rem[illegible] promptly. The habit of sitting or lying down on the ground [illegible] discouraged through health education.

4. Chikungunya Fever

A dengue-like disease caused by a group A virus, the ch[illegible] virus and transmitted by *Aedes, Culex* and *Mansonia* mosq[illegible] manifested by high fever and severe articular pains in the [illegible] spinal column *(14)*. The virus was first isolated from p[illegible]

[illegible] during an epidemic in Tanzania in 1952-53. Chikungunya [illegible] word meaning "doubling up" owing to excruciating joint [illegible] there was an outbreak of this disease in Calcutta in 1963-64 [illegible] ther in Madras in 1965, which gave rise to 3,00,000 cases in [illegible] city alone *(15)*. According to reports, the virus has not been [illegible] since 1965 *(16)*.

[illegible] TROL : (a) *Vector control:* The *Aedes aegypti* mosquito should [illegible] main target of control activities. It requires active community [illegible] effort to keep water storage containers free of mosquitoes and [illegible] eliminate the other breeding places of mosquitoes in and around [illegible] and dwellings *(17)*. The organophosphorus insecticide, *Abate* [illegible] is largely being used as a larvicide. It can prevent breeding for up [illegible] months when applied on sand granules; does not harm man and [illegible] does not affect the taste of water. Antilarval measures can prevent an [illegible] epidemic, but do not give immediate results when an epidemic has [illegible] already broken out. In such cases, antiadult measures alone can bring [illegible] about rapid interruption of transmission. A new technique [illegible] consisting of aerosol spray of ultra low-volume (ULV) quantities of [illegible] malathion or sumithion (250 ml/hectare) has been found to be [illegible] effective in interrupting transmission and stopping epidemics of DHF. [illegible] The droplets kill the mosquitoes in the air as well as on water. By [illegible] giving 2 ULV treatments at about 10 days apart, the Aedes Research [illegible] Unit in Bangkok was able to reduce adult mosquito densities by more [illegible] than 90 per cent for several weeks *(18, 19)*. (b) *Vaccine*. No vaccine has [illegible] been developed that is considered suitable for use.

[illegible] Fever

[illegible] a febrile illness caused by a group B arbovirus. The disease [illegible] occurs in Africa, the Middle East, South-West Asia and India, and [illegible] is spread by certain species of Culex mosquitoes. Clinically, it is [illegible] characterized by a sudden onset of fever, severe headache and malaise [illegible] for several days. In children, a maculopapular rash of short [illegible] duration may appear. In the aged, a fatal meningo-encephalitis may be [illegible] observed.

[illegible] Fever

[illegible] fever is known to occur in the arid regions of West Pakistan [illegible] and East. Its occurrence in India was thought to be doubtful. [illegible] In 1967, the Sandfly fever virus was isolated in Aurangabad [illegible] (Maharashtra) from febrile cases. The virus was also isolated from [illegible] cases *(20)*. The control of sandfly fever is based on the control of [illegible] vector.

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BRUCELLOSIS

Brucellosis is one of the major bacterial zoonoses, and in humans is also known as Undulent fever, Malta fever or Mediterranean fever. It is occasionally transmitted to man by direct or indirect contact with infected animals. It is caused by different species of the brucella group of organisms and characterised by intermittent or irregular febrile attacks, with profuse sweating, arthritis and an enlarged spleen. The disease may last for several days, months or occasionally years. Brucellosis is both a severe human disease and a disease of animals with serious economic consequences.

Probelm statement

Brucellosis is a recognized public health problem with world-wide distribution. It is endemic where ever cattle, pigs, goats and sheep are raised in large numbers. Important endemic areas for brucellosis exist in Mediterranean zones, Europe, Central Asia, Mexico and South America.

Animal brucellosis is reported from practically every State in India. However, no statistical information is available about the extent of infection in man in various parts of the country *(1)*.

The prevalence of human brucellosis is difficult to estimate. Many cases remain undiagnosed either because they are inapparent or because physicians in many countries are unfamiliar with the disease.

Agent factors

(a) *Agent :* The agents are small, Gram negative rod-shaped, non-motile, non-sporing and intracellular coccobacilli of the genus *Brucella*. Four species infect man. *B. melitensis, B. abortus, B. suis*, and *B. canis*. *B. melitensis* is the most virulent and invasive species; it usually infects goats and occasionally sheep. *B. abortus* is less virulent and is primarily a disease of cattle. *B. suis* is of intermediate virulence and chiefly infects pigs. *B. canis* is a parasite of dogs. (b) *Reservoir of infection :* Main reservoirs of human infection are cattle, sheep, goats, swine, buffaloes, horses and dogs. In animals the disease can cause abortion, premature expulsion of the foetus or death. Cross infections can often occur between animal species. The infected animals excrete *Brucella* in the urine, milk, placenta, uterine and vaginal discharges particularly during a birth or abortion. The animals may remain infected for life.

Host factors

Human brucellosis is predominantly a disease of adult males. Farmers, shepherds, butchers, and abattoir workers, veterinarians and laboratory workers are particularly at special risk because of occupational exposure. Immunity follows infection.

Environmental factors

Brucellosis is most prevalent under conditions of advanced domestication of animals in the absence of correspondingly advanced standards of hygiene. Overcrowding of herds, high rainfall, lack of exposure to sunlight, unhygienic practices in milk and meat production all favour the spread of brucellosis. The infection can travel long distances in milk and dust. The environment of a cow shed may be heavily infected. The organism can survive for weeks, or months in favourable conditions of water, urine, faeces, damp soil and manure.

Mode of transmission

Transmission is usually from infected animals to man. There is no evidence of transmission from man to man *(2)*. The routes of spread are :

(a) *Contact infection :* Most commonly, infection occurs by direct contact with infected tissues, blood, urine, vaginal discharge, aborted

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DIABETES FORUM

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Inside

The Diabetic Foot

In Greek mythology, there was once a great warrior called Achilles. When he was a small baby, the Gods told his mother to dip him in river Styx as this would make him completely safe from any harm or injury. The mother held him up by his heels and dipped him completely in the river. As Achilles grew up he became well known as a warrior and as nothing could hurt him, everybody feared fighting him. Until one day, another famous warrior called Paris shot a poisoned arrow which hit Achilles in his heel. This was the part of the body that had been held in the mother's hand when she had dipped him in the river and therefore was not safe from harm. The poisoned arrow could kill Achilles. From that day onwards, any vulnerable aspect of a person, has been called as the "Achilles heel" of that person. This could well be applied to the feet of any diabetic. We tend to pay so much attention to the long term complications of diabetes like eye, kidney and nerve problems, that we overlook the importance of foot care in diabetes.

The importance of preventing foot problems in a diabetic should NEVER be underestimated. If one were to see the number of indoor patients in any specialised diabetic clinic or hospital, one would find more than half are there because of some foot problem. These are also patients who need to stay in the hospital the longest. Whilst with excellent management, we are able to save many a foot, quite a number of the patients still need to undergo an amputation. The majority of foot problems occur in diabetics with insensitive feet, possibly without adequate circulation and are PRECIPITATED by infection, injury, or both. Due to the fact that there are usually definite precipitating factors, a vast number of the foot problems are preventable. Even if they do occur, it is possible to "catch" them at the earliest so that the management is simpler and the results much better. The tragedy is that neither adequate attention nor importance is given to this.

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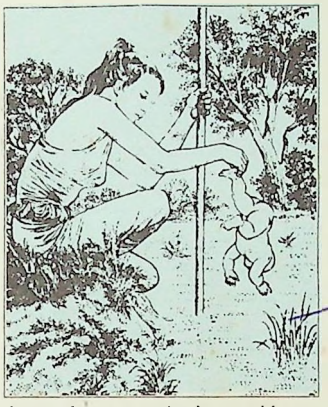


To quote a passage from the ancient chronicles, "In the thirty ninth year of his reign Asa was diseased in his feet, and his disease became severe; he sought help from his physicians but died in the forty first year of his reign." (II Chronicles XVI, 12-14). Some authorities regard this quotation as one of the earliest references to diabetic foot disease and many of the more sceptical ones feel that there is not much more that we can do today about diabetic foot problems compared to what was done for Asa. This attitude is unfortunate as foot problems in diabetes are preventable provided a few basic instructions are followed and proper care taken. Before the "do's and don'ts" about foot care can be discussed, it would be worthwhile to consider briefly some of the mechanisms which predispose the diabetic to foot problems. This would enable giving instructions to the patient in a much more rational manner rather than just giving him some "commandments" to observe.

Neuropathy

Nerve involvement due to diabetes is one of the commonest long-term complications of diabetes. Involvement of the sensory nerves going to the feet brings about many varied symptoms and signs but many patients have a marked reduction in the pain sensation and a significant number of patients go on to have insensitive feet, and are incapable of feeling any type of sensation.

Thus, he is incapable of feeling pain or other uncomfortable sensations. The patient may not be aware of any injury or infection until it has progressed to a severe stage, or are pointed out by a relative or the doctor This makes it difficult to detect injury or infection at an early stage when management would be simpler. Although diabetic neuropathy more commonly affects the sensory nerves, the motor nerves may also be involved. The motor nerves which innervate the small muscles of the feet help in maintaining the shape and the "arches" of the foot. When these nerves are affected, there is a wasting of the small muscles of the feet and this may change the configuration of the foot. The toes may become 'cocked' up and the area of the sole near the heads of metatarsals comes to bear most of the weight of the body. Areas which are not normally pressure bearing may have to bear the brunt of the body weight. This change in the normal architecture of the foot accompanied by a decrease in the sensitivity is one of the most important predisposing factors in diabetic foot disease.

One aspect of neuropathy that is rarely given its due is the autonomic nerve involvement. These are nerves that have many important functions in the body and of these many functions, the ones that are especially relevant to our discussion, are that they regulate the blood supply to the limbs, determine sweating and also maintain the normal texture of the skin. When these nerves are affected in diabetes, it causes a reduction or even a complete absence of sweating in the feet and the lower legs. A reduction in sweating causes the outer layers of the skin to become dry and this makes the skin of the feet, brittle and liable to develop cracks which may form entry points for infecting bacteria. The skin also loses its ability to stretch and therefore any change in the shape of the feet also tends to cause the development of breaks and cracks in the skin. This ab-

sence of sweating is extremely important as in one major study it was reported that diabetic ulcers only occurred in those feet that had a significant reduction in or a complete absence of sweating.

The autonomic nerves are also responsible for regulating the blood supply to the feet and this supply is affected when the nerves are involved. Surprisingly, feet that are affected with autonomic neuropathy appear warm with good peripheral pulsations and have been shown to have an increased blood supply. This increase in the warmth leads many to mistakenly feel that the circulation in the limb is adequate. This is definitely not the true situation. It is known that although the total blood going to the leg and feet may have increased,most of this blood is shunted directly from the small arteries to the veins, bypassing the capillaries. It should be remembered that it is at the capillary level that the real function of the blood circulation takes place. Therefore, although the total quantity of blood flow to the feet may appear to increase in diabetic neuropathy, this is of no real use and one could say that in practical terms there is a lack of blood supply to the feet.

Peripheral vascular diseases

The inadequate blood supply to the limb may be further compromised by the presence of peripheral vascular disease, which is much more common amongst diabetics than in non-diabetics. In this condition, the arteries supplying blood to the legs and the feet are narrowed down by atheroma formation. The earliest symptom of this could be pain in the legs whilst walking. Some patients get pain at night when they are lying down but this can be relieved by hanging the foot over the edge of the bed and is increased if the patient gets up and walks around. The feet may feel cold, skin appears dry and parched, the nails lose their lustre and the small amount of hair on the toes may be lost. The feet of a diabetic with autonomic neuropathy appear warm whilst with peripheral vascular disease, the skin is cold. This may sound confusing but it should be realised that diabetics do not have a clear-cut demarcation between those that have only a neuropathy and those that have only peripheral vascular problems. Most of them have varying degrees of both, and the clinical picture would depend upon the relative severity of the two conditions in any individual patient. In any case, neuropathy and vascular disease, in severe forms, presenting in the same patient is dangerous because the patient becomes prone to painless ulcers which are resistant to treatment.

Infection and trauma

It is apparent that neuropathic changes and peripheral vascular disease predispose the patient to serious foot complications. The majority of these foot problems need to be precipitated, or triggered by infection, trauma or both. Therefore, the focus in preventing serious foot complications would aim at efforts to avoid trauma and infection to the feet or in the least, diagnose their presence in the very early stages so that adequate measures can be taken at a time when management of the patient would be somewhat easier.

What are the ways and means to avoid trauma and infection in a foot already prone to complications?

If one reflects on the changes brought about by the neuropathy and the peripheral vascular disease, and understand that the aim is to avoid infections and trauma to the feet, then the significance about foot care is easily understood.

Inspecting the feet daily

This is of utmost importance in order to catch any problem at the earliest stage. But the patient must know what he should be inspecting! He should search for any breaks in the skin, scars and burn marks and any redness that may be a sign of infection, any puncture or injury marks, any darkening of the skin, presence of corns and callus formation. Two areas where he should look very carefully are between the toes and at pressure points at the bottom of the feet.

Some patients, especially those that are obese or have joint pains may find it difficult to lift up the feet for a close inspection. They can very easily use a mirror to examine the bottom of the feet. Failing this, a relative would have to be instructed in the means of foot care.

Patients with high risk to have foot problems

1. Peripheral neuropathy
2. Peripheral vascular disease
3. Severe retinopathy or nephropathy
4. Previous foot ulcers
5. A foot deformity for whatever reason
6. Blind or partially sighted patients, and those living alone

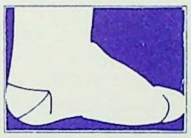
Foot Care for Diabetics

Wash feet daily with lukewarm water and soap, just as washing hands

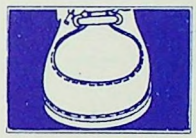
Dry feet well, also between the toes

Cut nails straight across Ingrown nails and calluses should receive expert attention

Keep the skin supple with a moisturizing lotion, but do not apply it between the toes



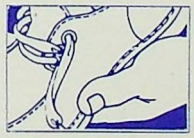
Change daily into clean, soft socks or stockings which must neither be too big nor too small



Keep your feet warm and dry Preferably wear cotton socks or stockings and leather shoes

Never walk barefoot-neither indoors nor outdoors

Always wear shoes that fit This applies also to sandals



Examine shoes every day for cracks, pebbles, nails and other irregularities which may irritate the skin

Washing the feet daily

This may not seem a common problem in our country with the habit of having a daily bath, but some diabetics are under the mistaken notion that diabetic feet should be kept scrupulously dry at all times so that even when they have a bath, they cover their feet with a plastic bag.

In addition to maintaining cleanliness (so important to avoid infection), washing the feet daily helps in the skin regaining some moisture that may be lacking because of the neuropathy. The water that is used to wash the feet should be tepid, neither very hot nor

(Contd. on pg. 9)

Diabetes among Indians

The prevalence of Diabetes Mellitus varies in different geographic regions and in different ethnic groups. Although many studies have been carried out in the past (Table 1) to look for the prevalence of diabetes in india, these have used varying criteria for the diagnosis of Diabetes Mellitus, and therefore the validity is open to question. Now that the W.H.O. has laid down standardised criteria, these will be used by all investigators and it should soon be possible to compare data from various regions and ethnic groups.

Epidemiological studies carried out in different parts of the world brought out an interesting finding that Indian migrants who were settled abroad had high prevalence of diabetes. These studies, indicate that Indians as an ethnic group have a high risk of developing diabetes and the susceptibility may be genetic in nature.

A survey conducted by the Diabetes Research Centre in Madras, two populations belonging to the same ethnic group but of different socio-economic status, living in urban and rural areas showed different rates of prevalence of diabetes (Table 2).

The age-adjusted prevalence of diabetes in the urban population was 8.2% and 2.4% in the rural population. This study showed the wide difference in the prevalence of diabetes in urban and rural India. The prevalence was 8.4% in urban men and 7.9% in urban women compared to 2.6% and 1.6% in rural men and women respectively. Age, body mass index (BMI) and the waist to hip ratios (WHR) showed positive association with diabetes in both the populations. The mean value of BMI was considerably lower especially in the rural population. Nevertheless, upper body adiposity and BMI were found to be positive risk factors in this relatively non-obese population.

A study from Orissa state and a multicentric study by the Indian Council of Medical Research (ICMR) have also shown that the prevalence of diabetes is higher in urban areas compared to that in rural areas.

Prevalence of undetected diabetes

In a developing country like India, illiteracy and a lack of awareness about diabetes are the major factors that determine the vast differences in the known to undetected diabetes between the urban and the rural areas. In our studies, we have recently reported a new to known ratio of 1:2 in an urban area and 3:1 in a rural population around Madras city. These observations are in agreement with data reported in several epidemiological studies of diabetes in urban: rural migrant Indians.

A. Ramachandran, *Deputy Director Diabetes Research Centre and M.V. Hospital for Diabetes, Madras*

Table 1

Epidemiological studies of the prevalence of Diabetes Mellitus in India

Year	Author	Place	Prevalence (%) Urban	Rural
1971	Tripathy *et al*	Cuttack	1.2	—
1972	Ahuja *et al*	New Delhi	2.3	—
1979	Johnson *et al*	Madurai	0.5	—
1979	Gupta *et al*	Multicentre	3.0	1.3
1984	Murthy *et al*	Tenali	4.7	—
1986	Patel	Bhadran	—	3.8
1988	Ramachandran *et al*	Kudremukh	5.0	—
1989	Kodali *et al*	Gangavathi	—	2.2
1989	Rao *et al*	Eluru	—	1.6
1992	Ramachandran *et al*	Madras	8.2	2.4

Impaired Glucose Tolerance (IGT)

Impaired Glucose Tolerance was classified as an entity different from diabetes because long term follow-up studies have shown that a large proportion of the subjects with IGT may remain as such or revert to normal tolerance and the prevalence of microvascular complications such as retinopathy is negligibly small. The ratio of the prevalence of IGT/diabetes varies in different populations and is usually around one.

An important observation made in the Madras Diabetes Survey was that the prevalence of IGT was almost similar in urban and rural populations (8.7% vs. 7.8%) despite a four-fold lower prevalence of diabetes in the latter (Table 2). This observation assumes significance in view of our earlier observation that 35% of the IGT do carry a high risk of progressing into overt diabetes. With increasing urbanisation there would be higher conversion rate from IGT to diabetes and this should result in an increase in the prevalence of diabetes in India in the near future.

Evidence from epidemiological studies described so far clearly point out that Indians as an ethnic group have a very high risk of developing diabetes. With increasing urbanisation and increased life expectancy combined with a rapid growth in population, a phenomenal increase like an epidemic of diabetes in India has been foreseen by many epidemiologists. In 1990, it was estimated that there were 15 million diabetics in India. This figure, with increasing incidence, is estimated to rise to a phenomenal 35 million by the year 2000! This may pose a major public health problem and impose a severe burden on the healthcare system in our country. This calls for appropriate and effective preventive measures to be adopted to curb or atleast delay the onset of diabetes.

Risk factors for NIDDM in Indian population

Diet

With recent urbanisation and modernisation of living conditions, there has been a transition from consumption of natural food rich in fibre content to more of refined food containing very little fibre. Moreover, lower utilisation of calories by lack of physical activity leads to unfavourable adiposity. The usefulness of a calorie-restricted fibre-rich diet in the management of diabetes has been unequivocally proved by the extensive studies of Professor M. Viswanathan and co-workers. This diet is similar to the diet of a common man in India and has high fibre content available from natural food sources.

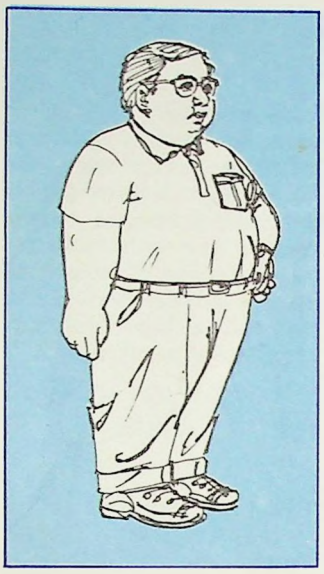
Obesity

The relationship between obesity and diabetes is complex and confounded by many heterogenous factors. It is generally agreed upon that obesity definitely contributes to the unmasking of the disease in a genetically prone individual. Association between upper body adiposity and greater risk of diabetes has been shown in several ethnic populations. It is interesting to note that at Diabetes Research Centre, Madras, we have observed a similar as-

Table 2

Age-adjusted prevalence of diabetes and IGT urban & rural populations

	No.	Prevalence % Diabetes	IGT
Urban Total	**900**	**8.2**	**8.7**
Men	457	8.4	8.8
Women	443	7.9	8.3
Rural Total	**1038**	**2.4**	**7.8**
Men	520	2.6	8.7
Women	518	1.6	6.4



sociation between diabetes and WHR even in the relatively non-obese South Indian population who had a significantly lower degree of obesity in comparison with their western counterparts.

In urban South Indians, the body mass index has been shown to be a strong predictive factor of diabetes in women, but only of marginal predictive nature in men.

Age, sex and parity

In an urban-rural survey in Madras, we found that the prevalence of diabetes was 21% in the group above 40 years and 41% in the age group of 55-64 years. Majority of the epidemiological surveys show a male preponderance among Indian diabetics. Parity has no independent effect on glucose tolerance in Indian women unless it is associated with obesity.

Hyperinsulinemia and Insulin Resistance

Hyperinsulinemia (a higher level of circulating insulin in the blood) and insulin resistance may be present many years before the onset of diabetes and clearly play an important role in its etiology. These two factors may also play an important causal role in hypertension, dyslipidemia and upper body adiposity and may eventually lead to an increased prevalence of coronary artery disease.

Genetic component in the etiology of NIDDM

Epidemiological evidences for a strong genetic component in the etiology of NIDDM are many and come from different sources.

Familial aggregation of diabetes

Asian Indians are shown to have increased familial aggregation of diabetes with higher prevalence of diabetes among the first degree relatives and vertical transmission through two or more generations. It was found that 45% of the Indians compared to 38% of the Europeans had positive family history of diabetes.

A recent analysis of family history in the NIDDM patients attending the Diabetes Research Centre, Madras showed that positive family history was present in 62% and 53% had first degree relatives with diabetes. It was also noticed by Viswanathan et al that the prevalence of diabetes increased with increasing family history of diabetes. They noted that the prevalence of diabetes among offspring with one diabetic parent was 36% which increased to 54% with positive family history of diabetes on the non-diabetic parental side also. The cumulative risk of developing diabetes in the offspring by the age of 70 years was 41%-64% in these two groups. The prevalence rate (62%) and the risk (73%) increase further when both parents have diabetes. The offspring of diabetic parents are reported to develop the disease at least a decade earlier than their parents.

Genetic susceptibility in ethnic groups

Certain ethnic populations show a high prevalence of NIDDM. It is interesting to note, Indians in Fiji and Singapore are shown to have higher prevalence of NIDDM compared to the host population; This disparity in the prevalence of diabetes in different ethnic groups points out their greater genetic susceptibility for NIDDM which gets unmasked by environmental influences.

Autosomal dominant inheritance of diabetes

Autosomal dominant inheritance of NIDDM with vertical transmission of the disease through at least three generations is quite frequent in Indians, Among the South Indians, we and others have reported younger age at onset of the disease compared to the western population. This phenomenon and the prevalence of NIDDM probably suggest the strong influence of genetic loading in unmasking diabetes early in life.

A great deal of interest is focussed on the identification of the genetic markers of NIDDM. Collaborative study from our centre and Dr G.A. Hitman in London has shown no association of NIDDM with the amylin gene, in the South Indians (Dravidians).

Conclusion

To summarise, it may be said that the probable causal factors in diabetes in Asian Indians include ethnic susceptibility, genetic factors, migration from rural to urban areas with a concomitant change in life style, socio-cultural factors, diet, physical activity, obesity, stress, age, sex, parity and presence of insulin resistance.

There is little doubt that Asian Indians have a genetic susceptibility to develop diabetes which appears to become exposed when they migrate and achieve improved socio-economic status. Present evidence suggests that insulin resistance may be a common feature in Indians leading to the high prevalence of diabetes and other metabolic disturbances that are possibly responsible for the high rates of coronary heart disease.

At the current rate of growth it is estimated that by the turn of the century, there will be about 35 million diabetics in India. This stresses the need for adopting intensive preventive measures to curb or atleast delay the onset of this disease.

Glycaemic control: Can it be achieved?

Advances in the realm of diabetes management have indeed been rewarding. Increasing numbers of patients have lived for longer years following diagnosis, thanks to the changed outlook about diabetes care.

The two major objectives of diabetes management are to restore the deranged metabolism to as 'normal' a state as possible and to enable the patient to live a life as close to 'normal' as possible. While the life of a diabetic may not be strictly normal, it can be made livable and enjoyable. It is true that for some diabetics, maintaining a normal blood glucose level at all times may be difficult. This however, does not mean that one should give up the goal of glycaemic control. It would be futile to set normal glucose levels as a goal of treatment if it were impossible to achieve.

The main treatment modalities in diabetes are patient education, dietetic adjustments, oral hypoglycaemic agents (OHA s) and insulin-no more, no less. There are no other therapeutic approaches either. The control of diabetes largely depends on the triad of diet, physical activity and insulin or OHA s which are interdependent and must be balanced properly. Not to forget the 'intangibles' - stress, timing, food absorption, etc., which can also influence the diabetic control.

The diabetic patient's involvement is crucial for achieving good control and hence education about diabetes and its treatment should constitute a vital part of the health-care plan. It is heartening that there is evidence linking good control with a favourable effect in reducing late diabetic complications such as diabetic retinopathy, nephropathy and neuropathy. Studies in animals and humans have also shown that good diabetic control may ameliorate and possibly help prevent chronic complications of diabetes. It is therefore, that glycaemic control has assumed further significance.

The attainment of optimal glycaemic control is essentially dependent on the ability to monitor the degree of control achieved. Presently, diabetic control can be assessed rather effectively with the help of laboratory parameters such as testing of urine for sugar and ketones, measurement of plasma or blood glucose concentrations and glycosylated haemoglobin or albumin levels. Effective monitoring is the key to achieving optimal control of blood glucose levels and successful management of diabetes.

The development of newer treatment regimens including highly purified insulin and human insulin, and increasing awareness about them, together with effective monitoring and patient education have made optimal glycaemic control a reality. This means that satisfactory diabetic control is an achievable proposition in many patients.

Emergency Surgery in Diabetes

It has been estimated that one half of all diabetic patients require surgery. They are at a slightly inreased overall risk of operative mortality (3.4%) versus their non-diabetic counterparts (2.5%). Of all those cases, about 5% are performed on an emergency basis. Surgeons often encounter either metabolic catastrophe such as profound hypoglycaemia, ketoacidosis (DKA) or hyperosmolar hyperglycaemic nonketotic dehydration (NHND) and coma accompanying an acute surgical condition. This group carries significant risk. Some are diagnosed to be having diabetes for the first time during the operative process. They are at high risk as well.

The frequent association of combined surgical and metabolic problems make diagnosis and management of diabetes difficult. The surgical problem may be the result of an uncontrolled hyperglycaemic state. Caution needs to be exercised regarding the reliability of physical findings or laboratory results in diabetic patients. The paucity of symptoms in diabetic patients with intra-abdominal pathology is seen frequently by general surgeons, a fact attributed to sensory neuropathy.

The presence of neuropathy may lead to gastroparesis which in turn can cause aspiration pneumonitis if timely nasogastric suction is not done or neuropathy involving bladder may lead to urinary retention demanding catheterization. Patients with cardiac autonomic neuropathy have an inherent risk of cardiorespiratory arrest leading to high mortality rate.

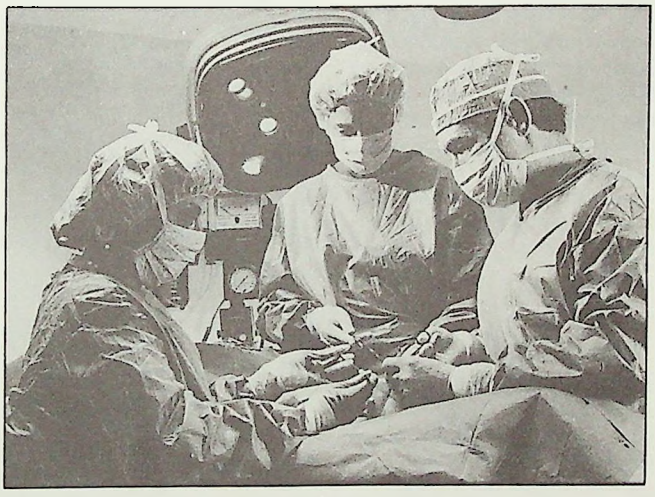
Physiological considerations

Hyperglycaemia during acute illness necessitating surgery may be caused by increased glucose production, decreased tissue utilisation and decreased renal clearance of glucose. Hyperglycaemia causes osmotic diuresis leading to water and electrolyte loss, resulting in dehydration. This may have detrimental effects on vascular volume and cellular membrane function.

"Ketone bodies", like acetoacetic acid and betahydroxybutyric acid are not toxic per se, but induce harmful effects by their ionization at body pH. If allowed to accumulate, these result in systemic acidosis which has detrimental effects on cellular function, eventually inducing cardiovascular collapse.

Major stress induces the secretion of stress hormones that counteract the effects of circulating insulin. Fever which is not uncommon during post-operative period leads to increase in stress hormones and thereby rise in blood glucose and ketone concentrations.

Col J.S. Saini, *Endocrinologist, Army Hospital, Delhi Cantt*



In the diabetic patient, insulin resistance also occurs, this calls for adjustment of insulin dose on an individual basis based on blood glucose monitoring.

Patients are usually fasted for at least eight hours prior to surgery so that aspiration does not occur. A decrease in external glucose results in stimulation of endogenous production of glucose and through mobilisation of fatty acids from adipose tissue to enhance hepatic ketone production to provide tissues with alternative energy source. A relative deficiency of insulin decreases the peripheral utilisation of ketone bodies, incresing their plasma concentration. Thus fasting sets the stage for the development of ketoacidosis.

Pre-operative considerations in emergency surgery

The major objective in the medical management of a diabetic during surgery is to maintain metabolic homeostasis, prevent fluid imbalance, protein catabolism, impaired wound healing and lessen the risk of infection. Majority of emergency operations are because of severe infections. These acute conditions are frequently conducive to DKA. The metabolic disturbances complicate the diagnosis and management of the underlying surgical problem; for example, DKA can cause abdominal pain mimicking surgical abdomen. Correction of metabolic disorder may alleviate the abdominal symptoms of DKA or permit more accurate localisation of the causative factors in the surgical abdomen.

Sufficient rehydration and electrolyte replacement and insulin treatment can be achieved within 4-6 hours to improve hyperglycaemia and to suppress ketogenesis. In the presence of DKA insulin is infused by pump at a rate of 6-10 units/hour after an i.v. bolus of 6-10 units. Saline solution should be started with administration of 1000 ml in 1-2 hour followed by 500 ml/hour thereafter. Potassium chloride/ phosphate frequently needs to be added to the second litre of solution. Plasma glucose concentration would decrease by 80-100 mg/dl hourly as a combined result of dilution through fluid replacement and insulin induced glucose uptake and decreased hepatic glucose output. For patients in hyperosmolar non-ketotic state, insulin doses are generally lower and obese patients with sepsis have significantly higher insulin requirements. Where the blood sugar level is reduced near 150-300 mg/dl, 5% dextrose is given in saline. If possible, surgery should be delayed until after this initial interval of metabolic correction.

Factors favouring metabolic decompensation

Several factors in the perioperative period predispose the diabetic patient to metabolic decompensation. These include:

1. Absolute or relative insulin deficiency
2. Stress hormone excess
3. Fasting
4. Dehydration

Management of ketonuria and hyperglycaemia require increase in dose of insulin whereas concurrent ketonuria and normoglycaemia require glucose infusion along with insulin. It is important to note that ketonuria may become more marked despite decreasing blood glucose level before improvement is noted. This is due to conversion of beta-hydroxybutyrate to acetoacetate during resolution of the ketonemic state, and the latter is measured in urine ketone test.

The patient presenting with metabolic crisis and requiring immediate surgery represents a substantial operative risk. There are two approaches that can improve the outcome. The first is the ability to temporize or delay the surgical procedure most often through the use of radiologic or endoscopic intervention. Examples of this include endoscopic percutaneous transhepatic drainage of the biliary tree in a patient with cholangitis or cholecystitis, or colonoscopic detorsion of a sigmoid volvulus. The second is the successful treatment of the metabolic disorder. However, if metabolic situation is secondary to a surgical problem and the patient is responding poorly despite appropriate therapy, surgery should not be delayed further.

Intraoperative management

In the past, regional anaesthetic techniques were preferred because

general anaesthetic agents resulted in sympathetic stimulation leading to release of epinephrine which aggravates hyperglycaemia and predisposes to DKA. Newer techniques however are very safe and without these side-effects. Intraoperatively, hypoglycaemia is most dangerous and difficult to diagnose. Therefore, frequent glucose monitoring is important. The blood sugar and electrolytes are maintained by using GKI drip and doses are adjusted appropriately.

Surgery and patients with IDDM

In IDDM patients, various methods have been advocated and these included giving various proportions of patient's usual morning insulin doses (one half or two thirds) followed by glucose infusions of various rates and concentrations. A more physiological attractive version is to give the full morning insulin dose, followed by a glucose infusion (usually 10%) at a rate to match the patient's usual dietary intake.

At present, a system which continuously provides intravenous glucose and insulin is preferred. One of the methods is the use of low doses of insulin (0.5 - 1.0 U/h) by infusion pump accompanied by glucose drip. However, glycaemic control using 'mini pump' system is not always good and with such low insulin delivery rates there is some risk of metabolic decompensation. Larger doses of both insulin (2-4 U/h) and glucose (5-10 g/h) are therefore preferable.

Practically, there are two methods by which insulin and glucose can be delivered. The first is to use separate intravenous lines, one to deliver glucose (usually 100 ml 10% dextrose solution given per hour) together with a 'piggy backed', infusion of insulin given by a syringe driver (usually at 2-4 U/h). The second 'combined infusion' method is now the most popular and comprises glucose and insulin mixed in a single infusion bag along with small amount of potassium to prevent hypokalaemia. This glucose- potassium-insulin, or 'GIK system' has gained widespread acceptance due to its simplicity and effectiveness. The combined GIK infusion generally contains 500 ml 10% dextrose, 10 mmol potassium chloride and 15 units short- acting insulin infused over five hours. The insulin content of the infusion is altered by substituting a new bag according to frequent blood glucose monitoring and potassium content is varied depending on the result of regular plasma electrolyte tests. Dilutional hyponatraemia may rarely occur when GIK infusion is prolonged. This should be treated by additional saline infusion and if necessary slowing GIK slightly.

Post-operative management

Once metabolic derangement has been corrected, the post-operative state is often uncomplicated. Reinstitution of normal pre-operative insulin regime is usually possible within a few days. Failure of improvement in metabolic status is indicative of infection which should be treated vigorously. Oral drugs can be started when the patient starts taking full meals.

Open-heart surgery, cardiac surgery with cardiopulmonary bypass requires greater amounts of insulin than other operations because of the fact that glucose containing fluids are used to prime the bypass pump. Also, unusual degree of surgical trauma, hypothermia and liberal use of inotropic drugs enhance the demand for insulin. In these settings, GIK system is not very effective and a separate line system with very frequent blood glucose monitoring is advisable.

Conclusion

The care of the diabetic patient needing emergency surgery requires expertise. The primary effort should be concentrated towards the correction of glucose and electrolyte abnormalities in a planned and controlled manner. The use of percutaneous drainage techniques can be extremely helpful in diabetic patients with undrained septic foci during the period of metabolic imbalance. An experienced anaesthesiologist and intraoperative cardiovascular monitoring can provide best opportunity for speedy and less complicated recovery.

Insulin in surgery

It is well known that intermittent injection of foreign protein produces greater antibody response. The same holds true for unpurified insulins. The fact that most diabetics undergoing surgery are non-insulin dependent and may require insulin only intermittently is good enough reason to use only purified insulin (ideally human insulins) in these patients.

Calories Chart

The calories given below are basically for a person weighing around 70 kgs. People who weigh less than this may spend relatively less calories in carrying out similar activities whilst those who are more than this weight would spend that much more calories.

Activity	Calories spent per minute
Lying down, sleeping, sitting.	1 to 1.25
Standing, strolling (1 mile per hour), playing cards, knitting, sewing, darning, desk work, car driving, electric typing, using calculators, etc.(NOTE : in many of these activities, the calories spent may increase if the activity is associated with stress, anxiety, anger etc.)	2 to 2.5
Level walking (2 miles per hour), level cycling (5 m.p.h.), horse-back riding (walking speed), playing musical instruments like piano, playing billiards and snooker, golf using a power cart to move around, manual typing, auto, T.V. and radio repair.	2.5 to 4
Walking at 3 m.p.h., cycling at 6 m.p.h., volleyball (6 men non-competitive), horse riding (sitting to trot), playing golf with lugging around the golf bag, sailing (handling small boats), badminton (social doubles), cleaning windows.	4 to 5
Walking at 3.5 m.p.h., cycling at 8 m.p.h., table tennis, golf (carrying clubs), dancing (at a pace of a dance like the fox-trot), badminton (social singles), tennis (social doubles), any callisthenics, painting walls, light carpentry (hobby).	5 to 6
Walking at 4 m.p.h., cycling at 10 m.p.h., roller skating, horse riding (trot), gardening (digging).	6 to 7
Walking at 5 m.p.h., cycling at 11 m.p.h., badminton (competitive), tennis (social singles), light downhill skiing, water skiing.	7 to 8
Jogging at 5 m.p.h., cycling at 12 m.p.h., basketball, vigorous downhill skiing, carrying loads of around 36 kgs.	8 to 10
Running at 5.5 m.p.h., cycling at 13 m.p.h., playing squash (social level), handball (social level), vigorous game of basketball.	10 to 11

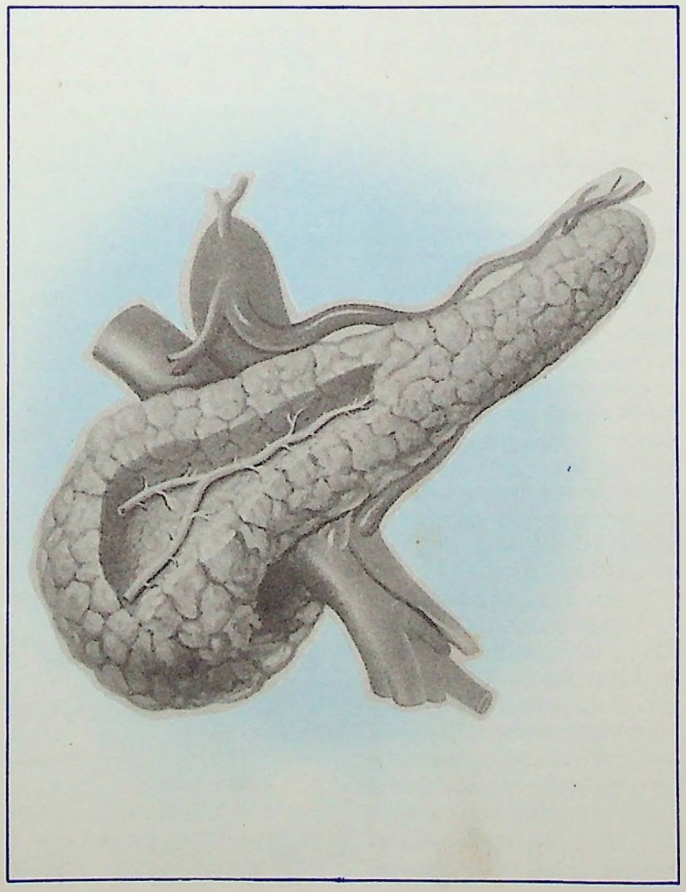
Looking Forward

Pancreatic Transplants

As insulin is secreted by the beta cells situated in the pancreas, it was but natural that efforts would be made to try and transplant a pancreas in insulin dependent diabetics who are completely deficient in the hormone. The hope being that if the transplanted pancreas does function, then it would secrete enough insulin to practically "cure" the patient! The first pancreatic transplant was performed way back in 1966 at the University of Minnesota by Dr. Richard Lillehei. The initial results were very disappointing, but surgical techniques and methods to help preserve the transplanted organ have steadily improved since then and Dr. Sutherland, from the same university who at present has the greatest experience with these transplants is optimistic about the future of this operation. Unfortunately, his optimism is not shared by most other authorities. As of last year around 2700 pancreas transplants had been done worldwide, most of these in association with kidney transplants. Inspite of all the advances only half the pancreas which are transplanted do show activity at the end of one year, and there is still considerable morbidity and the occasional mortality associated with this operation.

Besides the surgical skill involved, one of the major problems associated with pancreatic transplants is the need to immunosuppress the patient who receives the organ. In its absence, as with all other organ transplants, the immune system of the patient's body will reject the transplanted organ. A major breakthrough in transplantation surgery occurred in 1979 with the introduction of a drug called cyclosporin. It helps in immunosupression and now the use of cyclosprin along with other drugs such as imuran and prednisolone do improve the chances of pancreas survival after transplantation. Last year may have brought about a new breakthrough in immunosupression therapy. A Japanese company has introduced a new drug called FK-506, derived from soil fungus, which is undergoing trials. It is supposed to have 100 times the efficacy of cyclosporin without many of its side effects.

The use of immunosupression is associated with phenomenal side effects. The supression of the immune system means that the patient is defenseless against any infection. Even a minor illness which in normal circumstances would not even bother most people would lead to a major problem in these patients. There is also a fear that long-term intake of these drugs may lead to malignancies like lymphomas occurring in the patient. Add to this the cost of therapy and one will realise that pancreatic transplants still have too many problems which need to be sorted out.



Even if these problems are ironed out, there is still a major drawback, especially in our country, of producing enough organs for transplants. Unlike the kidneys, which we have two and one of which can be safely donated to a recipient, we have only one pancreas.

Although efforts have been made to see if half a pancreas can be removed, this does not seem to have worked in managing the diabetes in the recipient and, the safety for the donor has not really been worked out. Thus, the only method of getting a pancreas for transplants is from cadavers of people who have donated their bodies for such medical purposes. This is very rare in our country for social and, importantly, medico-legal reasons, and one would never be able to get adequate organs for transplants.

It was hoped that when pancreas transplants were carried out, the better glycemic control would improve other chronic complications like retinopathy and neuropathy. Unfortunately, studies from two major centres have shown that pancreas transplants do not lead to any improvement in the state of the retina or the nerves. This does not mean that good blood glucose control does not help in managing these complications, but it could be that the time at which the transplant is carried out is too late. Many of these chronic diabetic complications may reach a stage of such severity that glycemic control at this time may not be of substantial help. Thus, if the aim is to avert or at least minimise the chronic complications then the organ transplant should be done at a much earlier time. The need for an early transplant, the paucity of organs, morbidity associated with the operation and the cost and side effects of immunosuppressive therapy, all leads one to conclude that pancreatic transplants, presently, are associated with too many drawbacks to gain wide acceptability.

But from this concept, investigators have explored the possibility of transplanting the beta cells themselves, raising exciting possibilities.

Myths and Concerns about insulin use in Type II Diabetes

Once on insulin always on insulin

It is assumed that once a patient is put on insulin he will continue requiring insulin. While this is true of Type I diabetes, it is not so, in Type II diabetes. It is wrongly assumed that exogenously administered insulin will lead to disuse atrophy of the beta cells. This has never been shown to occur in practice; on the contrary, by reducing glucotoxicity, insulin therapy improves beta cell function. Thus it may be used intermittently during periods of disordered control to reverse the metabolic derangement quickly.

Case Study

Mr. P.F.A. a male aged 38 years was admitted in a comatose condition around 6 a.m. The accompanying relative informed the resident on duty that due to the fact that the patient regularly got up by 5.30 a.m., efforts had been made to wake him up, and when this failed, he had been urgently shifted to the hospital. The only history that the relative could give was that the patient was a diabetic, taking insulin, and the family physician had told them that the blood glucose control of the patient was poor, possibly due to erratic eating habits. The patient was comatose with a pulse of 110/minute, regular but "racy". The BP was recorded as 180/95. The patient felt cold and clammy and his clothes were wet possibly due to sweating. The heart sounds very well heard, there was no murmur. The examination of the chest and the abdominal system was normal except for sinus tachycardia, and the examination of a catheter specimen of urine showed the presence of trace of glucose but no ketones. The random blood glucose estimation done by pinprick and the use of a "stick" showed the blood glucose to be below 45 mg%. After blood had been collected for other routine tests, 100 c.c. 25% glucose was slowly infused and this was followed by a drip of 5% glucose. The patient gradually regained consciousness and in a few hours seemed to be back to normal.

At this time, a further detailed history was elicited. The patient informed us that he had been diagnosed as a diabetic about 2 months previously and had been put on a single prebreakfast dose of 20 units of Lente insulin. This had been gradually increased to 32 units of Lente insulin and a week back, when his blood glucose had been examined, the fasting blood glucose was found to be 160 mg% whilst the post lunch values were 172 mg%. Surprisingly, his glycosylated hemoglobin levels were in mid normal range. In view of the fasting hyperglycaemia, the prebreakfast dose of the insulin had been increased to 36 units and 3 days later to 40 units. Inspite of this, the fasting blood glucose levels could not be controlled, and in fact there was some rise seen. At this time, the patient volunteered the information that he had been feeling quite unwell, and when seen by his doctor had complained of tiredness and early morning headache. For the first time, his BP was found to be slightly high. His tiredness had been attributed to the uncontrolled diabetes and the headache to the high blood pressure.

The previous morning, he had been asked to take his usual dose of 40 units of Lente insulin. In addition, he was asked to take 10 units of Lente insulin at his pre-dinner time and also to restrict his bedtime quota to milk. It was hoped that this would decrease the raised early morning blood glucose levels.

This case report illustrates a relatively common clinical problem wherein the fasting blood glucose levels are higher than acceptable whilst the control throughout the rest of the day seems to be fairly good.

1) What are the common causes for this to occur?

2) How does one differentiate between these causes?

3) In the case report were there any "warnings" of impending hypoglycaemic coma which should have alerted the treating doctor?

4) How would you have approached this case, if the patient had come to you with rising blood glucose levels inspite of increasing doses?

5) What is the further management of this patient?

This case report illustrates a relatively common dilemma seen when patients are treated with "conventional" insulin regimens. The fasting blood glucose levels tend to be in the unacceptably high range whilst the control throughout the day may be quite fair! From a practical viewpoint, there seem to be three main causes for this:

1. A simple waning of the insulin effect so that there is not enough insulin at night-time to maintain control over the blood glucose levels in the early morning period
2. The Dawn phenomenon
3. The Somogyi phenomenon

It is of greater importance to correctly distinguish between the Dawn and the Somogyi phenomenon, as the management of these are radically different and a misdiagnosis can lead to quite a catastrophe, as happened in this patient.

Dawn phenomenon

When insulin dependent diabetics were closely monitored during intensive conventional therapy or whilst being on continuous subcutaneous insulin infusion (CSII), overnight monitoring of the blood glucose levels showed that they rose and the insulin requirements increased between 3 a.m. and 7 a.m. and this increase in the insulin requirements and the hyperglycaemia that followed was referred to as the "Dawn" phenomenon. Later, this phenomenon was also reported in non-insulin dependent diabetics and even in people who did not have diabetes.

It is now fairly well established that many hormones undergo diurnal variations. These changes in hormonal levels occur both, in diabetics as well as in non-diabetics, but tend to be more pronounced in a diabetic. Soon after the onset of sleep, there is a surge in the secretion of growth hormone. Cortisol shows a significant increase between 2 a.m. and 4 a.m. and reaches peak values shortly after waking up. The catecholamines gradually increase overnight whilst glucagon remains relatively constant. Growth hormone, cortisol and the cathecolamines are all potent insulin antagonists and these changes in the secretion of the antagonists seen during the overnight period could explain the increased insulin requirements and the consequent early morning hyperglycaemia.

Somogyi phenomenon

The Somogyi phenomenon is a manifestation of hyperglycaemia following, possibly unrecognised, hypoglycaemia. The clinical picture here, is one of worsening control in the face of increasing insulin doses and is manifested especially by early morning hyperglycaemia. Excessive insulin dosage could lead to hypoglycaemia, which occuring at night-time when the patient is asleep, may go unrecognised. But the counter-regulatory hormones (cathecolamines, glucagon, growth hormone and cortisol) rise as a consequence of the hypoglycaemia and their effect on the liver causes the blood glucose to rise. As there does not seem to be "fine tuning" of the magnitude of the counter regulatory responses and because these hormones also cause insulin resistance, there is often an overshoot of the blood glucose which tend to go much higher than normal. Increasing the amount of insulin in order to counter the fasting hyperglycaemia serves to intensify the nocturnal hypoglycaemia and increase the counter regulatory response. This may be seen as a further increase in the fasting hyperglycaemia inspite of increased insulin doses. Too much of an increase in the insulin dose could of course lead to a level of hypoglycaemia from which the counter regulatory hormones may not be able to increase the blood glucose levels and the patient may manifest with frank hypoglycaemic coma.

Waning of insulin effect

The third condition which commonly leads to a fasting hyperglycaemia is a waning of the insulin effect seen overnight. In other words, the amount of insulin in the body decreases overnight and this decreased amount of insulin is not enough to prevent a rise in the blood glucose levels. Fortunately, the management of the Dawn phenomenon and the problems of waning insulin levels is basically the same and therefore, the main clinical problems is the differentiation between the Dawn phenomenon and the Somogyi phenomenon.

Differential diagnosis

The best way to distinguish between the two would be to estimate the blood glucose levels at different times throughout the night. The patient could do self monitoring of the blood glucose levels at home, or if hospitalised, this could be done by more conventional methods. The estimations should be done before the post-dinner snack (or bedtime), at midnight and at 3 a.m.

In patients showing the Somogyi phenomenon, a typical pattern would be normal or near normal, blood glucose levels at bedtime and midnight which decrease appreciably around 3 a.m. It has been shown that a value of around 70 mg% at 3 a.m. would suggest the presence of the Somogyi phenomenon. In patients manifesting the Dawn phenomenon, the blood glucose levels at bedtime, midnight and at 3 a.m. would tend to be similar and after this time, the blood glucose levels would increase to reach hyperglycaemic levels by early morning.

If it is not possible to do the blood glucose estimations, one method that may give a clue to the presence of the Somogyi phenomenon is to do a urine glucose estimation (in a double voided specimen) around 3 a.m. and in the early hours. If the former shows no glucose but the latter is MARKEDLY positive, the patient may be experiencing post-hypoglycaemic hyperglycaemia. There are several other clues that may also point to this diagnosis. Rapid fluctuations in the results of the urine tests showing negative results and then suddenly changing to maximal values. Wide swings in the blood glucose levels which are not related to meal intake is another pointer to the diagnosis.

Management strategies

Once the diagnosis of the problem is clear, the management does not offer any difficulty. In patients with Dawn phenomenon, one needs to increase the overnight insulin whilst the reverse seems to be the case with the Somogyi phenomenon. How this is done would obviously depend on the individual patient. For the management of Dawn phenomenon, one or more of the following could be done:

(*Contd. on Pg. 12*)

The Diabetic Foot

(*Contd. from pg. 2*)

very cold. This means that the patient should always check the temperature of the water before pouring it on his feet. Some authorities advise that the temperature of the water be checked by the hands (if the patient withdraws the hand very rapidly, it is very hot). However, it needs to be borne in mind that neuropathy may also affect the nerves going to the hands and these too may be relatively insensitive to the heat. It would be best to check the temperature of the water using the elbows.

There is also a tendency in some patients to soak their feet for a while. This is especially true in those who use bathtubs and many patients who have aching feet use tubs or basins to soak their feet in order to get some relief. Worse still, some put a disinfectant solution in the basin, in the misconception that this will clean the feet better. One should never soak the feet as this often allows the patients' skin to come into contact with the warm water for too long. More importantly, this causes the skin to become macerated and such a skin is very prone to act as an entry point for infection. The use of disinfectants should be discouraged because the wrong disinfectant or even a mild one in a strong solution can damage the skin considerably.

Similarly, the soap that is used for the bath or the washing of the feet should be a very mild one, may be a baby soap. After all, the skin of a diabetic should be treated with as much care and attention as that of a newborn baby! The practice of using rough stones to scrub the feet, especially the soles should be avoided. It leads to too many small cracks and fissures in the skin.

Drying the feet

The towel used to dry the feet should be of the soft variety. Coarse towels can cause as much damage as the use of stones for scrubbing the feet. The feet should preferably be patted dry rather than be rubbed. Often, many patients give their feet a brisk rub down in the hope that this would improve the circulation. This really does not help and can cause minute breaks in the skin from where infection can enter. Careful attention is to be paid to patting dry the area between the toes as these often tend to remain wet and may macerate.

After the bath

This is the time that most of the patients should examine their feet. If the feet are very dry, then one may need to apply some mild lubricant like "baby" oil. Conversely, if the skin is moist, then it would be better to apply some mild powder especially between the toes. The point to remember is that the skin should be neither too dry or too moist and therefore there should be a fine balance between the use of lubricants and powder.

Nails

The best time to manage the nails is after washing the feet as the nails are relatively softer at this time. Nails should never be cut by the patient but he should only file them so that no sharp edge is left. There is no need for cutting the nails very close to the edge or trying to shape them by cutting the side edges inwards. The sharp edge of the scissors tends to cause minute injuries and infection often starts here.

Medical advice

Many of the patients we see with serious foot problems seem to have brought it upon themselves by trying to indulge in self doctoring. Some patients when they see a corn or a callus are tempted to take a knife or some other sharp object and try to remove it. Such self-inflicted wounds are often painless and may not be noticed until serious infection has supervened. One should never use commercial preparations which are available in the market for treating calusses and corns including corn pads and adhesives. In fact, it would be worthwhile to avoid applying any medication to the skin unless it is under medical supervision. Most of the medicines available for applying to the skin may turn out to be too strong for diabetic skin. Strong medicines burn the skin. Patients often use medicines like mercurochrome to treat mild infection or injuries. This is not only too strong but is usually ineffective. The colour of such medications tends to mask the redness that may be the only sign of spreading infection in the absence of any pain sensation.

Socks

The best socks to wear are soft cotton ones. These tend to absorb moisture but this may end up as an advantage in our country where one tends to perspire a lot. The socks should not have tight elastic top as this may interfere with the blood circulation to the feet. It may be better, under the circumstances, to allow the top of the socks to be loose even if they have a tendency to roll down a little.

Footwear

This is one of the most important areas in preventing trauma to the feet. Unfortunately, this is an area where one comes across the most obstinacy. A diabetic should never walk barefoot. The use of chappals really offers no protection against trauma and their use is as good as going out barefoot. Unfortunately, many patients refuse to change over from wearing these chappals inspite of all persuasion, saying that the use of shoes is not only uncomfortable but goes against their traditional dress style. In such cases, one compromise would be to insist that the chappals should be stitched and not have any nails in them..

Women also rebel against the use of shoes, but this is an argument that does not hold good especially in urban areas where excellent shoes for women are routinely available. Another argument that is used is that the shoes that the doctor feels are good for the feet are not fashionable enough! This may be true in many cases as unfortunately, the arbiters of fashion do not take into consideration the skin condition of a diabetic. At the same time decent looking shoes are available for women which may not be in the height of fashion but are nevertheless quite good looking and suitable for wearing by a diabetic. For those who are too vain to accept anything but the latest in fashion, the best thing is to tell them, "Think how fashionable you would look if you lost a foot by not taking care".Not that the problem of vanity is the sole prerogative of women, I have come across many men with the same attitude and have used the same argument with telling effect.

Let us now come to the type of shoes that should be worn. They should be made of soft leather rather than of any other synthetic material. They should be big enough to accommodate the foot spaciously, especially, the front part which should not bunch up the toes. Many Patients feel that such spacious shoes make them look awkward and have a tendency to choose shoes that are a size too small. The patient should be advised to make an imprint of his feet on a piece of paper and the shoes that he buys should be broad enough and long enough to completely cover the imprint. This should be done for both the feet . Often there maybe slight variations in the two feet and what may be allright for one foot may cause the same size shoes to be slightly tight for the other foot. In this case, he will either have to get shoes that may be slightly different in size or failing this he may have to have some shoes made specially for him. This may also have to be done by others who have special problems like cocked up toes, high arches of the feet or other structural deformities.

The shoes should have uppers made of soft leather. The soles of the shoes should also be of leather since rubber (or other synthetic) soles do not offer adequate protection. How often have we seen thorns or nails push right through the rubber soles? The shoes should be stitched rather than nailed together. The soles and the insides of the shoes must be closely examined before the shoes are put on everyday. One would be surprised by what may be sticking out through the soles or even be embedded inside the shoe.

All new shoes even if they are spacious, need breaking in. New shoes should never be worn for more than an hour at a time. What this implies is that one should not wait till the last minute before getting new shoes. It is advisable for the patients to keep two pairs of shoes so that the other pair can be comfortably worn in an emergency. They should also get some new shoes much before they would need to be worn regularly. These shoes can be then worn at home every day for a short while so that they would get gradually broken in and so could be worn regularly when the old shoes finally wear off. Similarly, many of us have the tendency to wear out the shoes completely and only change them when the soles of the shoes have given way completely. This is obviously a wrong practice as torn soles provide no protection and may be as good or bad as wearing no shoes at all!

If these simple guidelines are followed, the chances of preventing serious foot problems from developing would be very bright. Many patients may feel that this is all getting unduly finicky about the need for excellent foot care. But considering so many patients with serious foot problems and the morbidity associated with this, one must understand that there is no better treatment than prevention. After all, the old saying,

'For want of a nail,
the shoe was lost;
For want of a shoe,
the horse was lost;
For want of a horse,
a war was lost',

should not come chillingly true for the patient!

Practical Aspects of Insulin Therapy

Storage

One of the most common problems that patients seem to have is about the storage of insulin that they are using.

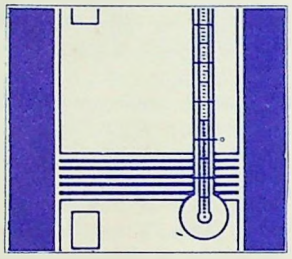
Insulin preparations are temperature sensitive and require proper storage. It is recommended that insulin vials be stored between 2-8°C i.e. in a refrigerator. If not stored in this way, the physiochemical characteristics of insulin preparation may be altered and may affect the potency. Insulin preparations should not be exposed to heat or sunlight.

The newer purified insulins that are being increasingly used these days are even more stable than the older insulins. This is due to the fact that the newer insulins have a neutral pH. Occassionally, when the longer acting insulins are not properly stored, they tend to form clumps. It may be difficult to withdraw the insulin through the needle and the vial should be discarded.

The older conventional insulins, which many still use, are available in most places in India. This is especially true for all the cities, towns and district places. The newer insulins are also available quite widely. With this relatively wide availablity of insulin, there does not seem to be any need for storing a large number of insulin vials for future use. It is advisable that the patients only keep one extra vial for use in an emergency like breakage of the vial currently in use. At the same time, there are patients who stay in villages where the insulin may not be freely available. Under these circumstances, there may be a need to stock insulin vials. However, extra vials, if any, must be stored in a refrigerator, especially in those places where the room temperature would be high for many months in a year. The vials should NEVER be kept in the deep freeze or the freezer section of the refrigerator. In fact, any insulin vial that has been kept in this freezer section should be thrown away. This point is, unfortunately, not too well known and many patients do tend to stock the insulin vials in the deep freeze or the freezer section under the mistaken notion that this would ensure that the insulin would be preserved well. In fact,many chemists keep their stock of insulin in the deep freeze and only remove the number of vials which they feel they would sell during the day, and then store these in the non-freezer section of the refrigerator. Only a few of the patients taking insulin, would need to store a large stock of insulin, whilst for most of the patients, storing of the insulin vials should never be a problem.

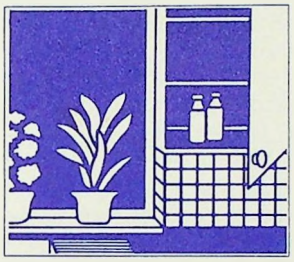
The insulin vial and the accessories can be carried in the travelling bag. The patient should carry the vial that is currently being used in the bag that he will carry with him personally rather than keeping it in the baggage compartment of the train or checking it in at the airport. Because it is rather doubtful that the patient and his baggage would arrive at the same place and at the same time! It is also highly advisable that patients should always carry extra insulin vials and syringes etc., to face emergencies like a stay of longer duration than expected, breakages, loss and other similar problems, especially if they are travelling to places where replacements may not be easily available.

Guidelines for Storing Insulins



Whenever possible, store the insulin in a refrigerator at the recommended temperature of 2-8°C (36-46°F)

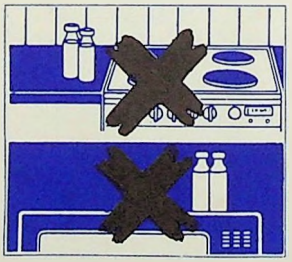
Do not use insulin that has been frozen (be careful not to place the insulin too near the cooling system of the refrigerator or the freezer box)



If you cannot store your insulin in a refrigerator, keep it in a cool and dark place

Keep the insulin out of sunlight, or else it will lose its effectiveness

Do not subject the insulin to high temperatures, such as can occur:-
a) in the glove compartment of a car
b) on a sunny window ledge



c) near a cooking rage
d) on top of electrical equipment, e.g. tape recorders, TVs etc.

When travelling in aeroplanes, the insulin should be carried in the cabin hand baggage and not in suitcases, etc. that will be transported in the hold

Carry extra vials while travelling abroad, as you may not get your brand or strength of insulins

Diabetes Post Line

I am often asked by my diabetic patients about the chances of their children also getting diabetes. Is there any way one can predict the risk for such children to get diabetes?

There is no way that one can confidently predict the chance of a child of a diabetic parent for getting diabetes. All such children should be considered at high risk and be tested yearly, so that the onset of diabetes can be diagnosed at the earliest. It would be prudent for such children to maintain ideal body weight, exercise regularly and treat any associated conditions like lipid problems, hypertension, etc.

At the same time, many years ago, Steinberg after studying the family histories of many patients had charted the relative probability of a person, with a family history of diabetes, for getting diabetes. This chart is shown below.

Such charts may not be directly useful for individual patients. After all, the patient who asks this question is not interested in statistics and probability, but is only interested in the risk for his children! Thus, the best answer that one can give for such questions is that all these children should consider themselves at a high risk for getting diabetes at some stage in their lives.

Probability (Per cent)	Diabetic relatives
100	Identical twins or both parents
50-80	One parent and a sibling of the non-diabetic parent
	One parent and a parent of the non-diabetic parent
	One parent, a sibling, and a grand-parent via the non-diabetic parent
50	One parent and one sibling
30-40	Two grand-parents (not spouses)
	One parent and first cousin on the non-diabetic parent's side
25	One sibling
20	Two grand-parents (spouses) or one parent
	One grand-parent
	Uncle or aunt
	First cousin

I have a patient on chlorpropamide who recently went into hypoglycaemic coma. He recovered when I injected intravenous glucose but then after a while, I was called in by the family again as the patient had again gone into coma. I again injected intravenous glucose and the patient became quite normal. Later a colleague of mine told me that he had been called by the family in the early morning, as the patient would not wake up. Was I dealing with something different from hypoglycaemia, and if so which conditions would make the patient recover with intravenous glucose and then relapse. How should such patients be treated?

From the history given, surely, you were dealing with chlorpropamide induced hypoglycaemic coma. Patients who get a severe hypoglycaemic attack whilst on chlorpropamide do have the tendency to go into repeated attacks of hypoglycemia, even though the correct treatment has been administered for the initial attack. The reason for this is the long duration of action of this drug. Although, most books mention that the effect of chlorpropamide lasts for around 36 hours, this is usually after a single dose has been administered. When patients are given 250-500 mg. of the drug daily, a steady state is reached after 4 days. When the drug is continued, the duration for which the drug stays in the body, and exerts its blood glucose lowering action, is much more. Studies have shown that if a patient takes the drug for 2 weeks and then discontinues it, the hypoglycaemic action may, in some patients, last for a further 2 weeks. The effect of the drug often lasts for around 5-7 days after a severe hypoglycaemic attack in patients taking 250 mg. of chlorpropamide daily.

When intravenous glucose is injected, the rise in the blood glucose levels allows the patient to regain consciousness. But the hypoglycaemic action of the drug may last even after the effect of this administered glucose is over. Thus, the patient relapses, and this sequence may be repeated. The best way to manage such episodes in general practice is to inject intravenous glucose, see that the patient takes a snack of complex carbohydrates and then hospitalise the patient. Often, such patients, especially if they are older and have had a severe attack may require continuous intravenous glucose for several days before the effect of the drug wears off.

What is the time that should elapse between the taking of the injection and the subsequent meal.?

This is a very common problem and leads to funny situations where the patient sits with the food plate in front of him and then as soon as the injection is given he starts eating very fast, feeling afraid that otherwise he may go into coma. This misconception should be removed.

Generally speaking, the patient is advised that the insulin injection should be taken about 20 to 30 minutes before the meal. This is to allow some of the injected insulin to get absorbed into the blood stream so that the insulin levels have alredy started to rise as the food is digested and absorbed. This prevents the post-meal blood glucose from rising excessively. Even normally, it has been shown that the insulin secretion from the pancreas precedes the absorption of the food from the gut. Thus, giving the injection 20 to 30 minutes before the food is an attempt to mimic the normal physiology.

All the same, the time period between the injection and the eating needs to be individualised. There are some patients who are "slow activators". Such individuals tend to show a delay in the absorption of the injected insulin, the levels of which start to rise much later than expected. Normally, when a short acting insulin is injected, one expects that the effect would start around 30 minutes after injection and that it would peak after 2 hours and have an onset of action after about 6 hours.This is why one advises that the injection be taken around 30 minutes before the food. This allows the insulin levels to rise and have an effect when the food is absorbed. In slow activators, the rise in the insulin levels may be seen after possibly 60 minutes, the peak may be at 3 to 4 hours and the onset after 8 to 10 hours. The problem in such patients would be that insulin would not be available in adequate amounts at the time when required. When the peak rise in the post food blood glucose is seen at 2 hours after the food, the insulin action would not be at its peak. This would lead to an excessive rise of postprandial blood glucose levels. Later, about 3 to 4 hours after the meal, when the blood glucose is falling, the insulin action would be peaking and this may lead to a "hypo" at this time! In such patients, it may be necessary to prolong the time between the injection and the meal such that the peak insulin activity coincides with the period when the postprandial blood glucose would tend to be the highest.

Conversely, in "early activators," the peak activity may be seen with an hour and in such cases, there may be a possibility that the patient gets a "hypo" at this time as the eaten food may not have been digested and absorbed in this short period. In such circumstances, it may be necessary to eat right after the injection is taken. In severe case, the injection may need to be given after the meal although this is quite rare. Thus, whilst most of the patients may need to have a gap of about 20 to 30 minutes between their insulin injection and the meal, this time interval needs to be individualised in many cases.

Diabetes Post Line

As a part of its activities, Diabetes Forum has started a Diabetes Post Line where our panel of experts will answer all queries which you may have regarding any aspect of diabetes management.

Please send your letters, questions, problems in management, etc. to:

Diabetes Post Line

MAGNUS

A Division of

Boots Pharmaceuticals Ltd

17 R Kamani Marg, Bombay 400 038

Replies will be sent individually. Queries which may be of general interest to practising doctors will be published in the issues of Diabetes Forum with your identity being withheld, if you so desire, We look forward to hearing from you.

Case Study

(*Contd. from pg. 8*)

1. Give a small pre-dinner dose of intermediate-acting insulin, if the patient is not already taking one
2. If the patient is taking a dose of an intermediate-acting insulin before dinner, this could either be judiciously increased or the timing of the injection delayed so that the maximum activity could be seen around 2-4 a.m. and
3. A small dose of regular insulin could be given before the post-dinner snack

The purpose of all these manipulations would be to increase the amount of insulin available in the body during the crucial 2-4 a.m. period. It is obvious that if an additional injection is given or the dose of the insulin is increased, corresponding changes must be made in the dose of insulin given at other times in order to prevent day time hypoglycaemia.

The strategies to manage the Somogyi phenomenon could be:

1. Decrease the dose of the evening insulin injection
2. Add, or increase, the amount of dinner or, preferably, the bedtime snack
3. Decrease the amounts of intermediate or long-acting insulins given during the morning

The patient, Mr. P.F. A., was obviously manifesting the Somogyi phenomenon in the previous days. There were several clues present which could have pointed to the correct diagnosis. There was a worsening of blood glucose control inspite of increasing the insulin dose as could be seen from the increase in the levels of early morning hyperglycaemia. The patient also complained of feeling extremely tired in the morning and of having a headache. His BP was found to be high for the first time after the increase in the insulin doses. An indirect clue would be the midnormal levels of glycosylated haemoglobin inspite of unacceptable fasting blood glucose levels. Since glycosylated haemoglobin tends to "average" out the ambient blood glucose levels, a midnormal glycosylated haemoglobin estimation along with high fasting blood glucose and "fair" post-meal values should have pointed out to the possibility that the patient may have been undergoing episodes of very low levels of blood glucose at some time during the 24 hours.

VIII National Diabetic Congress
December 3-5,1993
organised by
Diabetic Association of India.

Venue
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VIRAL HEPATITIS

What is Jaundice?

Jaundice is not a disease. It is a manifestation of an underlying disease like the fever in Typhoid or 'flu'. Although most often it occurs as a result of an underlying liver disease, it can occur as part of various other problems quite unrelated to the liver. The lesson here is, when jaundice occurs find the cause for it and then only a proper treatment can be given. As many of the drugs taken have to be metabolised by the liver to make them effective, excrete or detoxify, do not blindly take remedial measures which may infact further harm the already sick liver.

What is Hepatitis B (HB) ?

It is a virus (HBV) infection which is highly infectious. It makes the liver swell and affects the functions of its cells. Most

often it may be a self limited process but sometimes it can perpetuate the damage leading to cirrhosis (scarring of the liver) with an increased risk of developing liver cancer. In India, we do not know how many people are affected by this illness every year due to lack of proper statistics. When a developed country like U.S.A., more than 25,000 people contact Hepatitis B per year, certainly the figure must be much higher in our country.

Fortunately, for many people the body's natural defences will fight off the virus and develop immunity to it. However, about 2-10% of adults infected with HBV will become carriers or develop chronic HB which means that they remain infectious.

How is HBV transmitted?

HBV is 100 times more contagious than the AIDS virus. It may be transmitted through contact with infected body fluids, blood, urine, saliva, seminal fluid, vaginal secretions and breast milk. HBV can be transmitted through sexual contact (both hetero and homosexual) as well as through sharing of razors, toothbrushes IV needles, tattooing and other sharp instruments.In this context, I must mention that a preliminary report of a study conducted by me tends to show that a significant number of patients could have got infected by receiving injections with contaminated needles. It is therefore best to avoid injections when the same illness can be treated with oral medications and if injections are necessary to be used, only disposable needles and syringes should be used. It is a myth that a drug given by injection will be curing an illness more effectively in all instances.

Anyone who is exposed to blood or body fluids of an infected person is at risk. The hepatitis B virus can live outside the body for upto ten days on a dry surface.

Is there a vaccine for HBV?

There are safe and effective vaccines against Hepatitis B. Three injections are required for full protection. The second injection is given one month after the first, and the third is given six months later. The vaccine provides immunity for ten years or longer. As it will not 'cure' a person who is already infected with HBV it is advisable to check your blood to see whether you are already infected before the vaccination. The World Health Organization has recommended Hepatitis B vaccination to be included in the routine vaccination of children.

What are the Symptoms?

Very often this illness may have only minor symptoms and may be mistaken for an attack of flu and not all may even develop jaundice. The stronger the response of the body's immune response the more severe would be symptoms including the jaundice. Thus the

chronicity leading to cirrhosis of the liver may occur more often in perons who had a milder attack and neglected taking proper care and rest.

What is a carrier?

When the virus continues to persist in the body for more than six months, that person is considered a carrier. The most important fact about the carrier is that this person can pass the virus, unknowingly to others and yet remain without symptoms even for a lifetime. However, carriers have a higher risk of developing cancer of the liver.

What should a carrier do ?

Carriers should remember that the virus is present in all body fluids like blood, semen, vaginal fluids, urine and saliva. A carrier should never have unprotected sex unless the other person is immune to HBV or has received the vaccine. They should get a check-up every six months to assess the status of the liver. Alcohol which is toxic to the liver should be avoided.

For more information on Hepatitis B, please contact

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VDRL IN PREGNANCY

Q: Recently we have started doing routine VDRL for all antinatal patients. Occassionally we come across positive cases. Sometimes the dilutions are very low eg: 1:2 or 1:4. Should we treat these patient?

A: Routine Blood VDRL done during pregnancy is an excellent practise. The unfortunate occurence of congenital syphilis can be eliminated if this is done everywhere. Ideally VDRL is done at the first antenatal visit and at 32 weeks to eliminate the possibility of infection occuring after the 1st visit.

VDRL must be done correctly and whenever reactive, in higher dilutions to find the maximum dilutions. Later follow up will be possible only if these dilutions are documented.

VDRL is a non-specific test and can be reactive in conditions other than Syphilis which includs pregnancy. So it is not correct to assume that the person is suffering from syphilis whenever a VDRL [illegible] comes as reactive.

Please remember that any dilutions is possible in syphilis. But very high dilutions do not occur in other conditions. So high dilutions like 1:32 and above can be assumed to be due to syphilis and to be treated. The lower dilutions may or may not be due to syphilis. Other factors like history and clinical features should be looked into. If further doubt exists it is advisible to do specific test, eg:TPHA, FTAABS etc. But unfortunately very few centres in this country are doing this. So if it is impossible to rule out syphilis in pregnancy it is advisible to treat as for syphilis if patient is not allergic to pencillin. In such situation it is the duty of the doctor to explain to the couple that though the test may be positive due to pregnancy itself, it is also possible it is due to syphilis infection. The decision of giving treatment should be made only after taking the consent of the patient.

Often doctors do not realise the emotional and psychological reactions when some one is treated for VD. Occassionally, this can be a legal matter.

The treatment for early syphilis (ie. all manifestations that appear during the first 2 years after the infection) is as follows:

1. Inj. Benzathene Pencillin 24 lacs (PENIDURE LA 24) IM as a single dose.

   OR

2. Inj. PROCAINE PENCILLINE 8 lacs (PPF) IM daily x 10 days.

If the [illegible] or admitted procaine penicillin should be [illegible]

If the patient is reliable or admitted, procaine pencillin should be prefered.

If the patient is allergic to pencillin:

1. Cap Tetracycline 500 mg 6th hourly on an empty stomach x 15 days. OR
2. Tab Erythromycin 500 mg 6th hourly x 15 days.

Pregnant woman and children below 12 years should not receive Tetracycline.

After the treatment VDRL should be repeated once in 3 months x 1 year and once in 6 months till it become Non Reactive.

The VDRL is expected to become non-reactive 3 months - 18 months in early syphilis after the treatment.

DIS-1A.19

GENITAL HERPES

Genital herpes is a common sexually transmitted disease. It usually occurs for the first time within 2 weeks of sexual intercourse with a person having active disease. In some, the condition keep coming again and again even without further sexual intercourse. How often this may take place is difficult to predict.

The disease starts as one or more water bubble like blisters. Later these blisters break and small ulcers are formed. When these ulcers are present the person can infect the sexual partner during intercourse. When the ulcers are not present the person is not infectious to others. So it is advisable to avoid intercourse or use some protective device when ulcers are present.

If the partner also has the same disease there is no need to take any precautions.

Usually this disease does not affect the health of the person. But it can be a cause of much guilt, worry and anxiety.

Plenty of research is going on, to find a permanent cure for this commonest VD of the world. Some drugs are useful to cut short the duration of the ulcers. (eg. Acyclovir) These are used only in some special situations.

Many new drugs are being tried all over the world for a permanent cure. Till such medicines are available the persons suffering from the disease must accept the fact that there is not anything available anywhere in the world which is useful in stopping the disease coming again and again.

A simple test, usually done only by the skin specialists (TZANCK Test), can give evidence about the disease, if the patient comes at the stage of the blisters.

Blood VDRL which is done to detect syphilis will be non reactive (negative) in this disease. But it is done as a routine to rule out associated syphilis infection.

TREATMENT

Only gentle cleaning the area with plain water and soap is needed and the ulcers will heal within 3-7 days. If there is much pain or pus or ulcers take longer time to heal the patient should consult the treating doctor.

Venereal Diseases

1. There are about 15 venereal diseases. Most of them are easily treatable.
2. Venereal diseases are only transmitted by intimate and direct contact (usually by sexual intercourse) with a patient who is suffering from the same disease.
3. It is possible, though rare to get VD through unsterile injection needles and blood transfusion, but not by using a public urinals or eating strange foods.
4. It is possible to get more than one venereal disease from one contact. So it is important to complete the follow up with necessary urine and blood tests before assuming that one is cured of venereal diseases.
5. The blood VDRL is done to detect only one venereaal disease i.e. Syphilis. The usual cost of the test is Rs.10 to 20/-
6. VDRL can be positive even when there is no syphilis. Sometimes it is positive in other diseases and occasionally even in normal healthy individuals.
7. Blood VDRL can be positive in normal pregnancy without syphilis, and blood VDRL is routinely done in pregnancy
8. Specific tests (eg.TPHA) are available to know whether the patient is really suffering from syphilis.
9. Venereal diseases can be present in the body even though patient do not have any complaints but these diseases can be detected easily with simple urine and blood tests.
10. As a rule a period of 6 months should elapse before one can be certain of not getting VD after the previous contact. After this period if the patients blood + urine tests are allright and do not have any external sign of VD it can be safely assumed that the person is not suffering from any V.D.
11. Diseases other than VD can affect the genital parts producing much anxiety and guilt. It is important to consult a competent doctor and take an opinion.
12. Many normal conditions like pearly penile papules, Fordyce spots, cutis anserina, nocturnal emissions, spermatorrhea etc. can produce doubts as to the existence of VD. But a competent doctor can easily recognise these conditions and reassure the patients.
13. Pencillin injections are given for only 2 venereal diseases i.e. for syphilis and occasionally for gonorrea.

...2...

14. Medication to prevent the development of VD after suspected contact with the disease is not recommended at all.
It is virtually impossible to take treatment for all the 15 venereal diseases.

The wrong medication or inadequate dosage often suppress the manifestations of the VD and produce difficulties in the diagnosis and treatment of the V.D. later.

15. Some patients develop fear of venereal disease (Venereophobia) though they do not suffer from any VD or sometimes after getting cured from VD. In mild forms reassurance by the doctor is sufficient. In severe forms medications and sometime psychiatric help may be necessary.

16. The V.D. patient is the usual target of unscruplous laboratories and doctors who scare the patients informing them they are suffering from VD and offer very expensive investigations and treatment which sometimes run into thousands of rupees. Rarely patients take action about them due to the social stigma attached to these diseases. When in doubt, it is always safer to take another opinion from a reputed institution.

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German Foundation for International Development
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THE ORAL HEALTH ALLIANCE

THE BERLIN DECLARATION ON ORAL HEALTH AND ORAL HEALTH SERVICES IN DEPRIVED COMMUNITIES

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Proceedings of an International Workshop
held at
The German Foundation for International Development (DSE)
Conference Centre, Villa Borsig, Berlin
September 1992

FOREWORD

The oral health is improving in industrialized countries and deteriorating in developing countries. But within the more affluent countries improvements in oral health are not occurring in most deprived communities. Indeed, the oral health of children in many developing countries is worse than in developed countries and the former cannot afford the appropriate resources to deal with the diseases. In all countries the dental profession and governments place most emphasis on the curative and technological aspects of dentistry rather than promoting prevention and community programmes for oral health which are more effective at improving oral health and would have greater impact on the oral health of dentally deprived communities. The curative methods cannot be sustained at all levels of development.

Because the German Foundation for International Development (DSE) and the NGO "Dental Aid" (Zahnmedizinische Entwicklungshilfe - ZME) were aware of this problem they invited dentists, health workers, directors of health service projects and programmes and academics from twenty-eight countries to an international workshop on "Improving Oral Health and Oral Health Services for Deprived Communities". The workshop was held from September 14th to 19th at the DSE Conference Centre at Villa Borsig in Berlin, before the 80th FDI Annual World Congress.

The workshop provided an unique forum for people who work in community- based and community-oriented oral health programmes to

exchange experiences and discuss issues in primary oral health care at an international level.

The objectives were to:

- identify and discuss issues and actions relevant to community-oriented and community-based oral health programmes;
- identify problems and barriers in planning and implementing oral health programmes for deprived groups and discuss approaches to overcome them;
- formalize a network between the different groups and projects represented;
- inform the public and decision makers and raise awareness of these issues among the dentists by publishing the workshop proceedings.

During the workshop the need was expressed for better and continuing communication between oral health workers concerned about improving the oral health of all people including those who are deprived. Therefore a new group was constituted which was named the "Oral Health Alliance". The group formulated the Berlin Oral Health Declaration and recommended strategies based on the principles in the declaration. A newsletter and periodical critical reviews of current concepts in relation to oral health issues will be published and widely distributed.

THE BERLIN DECLARATION ON ORAL HEALTH 1992

BACKGROUND

When the Alma-Ata Declaration was formulated in 1978, there were two developments which were not foreseen or anticipated. These are the rapid global economic crisis of the 1980s and the AIDS pandemic with the current problems of its prevention and control. The economic, social as well as political crises in most developing countries have adversely affected health in terms of:

- provision of services
- access and coverage
- financing of services
- quality of services
- status of health

Deprived communities exist at the country level and deprived groups exist within countries. At the country level it was a description of the world's poorest nations. Many of these nations have low per capita incomes and increases in their populations of around 3% per annum. Their health and oral health services are grossly maldistributed. The dental caries level is relatively low in the rural poor - a DMFT at twelve years of age of 1.5 or less, but in the urban poor decay rates are higher than among the rich. Dental caries is common in poor countries and the severity is rising. The deprived have a high proportion of their decayed teeth unfilled or treated by extraction.

For deprived groups within countries the definition is more difficult but the following characteristics are common: poverty in comparison to the rest of the country (the poverty line has been set at 370 U.S. dollars annual income per capita in 1985 purchasing power parity dollars), marginalization either by geographical remoteness or because they have been dispossessed of their land/homes or because they feel that the existing services are not for them. This is often exacerbated by high levels of illiteracy and a lack of formal education. Frequently they do not feel a part of the larger group.

The direct problems of poverty are unemployment, low income, limited education and inadequate diet. This leads to overcrowding, poor housing, inadequate water and sanitation, lack of land to grow food, which in turn are related to infectious diseases, pollution, accidents, stress, alienation, instability and insecurity. The urban poor are at the

interface between underdevelopment and industrialization and their disease patterns reflect the problems of both. From the first they carry a heavy burden of infectious diseases and malnutrition, while from the second they suffer the typical spectrum of chronic and social diseases.

Good health of individuals, families and communities requires a decent environment built on a solid foundation of fairness, respect and equality. Until recently it was predicted that, by the year 2000, the number of the people in the world living below the poverty line would be reduced by 300 million. Now the World Bank suggests that the numbers in poverty have increased substantially - from 1,051 million to 1,133 from 1985 to 1990.

In any society, the principles of natural justice are based on equity, on balancing the scales between right and wrong, between what is fair and what is unfair. "Equity in health implies that ideally everyone should have a fair opportunity to attain their full potential and, more pragmatically, that no one should be disadvantaged from achieving this potential, if it can be avoided." (WHO, 1986, Social Justice and Equity in Health, WHO Europe, Copenhagen). By definition, inequity means injustice. Despite efforts in the past decade to make health systems more equitable, in the poorer countries and communities things are getting worse in terms of people's health and access to health care. The first priority is to get the international community and national governments to make a commitment, through positive actions, towards solving some of the causes of poverty. It is a human right to have access to the basic necessities for healthy living.

HEALTH IS A BASIC HUMAN RIGHT and oral health is a significant component of general health. Although oral diseases are not life-threatening, they are important public health problems. The reasons for their importance are their high prevalence, public demand, and their impact on individuals and society in terms of pain, discomfort, social and functional limitation and handicap and the effect on the quality of life. In addition the financial impact on the individual and community is very high and this is going to increase due to the infection control measures required for the prevention and control of hepatitis and HIV/AIDS.

Oral health has not been given the attention it deserves. As a result, people who are poor and living in difficult circumstances, whether in developing or industrialized countries, continue to be dentally neglected, a situation that has every likelihood of worsening as the global debt crisis and privatization of health services occur.

An important feature of oral diseases is that effective preventive methods which are simple and cheap are available, but in many cases these methods are not appropriately applied. The development of dental programmes incorporating effective simple and cheap preventive methods and which involve local people and build upon their strengths and capacities have a better chance of ultimately improving both services and community oral health. These community-based programmes are more likely to lead towards self-reliance and self-management of oral health programmes.

The purpose of this document is to provide guidelines to health planners for planning, implementing and evaluating relevant oral health projects and programmes. This may be accomplished when planners:

- move from a biomedical model to a social development model of health care and health care education;
- initiate ongoing monitoring and evaluation based upon the principles of community-based action research;
- involve community members in assessing needs and project/programme development. The intention is to reduce dependency, enhance personal autonomy and enable oral health and well being to occur;
- Prepare and support health workers to work with local people for health and equity in dentistry.

Oral health projects and programmes aimed at improving oral health and oral health services in deprived communities should be developed recognizing certain issues and principles. These are spelled out in the remainder of this declaration.

ISSUES TO BE CONSIDERED

It is important for health planners, administrators and all advocates to be aware of a number of issues that encompass the world of workers wanting to provide primary care and nurture oral health in dentally deprived communities.

Underdevelopment:

The recognition that many communities are dentally deprived has unfortunately produced a number of inappropriate responses. The worst is that programmes developed in one setting have been transferred without adaptation to a completely different one. The

results of such efforts have been to create or increase the state of dependency of deprived communities and to reinforce their sense of powerlessness and their lack of ownership of the programmes. The fragmentation, within many deprived communities, exacerbated by poverty, lack of formal education and high illiteracy levels, result in little participation by the people in such programmes. These problems will not be resolved by any attempt to put the blame for their existence on any one community, group or individual.

Ethical values:

Communities frequently resent unsolicited research activities being carried out in their midst. Similarly, appointment of external consultants to programmes by outside bodies with neither the agreement of the communities involved in programmes nor with clearly defined tasks, results in the disaffection and dissatisfaction of the community.

Health Promotion:

Most present programmes concentrate their efforts on the provision of curative services giving little attention to health promotion.

Goals and evaluation procedures:

Many programmes frequently lack clearly defined realistic goals developed in collaboration with the community. Seldom are appropriate socio-dental indicators applied. They take into account the measurement of well-being and the quality of care. This results in a lack of well defined appropriate and comprehensive process and impact measures, which take into account health promotion and community development activities.

Resources:

There is a misallocation of resources. The majority of the resources are used by the minority of the population who require the least care. Eighty percent of the resources are consumed in clinics which serve twenty percent of the population.

The training provided for many oral health care workers is frequently carried out in inappropriate settings. Consequently workers are ill-equipped to function where they are most needed and thus redress the imbalance in availability of appropriate care.

In many countries the migration of the population to the urban areas is resulting in large underserved peri-urban populations.

Programme organization and support.

The progress of many programmes is impeded because of frequent changes in administrations. Such situations are often made worse because the administrative infrastructures are weak. Often morale is low because of poor training and limited job satisfaction.

Little understanding of these problems and a reluctance within the dental profession to share and delegate responsibilities for oral health in the community further inhibits the development of appropriate programmes.

GUIDING PRINCIPLES FOR IMPROVING ORAL HEALTH

When developing a programme or project to improve oral health by working with people it is important to take into account each of the following principles.

The Role of Governments

Article V of the Declaration of the Alma Ata Conference (1978) states, that "Governments have a responsibility for the health of their people which can be fulfilled only by the provision of adequate health and social measures." A main social target should be the attainment of the world of a level of health that will permit all people to lead a socially and economically productive life. Primary health care is the key to attaining this target as part of development in the spirit of social justice.

Reducing Inequalities

Principles for action are:

1. Policies should focus on improving living and working conditions, adequate and safe housing, access to high quality food, control of pollution, safe water supplies, employment policy, welfare system.
2. Policies need to be directed towards enabling people to adopt healthier lifestyles. Distribution of cheap and nutritious food, control of advertising of health damaging products, provision of clear information, access to leisure and exercise facilities.
3. Policies require a genuine commitment to decentralizing power and decision making, encouraging people to participate in every stage of policy planning process
4. Health care based on principle of making high quality health care accessible to all. Resource allocation in relation to social and health needs, geographical distribution of services linked to measures of need and access, focus on acceptability of care from disadvantaged groups.

Underdevelopment

Oral health development is not achieved through the unmodified transfer of skills or programmes, personnel or equipment to deprived communities. Adaptation rather than uncritical adoption should be the rule.

Dependency

Dependency is disabling. Although it is recognized that most projects can not be completely self-financing and require state or institutional support, they should be deliberately developed and implemented in ways that ensure continuation by the communities in which they are located.

Empowerment

Communities must be enabled to obtain and contribute resources to sustain, self-manage, and satisfy their programme objectives and needs.

Community Involvement

Oral health decisions should always be made collectively with local people who best know their own problems, needs and capacities, so that control of dental projects and programmes rests with the community being served. The right of all communities deprived of oral health care, to organize services and training programmes should be acknowledged.

Partnerships

Projects should be developed and implemented mutually (by institutions and persons) on the basis of honesty, respect and reciprocity in that partners share and learn from each other. Partners must declare their assumptions and expectations, and decide how to work together to resolve problems and make decisions.

Consultants and co-operating agencies have an obligation to be responsive and responsible commitment to project objectives is essential.

Assessing needs

The assessment of needs has to be based on collaborative efforts between users and providers. This should lead to the agreement of goals and targets, that reflect people's needs and to the implementation of services and evaluations that are based on these set goals.

Epidemiology is a fundamental tool in the development and evaluation of health plans and programmes. It is necessary to develop indicators different from those normally used. They should measure social, economic and health impacts. Socio-dental indicators are more relevant measures of needs and should reflect pain, discomfort, function and aesthetics as well as clinical indicators of dental health such as caries, bleeding gums and pocketing, number and position of teeth. Other impact measures include loss of sleep, work loss and opportunity costs.

Priorities

Priorities should not be developed solely on the basis of the demand for treatment. Health promotion can alter a community's perception of the problems and hence priorities. Priorities should be established through a partnership between the community and the professional advocates for oral health.

Goals

The community should be involved in setting goals that are stated in terms of oral health, oral disease, health promotion, equity, training and personnel and health service.

Oral Health Promotion

Oral Health Promotion should have the highest priority and follow the principles as defined in the Ottawa Charter for Health Promotion (1986). Health Promotion means building healthy public policy, creating supportive environments, strengthening community action, developing coping skills and re-orienting dental services. Health promotion policy must take into consideration: the uneven distribution of health and disease, the uneven distribution of health hazards in the physical and social environment and of personal behavioural risk factors and

opportunities to adopt a healthier personal lifestyle as well as the uneven distribution and quality of health care.

Preventive Strategies

Authorities should recognize that the preventive approach is an essential and inescapable recommendation.

Governments should apply mass preventive measures. Oral health strategies should be integrated with general preventive approaches within an overall context of health which lead to improvements in the quantity and quality of life.

The preventive measures should be simple and effective and not contradict each other or confuse the community.

Evaluation

Evaluation should not be seen as a mere technical activity but more as an educational learning process in order to improve social conditions and human life. It should therefore shift from being autocratic to a more democratic participatory exercise. The community should be involved in the formulation and assessment of the impacts of the programme. Process measures should not only include the number of procedures performed but health promotion and community development activities. The results should be discussed and acted upon.

Treatment strategies

The services should be based on the development of local human resources. Community needs should be defined and mechanisms established for regionalising services. This should be done on the basis of levels of care which requires referral and redefines the roles of different health personnel.

It is also important to ensure that proposed strategies are compatible with scientific knowledge and technological advances.

Ethics

All activities performed should comply with ethical standards accepted and outlined in the Helsinki Declaration and by a local ethical committee.

Experts/Consultants

Local people are experts on community life, functions, and potential. Outside consultants who have a particular expertise should only be selected after full consultation and at the request of the host community.

The experience and qualifications of the consultants should be vetted to ensure that they are suitable for the task on hand.

In all cases local/regional expertise should be given priority before considering consultants from further afield.

Once the invitation is agreed, the host country and community is to ensure that adequate facilities are available to enable that the tasks agreed upon are carried out. The consultant has an obligation to carry out these tasks and submit a report to the local counterparts before submitting it to the sponsoring agency.

Resources

Resources (human, facilities, finance) should be shifted from central offices and hospitals to the communities.

Communities are a very important resource for improving health. They should be mobilized to play a more active role in promoting health. These roles include needs assessment, design of appropriate health programmes, decision making and local generation of resources to promote self-sufficiency.

Whenever possible, appropriate technology in terms of personnel, equipment and materials should be used. There are many simple clinical methods and ways of organizing the provision of services which are easily sustainable and can be made readily available through the use of basic instruments and local assembly or production. These should be chosen in preference to more sophisticated technologies.

Personnel Preparation

Preparation of oral health personnel should emphasize both education and training. The curricula of all oral health workers should reflect the major shift from the medical to the social model of health which implies the incorporation of social and behavioural sciences.

Personnel preparation should be part of a process which ensures career advancement and flexibility. In considering the curricula for oral health workers the civil service grade of the worker on qualification should be used in a positive rather than negative way to ensure more community orientation as well as adequate remuneration and not merely to prolong the length of the training unnecessarily to qualify for a higher grade.

Planning the numbers and types of oral health personnel should not rely purely on a dental perspective. Training of personnel should occur when the context can absorb and support the graduates. Oral health personnel should be trained alongside other health personnel.

Oral health personnel trained at the community's expense should be encouraged to work in the community. Status and salaries, training, promotion and position should be equalized to those of general health personnel.

Rights of Health Workers

Professional bodies must ensure that the human rights of oral health personnel are guaranteed in accordance with the UN Charter of Human Rights.

The rights of health workers should be protected so that they can provide services without sexual, political and professional harassment.

Caring for deprived communities should not be considered a subversive activity but rather governments are responsible for the human rights of oral health personnel.

Health workers have a right to be informed about the possible hazards in their working environment, for example radiation, mercury intoxication and cross-infection.

Health workers have a right to continuing education. The employer should actively encourage and assist health workers to acquire appropriate continuing education and training.

Scientific Basis of Oral Health Strategies

Services and oral health promotion strategies should be modified on the basis of scientific knowledge regarding the effectiveness, efficiency and cost-benefit of common interventions. This implies a constant review of the scientific basis for health education methods and messages, training and education of health workers, life history of oral diseases, oral pathology, preventive and treatment strategies, infection control, research and research methods, social science in oral health and community based programmes.

Research

Research can be beneficial and enabling if it is applied and focuses on real community problems , includes local people in both its development and implementation (participatory and educational), and leads to immediate feed-back and actions for oral health. It should contribute to the kind of longer term sociopolitical changes that are necessary.

RECOMMENDED STRATEGIES FOR DEVELOPING ORAL HEALTH CARE PROGRAMMES IN DEPRIVED COMMUNITIES

I INTRODUCTION

Although there are sufficient dentists in the world today, the majority of people do not have access to adequate, affordable and acceptable oral health services. The following strategies should redress the imbalance in oral health between the deprived and other citizens.

Because of the wide variation in the circumstances of different communities and countries, detailed universal prescription cannot be made. However general guidelines can be outlined.

Two principles of the primary health care approach have to be considered in almost all programmes if lasting solutions to problems are to be found. These are community participation and multisectoral co-operation and integration. An important element in achieving equity in oral health and oral health services is the success of the multisectoral approach in securing community development. The responsibility of governments and co-operating and partnership agencies in this respect must be underlined.

Sothat efforts do not become mere palliatives reinforcing the unjust structures that perpetuate poor health services, health should be viewed as inter-related with the problems of unemployment, high prices and inadequate housing. Oral health care, to be liberating in action for the poor and deprived, should take into consideration the root causes of ill-health. The root causes of the health problems of the poor are poverty and powerlessness.

II STRATEGIES FOR ORAL DISEASE PREVENTION

Prevention should be based on the principles of Health Promotion: re-orienting oral health services, creating supportive environment, building healthy public policy, supporting community action, developing coping skills.

In addition to the general principles outlined in the Berlin Oral Health Declaration the following points should be considered in all programmes.

- The application of preventive measures depends very much on the socio-economic situation of the country and therefore a flexible approach to preventive programmes should be taken which will permit adaptation to suit existing circumstances. Such adaptation must be undertaken by utilising scientifically tested methods of health education and prevention;

- In most deprived communities, countries and population groups, other health problems are much more important than oral health problems;

- Appropriate technology should be used (eg. chewing sticks) and people should be enabled to make their own toothbrushes/toothpaste and self-care should be encouraged. Families should be enabled to carry out their own set of preventive activities;

- Some combinations of different fluoride regimens do not always lead to a clinically significantly better cumulative effect.

Specific Strategies include:

1. Fluoridation:
The promotion of fluoridated toothpaste is the most appropriate way for many countries to reduce the levels of caries. The highest priority should be to make cheap fluoridated toothpaste widely available. In furtherance of this strategy therefore:

- International companies should be persuaded to sell cheap fluoride toothpaste in bulk;

- Health education messages about tooth cleaning for caries reduction should be given less emphasis until people can get fluoride toothpaste on a regular basis;

- International co-operation agencies should be encouraged to distribute fluoride toothpaste.

2. Health education:
Health education should be put in a health promotional context. Thus it should be combined with the provision of curative services for teachers,

families and students and carried out in a supportive environment where there is clean water and healthy food in stores near to schools.

3. Integration of health promotional activities:
Oral health messages and activities should be included in general health messages and actions. Integration and a common risk factor approach outlined below should be the cornerstones of health promotional activities. The fundamental concepts are:

- Tackling causes common to a number of chronic diseases;
- Including oral hygiene education as part of general hygiene;
- Developing population rather than high-risk strategies.

The approach can be developed because of risk factors common to a number of chronic diseases, including dental caries, periodontal disease and oral cancer. Diets which lead to caries also contribute to obesity, coronary heart disease and diabetes. Periodontal diseases and oral cancer are related to smoking. Smoking causes cancers elsewhere in the body and respiratory diseases. Integrating activities with groups concerned about those chronic diseases should be more effective than disease specific activities

Oral cleanliness should be seen as part of grooming and body cleanliness. It is logical that instead of separating oral cleanliness education from general hygiene they should be combined. These programmes should emphasize lay competence, be supportive and non-mystifying, and should not blame the victim.

A population strategy decreases the overall level of disease in the population and does not concentrate entirely on high risk individuals. It tackles the underlying causes of the distribution of the disease rather than the causes of the incidence of disease in individuals.

III ORAL HEALTH SERVICES

A basic necessity is a budgeted oral health plan which takes into account the health situation of the community including an inventory of its own resources and is developed with the participation of the community. A national plan can provide a useful framework for local groups to begin planning their own service. In this respect the advocacy or facilitator role of the dental public health professional is important in providing expert information and advice.

In planning oral health services all possible resources should be considered, including the role of independent practitioners, which should be complimentary to that of government service staff.

The six A's should always be considered in order to improve health services. These are availability, accessibility, accountability, affordability, accommodation and acceptability.

The problem of unequal distribution of oral health personnel within the health service exists in nearly all countries. There have been several approaches to motivate dentists to work in rural and deprived areas. All have failed although training, salary and conditions were favourable. Greater success has occurred with auxiliary personnel. Training more auxiliary personnel may be an important way of increasing coverage. But this should not be allowed to lead to the creation of a two-tier service in respect of appropriateness and quality of care. The important role that such auxiliary personnel can play needs therefore to be supported strongly by governments and professional bodies.

The development of a referral system that is sensitive to the needs and difficulties of those using it should be part of the planning process. The importance of such a referral system consists not only in providing for the necessary treatment for the more complex problems but also in legitimizing and supporting the role of the primary care level and the workers providing it. This should be recognized. The referral system can also act as a barrier to the development of a two-tier system of health care when seen in this way. It is the responsibility of governments to ensure a health service which is of equal status and quality for all citizens.

How can the inclination of professionals to perform complex clinical procedures be reconciled with the frequent need for simple care without causing frustration and sometimes a 'braindrain'? Job satisfaction could be achieved by:

- Balancing the number of dentists (higher technical workers) in training with needs. This may well entail the training of more auxiliaries and many fewer dentists;
- Providing a range of alternative activities which are demonstrated by role models as being both valid and legitimate;
- Encouraging dental workers to integrate their work with that of the health team. This implies the socialization or popularization of the practice of dentistry as is occurring in a number of community-based projects;
- Utilizing selection procedures for dental students which emphasize the ability to engage in non-clinical activities as an important part of their future work;
- Developing career pathways for all oral health personnel including general practitioners.

Job functions need to be clearly stated. For example, suggested functions and activities of a community dentist are:

Functions	Activities
Manager	Leader of primary oral health care team
	Monitoring and controlling the oral health sub-system
	Organise/co-ordinate preventive, treatment, and referral services
	Help data analysis research and information dissemination
	Help plan, supervise and evaluate the oral activities
Agent of socio-economic development	Development of community participation in oral health
	Liaison with the public, politicians and other sectors
	Participate in community meetings and development activities
	Participate in intersectoral projects e.g., food and water
	Advocacy of better oral health
	Critical analysis of intersectoral plans for oral health implications
	Influence politicians to make healthful decisions
	Support appropriate development e.g., local food production
Dental officer	Complex treatment of patients
	Promotion of oral health at community, family and individual levels
Educator	
	Continuing education of colleagues
	Training of lower level oral health workers
	Oral health education of families and communities

It is essential to develop secure funding for the budget. This can come from within the community and/or outside it.

Economic constraints will determine the coverage achieved by health services. Without economic development extensive coverage with oral health care will not be possible. This leads to rationing where decisions have to be made concerning 'strategic teeth' that should receive priority.

Developing countries may need support to strengthen their health services, but they do not want or need conditional help. They do not need operating dentists but rather help in strengthening training programmes and with clinical and educational materials.

Donor agencies may play unfortunate roles in funding postgraduate studies in areas which are of no relevance to existing national situations. Increased responsiveness, sensitivity and responsibility to local situations in this respect is called for.

Comprehensive planning procedures involve intersectoral co-operation and integration, frequently involving ministries of health, education, finance and social-welfare, universities, schools for oral therapists, oral

associations, professional interest groups and international bodies. The planning group must involve all these structures as appropriate.

Equally important however, the planning activity must involve finding out the needs of the community in relation to oral health. Hence the planning of personnel training for oral health cannot be carried out without being built up from a comprehensive community diagnosis. As such it is best if the planning group is as broadly representative as possible and that it should be able to call on the professional skills of a range of specialist advisers particularly those in the social sciences.

There should be support and encouragement for local initiatives in developing oral health services, including assistance with the development of appropriate technology in the form of personnel, equipment and materials.

The role of international bodies in calling the attention of the oral profession to their responsibility in providing for the oral health needs of the whole population rather than selected groups needs to be emphasized.

IV COMMUNITY INVOLVEMENT

Genuine partnership between oral health personnel and the community depends upon mutual respect and trust. To build such a partnership is often difficult and may require a variety of contacts with different groups of the community as there are likely to be different levels of participation and interests. It is clear that it will be necessary to approach the community through local leaders and involve them in the planning, implementation, and evaluation of the community-oriented service as a first step. Later this should lead to the development of a fully community-based service, which is perceived by the community as their service.

There may be existing health workers in the community. It is important to include them and to define and develop their advocacy and facilitator roles within the group.

Neither the scientific basis of community based oral health programmes nor the capacity of the community to participate in such programmes on a scientific basis should be undervalued.

For many deprived communities health is defined in terms like caring, sharing, belonging and being able to look forward to tomorrow. A

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22nd February 1994

Dr Thelma Narayan
No 376 "Srinivasa Nilaya"
Jakkasandra, 1st Main
1st Block, Koramangala
Bangalore 560 034

Dear Dr Narayan,

I enclose a copy of the Berlin Declaration as requested.

Your pamphlets and newsletter are interesting. There are no easily transferable materials I can send you. Please come and see me if you come back to London.

Yours sincerely,

Aubrey Sheiham

Encl.: Berlin Declaration

To SPT / Dr CMF / Dr VB / RN for info. I would Health Action be interested to feature a note about the Conference & Declaration?
JN 14/3

NG Could we start an oral health file in the above unit.
"ORAL HEALTH" file opened. See "Diseases & Diagnosis"
NG 15/4/94.

1604/3/3/94 HMV

Deutsche Stiftung für internationale Entwicklung

German Foundation for International Development

Fondation Allemande pour le Développement International

Fundación Alemana para el Desarrollo Internacional

THE ORAL HEALTH ALLIANCE

THE BERLIN DECLARATION ON ORAL HEALTH AND ORAL HEALTH SERVICES IN DEPRIVED COMMUNITIES

DSE

THE ORAL HEALTH ALLIANCE

THE BERLIN DECLARATION ON ORAL HEALTH AND ORAL HEALTH SERVICES IN DEPRIVED COMMUNITIES

Proceedings of an International Workshop
held at
The German Foundation for International Development (DSE)
Conference Centre, Villa Borsig, Berlin.
September 1992

FOREWORD

The oral health is improving in industrialized countries and deteriorating in developing countries. But within the more affluent countries improvements in oral health are not occurring in most deprived communities. Indeed, the oral health of children in many developing countries is worse than in developed countries and the former cannot afford the appropriate resources to deal with the diseases. In all countries the dental profession and governments place most emphasis on the curative and technological aspects of dentistry rather than promoting prevention and community programmes for oral health which are more effective at improving oral health and would have greater impact on the oral health of dentally deprived communities. The curative methods cannot be sustained at all levels of development.

Because the German Foundation for International Development (DSE) and the NGO "Dental Aid" (Zahnmedizinische Entwicklungshilfe - ZME) were aware of this problem they invited dentists, health workers, directors of health service projects and programmes and academics from twenty-eight countries to an international workshop on "Improving Oral Health and Oral Health Services for Deprived Communities". The workshop was held from September 14th to 19th at the DSE Conference Centre at Villa Borsig in Berlin, before the 80th FDI Annual World Congress.

The workshop provided an unique forum for people who work in community- based and community-oriented oral health programmes to

exchange experiences and discuss issues in primary oral health care at an international level.

The objectives were to:

- identify and discuss issues and actions relevant to community-oriented and community-based oral health programmes;
- identify problems and barriers in planning and implementing oral health programmes for deprived groups and discuss approaches to overcome them;
- formalize a network between the different groups and projects represented;
- inform the public and decision makers and raise awareness of these issues among the dentists by publishing the workshop proceedings.

During the workshop the need was expressed for better and continuing communication between oral health workers concerned about improving the oral health of all people including those who are deprived. Therefore a new group was constituted which was named the "Oral Health Alliance". The group formulated the Berlin Oral Health Declaration and recommended strategies based on the principles in the declaration. A newsletter and periodical critical reviews of current concepts in relation to oral health issues will be published and widely distributed.

THE BERLIN DECLARATION ON ORAL HEALTH 1992

BACKGROUND

When the Alma-Ata Declaration was formulated in 1978, there were two developments which were not foreseen or anticipated. These are the rapid global economic crisis of the 1980s and the AIDS pandemic with the current problems of its prevention and control. The economic, social as well as political crises in most developing countries have adversely affected health in terms of:

- provision of services
- access and coverage
- financing of services
- quality of services
- status of health

Deprived communities exist at the country level and deprived groups exist within countries. At the country level it was a description of the world's poorest nations. Many of these nations have low per capita incomes and increases in their populations of around 3% per annum. Their health and oral health services are grossly maldistributed. The dental caries level is relatively low in the rural poor - a DMFT at twelve years of age of 1.5 or less, but in the urban poor decay rates are higher than among the rich. Dental caries is common in poor countries and the severity is rising. The deprived have a high proportion of their decayed teeth unfilled or treated by extraction.

For deprived groups within countries the definition is more difficult but the following characteristics are common: poverty in comparison to the rest of the country (the poverty line has been set at 370 U.S. dollars annual income per capita in 1985 purchasing power parity dollars), marginalization either by geographical remoteness or because they have been dispossessed of their land/homes or because they feel that the existing services are not for them. This is often exacerbated by high levels of illiteracy and a lack of formal education. Frequently they do not feel a part of the larger group.

The direct problems of poverty are unemployment, low income, limited education and inadequate diet. This leads to overcrowding, poor housing, inadequate water and sanitation, lack of land to grow food, which in turn are related to infectious diseases, pollution, accidents, stress, alienation, instability and insecurity. The urban poor are at the

interface between underdevelopment and industrialization and their disease patterns reflect the problems of both. From the first they carry a heavy burden of infectious diseases and malnutrition, while from the second they suffer the typical spectrum of chronic and social diseases.

Good health of individuals, families and communities requires a decent environment built on a solid foundation of fairness, respect and equality. Until recently it was predicted that, by the year 2000, the number of the people in the world living below the poverty line would be reduced by 300 million. Now the World Bank suggests that the numbers in poverty have increased substantially - from 1,051 million to 1,133 from 1985 to 1990.

In any society, the principles of natural justice are based on equity, on balancing the scales between right and wrong, between what is fair and what is unfair. "Equity in health implies that ideally everyone should have a fair opportunity to attain their full potential and, more pragmatically, that no one should be disadvantaged from achieving this potential, if it can be avoided." (WHO, 1986, Social Justice and Equity in Health, WHO Europe, Copenhagen). By definition, inequity means injustice. Despite efforts in the past decade to make health systems more equitable, in the poorer countries and communities things are getting worse in terms of people's health and access to health care. The first priority is to get the international community and national governments to make a commitment, through positive actions, towards solving some of the causes of poverty. It is a human right to have access to the basic necessities for healthy living.

HEALTH IS A BASIC HUMAN RIGHT and oral health is a significant component of general health. Although oral diseases are not life-threatening, they are important public health problems. The reasons for their importance are their high prevalence, public demand, and their impact on individuals and society in terms of pain, discomfort, social and functional limitation and handicap and the effect on the quality of life. In addition the financial impact on the individual and community is very high and this is going to increase due to the infection control measures required for the prevention and control of hepatitis and HIV/AIDS.

Oral health has not been given the attention it deserves. As a result, people who are poor and living in difficult circumstances, whether in developing or industrialized countries, continue to be dentally neglected, a situation that has every likelihood of worsening as the global debt crisis and privatization of health services occur.

An important feature of oral diseases is that effective preventive methods which are simple and cheap are available, but in many cases these methods are not appropriately applied. The development of dental programmes incorporating effective simple and cheap preventive methods and which involve local people and build upon their strengths and capacities have a better chance of ultimately improving both services and community oral health. These community-based programmes are more likely to lead towards self-reliance and self-management of oral health programmes.

The purpose of this document is to provide guidelines to health planners for planning, implementing and evaluating relevant oral health projects and programmes. This may be accomplished when planners:

- move from a biomedical model to a social development model of health care and health care education;
- initiate ongoing monitoring and evaluation based upon the principles of community-based action research;
- involve community members in assessing needs and project/programme development. The intention is to reduce dependency, enhance personal autonomy and enable oral health and well being to occur;
- Prepare and support health workers to work with local people for health and equity in dentistry.

Oral health projects and programmes aimed at improving oral health and oral health services in deprived communities should be developed recognizing certain issues and principles. These are spelled out in the remainder of this declaration.

ISSUES TO BE CONSIDERED

It is important for health planners, administrators and all advocates to be aware of a number of issues that encompass the world of workers wanting to provide primary care and nurture oral health in dentally deprived communities.

Underdevelopment:

The recognition that many communities are dentally deprived has unfortunately produced a number of inappropriate responses. The worst is that programmes developed in one setting have been transferred without adaptation to a completely different one. The

results of such efforts have been to create or increase the state of dependency of deprived communities and to reinforce their sense of powerlessness and their lack of ownership of the programmes. The fragmentation, within many deprived communities, exacerbated by poverty, lack of formal education and high illiteracy levels, result in little participation by the people in such programmes. These problems will not be resolved by any attempt to put the blame for their existence on any one community, group or individual.

Ethical values:

Communities frequently resent unsolicited research activities being carried out in their midst. Similarly, appointment of external consultants to programmes by outside bodies with neither the agreement of the communities involved in programmes nor with clearly defined tasks, results in the disaffection and dissatisfaction of the community.

Health Promotion:

Most present programmes concentrate their efforts on the provision of curative services giving little attention to health promotion.

Goals and evaluation procedures:

Many programmes frequently lack clearly defined realistic goals developed in collaboration with the community. Seldom are appropriate socio-dental indicators applied. They take into account the measurement of well-being and the quality of care. This results in a lack of well defined appropriate and comprehensive process and impact measures, which take into account health promotion and community development activities.

Resources:

There is a misallocation of resources. The majority of the resources are used by the minority of the population who require the least care. Eighty percent of the resources are consumed in clinics which serve twenty percent of the population.

The training provided for many oral health care workers is frequently carried out in inappropriate settings. Consequently workers are ill-equipped to function where they are most needed and thus redress the imbalance in availability of appropriate care.

In many countries the migration of the population to the urban areas is resulting in large underserved peri-urban populations.

Programme organization and support:

The progress of many programmes is impeded because of frequent changes in administrations. Such situations are often made worse because the administrative infrastructures are weak. Often morale is low because of poor training and limited job satisfaction.

Little understanding of these problems and a reluctance within the dental profession to share and delegate responsibilities for oral health in the community further inhibits the development of appropriate programmes.

GUIDING PRINCIPLES FOR IMPROVING ORAL HEALTH

When developing a programme or project to improve oral health by working with people it is important to take into account each of the following principles.

The Role of Governments

Article V of the Declaration of the Alma Ata Conference (1978) states, that "Governments have a responsibility for the health of their people which can be fulfilled only by the provision of adequate health and social measures." A main social target should be the attainment of the world of a level of health that will permit all people to lead a socially and economically productive life. Primary health care is the key to attaining this target as part of development in the spirit of social justice.

Reducing Inequalities

Principles for action are:

1. Policies should focus on improving living and working conditions, adequate and safe housing, access to high quality food, control of pollution, safe water supplies, employment policy, welfare system.
2. Policies need to be directed towards enabling people to adopt healthier lifestyles. Distribution of cheap and nutritious food, control of advertising of health damaging products, provision of clear information, access to leisure and exercise facilities.
3. Policies require a genuine commitment to decentralizing power and decision making, encouraging people to participate in every stage of policy planning process
4. Health care based on principle of making high quality health care accessible to all. Resource allocation in relation to social and health needs, geographical distribution of services linked to measures of need and access, focus on acceptability of care from disadvantaged groups.

Underdevelopment

Oral health development is not achieved through the unmodified transfer of skills or programmes, personnel or equipment to deprived communities. Adaptation rather than uncritical adoption should be the rule.

Dependency

Dependency is disabling. Although it is recognized that most projects can not be completely self-financing and require state or institutional support, they should be deliberately developed and implemented in ways that ensure continuation by the communities in which they are located.

Empowerment

Communities must be enabled to obtain and contribute resources to sustain, self-manage, and satisfy their programme objectives and needs.

Community Involvement

Oral health decisions should always be made collectively with local people who best know their own problems, needs and capacities, so that control of dental projects and programmes rests with the community being served. The right of all communities deprived of oral health care, to organize services and training programmes should be acknowledged.

Partnerships

Projects should be developed and implemented mutually (by institutions and persons) on the basis of honesty, respect and reciprocity in that partners share and learn from each other. Partners must declare their assumptions and expectations, and decide how to work together to resolve problems and make decisions.

Consultants and co-operating agencies have an obligation to be responsive and responsible commitment to project objectives is essential.

Assessing needs

The assessment of needs has to be based on collaborative efforts between users and providers. This should lead to the agreement of goals and targets, that reflect people's needs and to the implementation of services and evaluations that are based on these set goals.

Epidemiology is a fundamental tool in the development and evaluation of health plans and programmes. It is necessary to develop indicators different from those normally used. They should measure social, economic and health impacts. Socio-dental indicators are more relevant measures of needs and should reflect pain, discomfort, function and aesthetics as well as clinical indicators of dental health such as caries, bleeding gums and pocketing, number and position of teeth. Other impact measures include loss of sleep, work loss and opportunity costs.

Priorities

Priorities should not be developed solely on the basis of the demand for treatment. Health promotion can alter a community's perception of the problems and hence priorities. Priorities should be established through a partnership between the community and the professional advocates for oral health.

Goals

The community should be involved in setting goals that are stated in terms of oral health, oral disease, health promotion, equity, training and personnel and health service.

Oral Health Promotion

Oral Health Promotion should have the highest priority and follow the principles as defined in the Ottawa Charter for Health Promotion (1986). Health Promotion means building healthy public policy, creating supportive environments, strengthening community action, developing coping skills and re-orienting dental services. Health promotion policy must take into consideration: the uneven distribution of health and disease, the uneven distribution of health hazards in the physical and social environment and of personal behavioural risk factors and

opportunities to adopt a healthier personal lifestyle as well as the uneven distribution and quality of health care.

Preventive Strategies

Authorities should recognize that the preventive approach is an essential and inescapable recommendation.

Governments should apply mass preventive measures. Oral health strategies should be integrated with general preventive approaches within an overall context of health which lead to improvements in the quantity and quality of life.

The preventive measures should be simple and effective and not contradict each other or confuse the community.

Evaluation

Evaluation should not be seen as a mere technical activity but more as an educational learning process in order to improve social conditions and human life. It should therefore shift from being autocratic to a more democratic participatory exercise. The community should be involved in the formulation and assessment of the impacts of the programme. Process measures should not only include the number of procedures performed but health promotion and community development activities. The results should be discussed and acted upon.

Treatment strategies

The services should be based on the development of local human resources. Community needs should be defined and mechanisms established for regionalising services. This should be done on the basis of levels of care which requires referral and redefines the roles of different health personnel.

It is also important to ensure that proposed strategies are compatible with scientific knowledge and technological advances.

Ethics

All activities performed should comply with ethical standards accepted and outlined in the Helsinki Declaration and by a local ethical committee.

Experts/Consultants

Local people are experts on community life, functions, and potential. Outside consultants who have a particular expertise should only be selected after full consultation and at the request of the host community.

The experience and qualifications of the consultants should be vetted to ensure that they are suitable for the task on hand.

In all cases local/regional expertise should be given priority before considering consultants from further afield.

Once the invitation is agreed, the host country and community is to ensure that adequate facilities are available to enable that the tasks agreed upon are carried out. The consultant has an obligation to carry out these tasks and submit a report to the local counterparts before submitting it to the sponsoring agency.

Resources

Resources (human, facilities, finance) should be shifted from central offices and hospitals to the communities.

Communities are a very important resource for improving health. They should be mobilized to play a more active role in promoting health. These roles include needs assessment, design of appropriate health programmes, decision making and local generation of resources to promote self-sufficiency.

Whenever possible, appropriate technology in terms of personnel, equipment and materials should be used. There are many simple clinical methods and ways of organizing the provision of services which are easily sustainable and can be made readily available through the use of basic instruments and local assembly or production. These should be chosen in preference to more sophisticated technologies.

Personnel Preparation

Preparation of oral health personnel should emphasize both education and training. The curricula of all oral health workers should reflect the major shift from the medical to the social model of health which implies the incorporation of social and behavioural sciences.

Personnel preparation should be part of a process which ensures career advancement and flexibility. In considering the curricula for oral health workers the civil service grade of the worker on qualification should be used in a positive rather than negative way to ensure more community orientation as well as adequate remuneration and not merely to prolong the length of the training unnecessarily to qualify for a higher grade.

Planning the numbers and types of oral health personnel should not rely purely on a dental perspective. Training of personnel should occur when the context can absorb and support the graduates. Oral health personnel should be trained alongside other health personnel.

Oral health personnel trained at the community's expense should be encouraged to work in the community. Status and salaries, training, promotion and position should be equalized to those of general health personnel.

Rights of Health Workers

Professional bodies must ensure that the human rights of oral health personnel are guaranteed in accordance with the UN Charter of Human Rights.

The rights of health workers should be protected so that they can provide services without sexual, political and professional harassment.

Caring for deprived communities should not be considered a subversive activity but rather governments are responsible for the human rights of oral health personnel.

Health workers have a right to be informed about the possible hazards in their working environment, for example radiation, mercury intoxication and cross-infection.

Health workers have a right to continuing education. The employer should actively encourage and assist health workers to acquire appropriate continuing education and training.

Scientific Basis of Oral Health Strategies

Services and oral health promotion strategies should be modified on the basis of scientific knowledge regarding the effectiveness, efficiency and cost-benefit of common interventions. This implies a constant review of the scientific basis for health education methods and messages, training and education of health workers, life history of oral diseases, oral pathology, preventive and treatment strategies, infection control, research and research methods, social science in oral health and community based programmes.

Research

Research can be beneficial and enabling if it is applied and focuses on real community problems , includes local people in both its development and implementation (participatory and educational), and leads to immediate feed-back and actions for oral health. It should contribute to the kind of longer term sociopolitical changes that are necessary.

RECOMMENDED STRATEGIES FOR DEVELOPING ORAL HEALTH CARE PROGRAMMES IN DEPRIVED COMMUNITIES

I INTRODUCTION

Although there are sufficient dentists in the world today, the majority of people do not have access to adequate, affordable and acceptable oral health services. The following strategies should redress the imbalance in oral health between the deprived and other citizens.

Because of the wide variation in the circumstances of different communities and countries, detailed universal prescription cannot be made. However general guidelines can be outlined.

Two principles of the primary health care approach have to be considered in almost all programmes if lasting solutions to problems are to be found. These are community participation and multisectoral co-operation and integration. An important element in achieving equity in oral health and oral health services is the success of the multisectoral approach in securing community development. The responsibility of governments and co-operating and partnership agencies in this respect must be underlined.

Sothat efforts do not become mere palliatives reinforcing the unjust structures that perpetuate poor health services, health should be viewed as inter-related with the problems of unemployment, high prices and inadequate housing. Oral health care, to be liberating in action for the poor and deprived, should take into consideration the root causes of ill-health. The root causes of the health problems of the poor are poverty and powerlessness.

II STRATEGIES FOR ORAL DISEASE PREVENTION

Prevention should be based on the principles of Health Promotion: re-orienting oral health services, creating supportive environment, building healthy public policy, supporting community action, developing coping skills.

In addition to the general principles outlined in the Berlin Oral Health Declaration the following points should be considered in all programmes.

- The application of preventive measures depends very much on the socio-economic situation of the country and therefore a flexible approach to preventive programmes should be taken which will permit adaptation to suit existing circumstances. Such adaptation must be undertaken by utilising scientifically tested methods of health education and prevention;

- In most deprived communities, countries and population groups, other health problems are much more important than oral health problems;

- Appropriate technology should be used (eg. chewing sticks) and people should be enabled to make their own toothbrushes/toothpaste and self-care should be encouraged. Families should be enabled to carry out their own set of preventive activities;

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Oral health messages and activities should be included in general health messages and actions. Integration and a common risk factor approach outlined below should be the cornerstones of health promotional activities. The fundamental concepts are:

- Tackling causes common to a number of chronic diseases;
- Including oral hygiene education as part of general hygiene;
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The approach can be developed because of risk factors common to a number of chronic diseases, including dental caries, periodontal disease and oral cancer. Diets which lead to caries also contribute to obesity, coronary heart disease and diabetes. Periodontal diseases and oral cancer are related to smoking. Smoking causes cancers elsewhere in the body and respiratory diseases. Integrating activities with groups concerned about those chronic diseases should be more effective than disease specific activities

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In planning oral health services all possible resources should be considered, including the role of independent practitioners, which should be complimentary to that of government service staff.

The six A's should always be considered in order to improve health services. These are availability, accessibility, accountability, affordability, accommodation and acceptability.

The problem of unequal distribution of oral health personnel within the health service exists in nearly all countries. There have been several approaches to motivate dentists to work in rural and deprived areas. All have failed although training, salary and conditions were favourable. Greater success has occurred with auxiliary personnel. Training more auxiliary personnel may be an important way of increasing coverage. But this should not be allowed to lead to the creation of a two-tier service in respect of appropriateness and quality of care. The important role that such auxiliary personnel can play needs therefore to be supported strongly by governments and professional bodies.

The development of a referral system that is sensitive to the needs and difficulties of those using it should be part of the planning process. The importance of such a referral system consists not only in providing for the necessary treatment for the more complex problems but also in legitimizing and supporting the role of the primary care level and the workers providing it. This should be recognized. The referral system can also act as a barrier to the development of a two-tier system of health care when seen in this way. It is the responsibility of governments to ensure a health service which is of equal status and quality for all citizens.

How can the inclination of professionals to perform complex clinical procedures be reconciled with the frequent need for simple care without causing frustration and sometimes a 'braindrain'? Job satisfaction could be achieved by:

- Balancing the number of dentists (higher technical workers) in training with needs. This may well entail the training of more auxiliaries and many fewer dentists;
- Providing a range of alternative activities which are demonstrated by role models as being both valid and legitimate;
- Encouraging dental workers to integrate their work with that of the health team. This implies the socialization or popularization of the practice of dentistry as is occurring in a number of community-based projects;
- Utilizing selection procedures for dental students which emphasize the ability to engage in non-clinical activities as an important part of their future work;
- Developing career pathways for all oral health personnel including general practitioners.

Job functions need to be clearly stated. For example, suggested functions and activities of a community dentist are:

Functions	Activities
Manager	Leader of primary oral health care team
	Monitoring and controlling the oral health sub-system
	Organise/co-ordinate preventive, treatment, and referral services
	Help data analysis research and information dissemination
	Help plan, supervise and evaluate the oral activities
Agent of socio-economic. development	Development of community participation in oral health
	Liaison with the public, politicians and other sectors
	Participate in community meetings and development activities
	Participate in intersectoral projects e.g., food and water
	Advocacy of better oral health
	Critical analysis of intersectoral plans for oral health implications
	Influence politicians to make healthful decisions
	Support appropriate development e.g., local food production
Dental officer	Complex treatment of patients
	Promotion of oral health at community, family and individual levels
Educator	
	Continuing education of colleagues
	Training of lower level oral health workers
	Oral health education of families and communities

It is essential to develop secure funding for the budget. This can come from within the community and/or outside it.

Economic constraints will determine the coverage achieved by health services. Without economic development extensive coverage with oral health care will not be possible. This leads to rationing where decisions have to be made concerning 'strategic teeth' that should receive priority.

Developing countries may need support to strengthen their health services, but they do not want or need conditional help. They do not need operating dentists but rather help in strengthening training programmes and with clinical and educational materials.

Donor agencies may play unfortunate roles in funding postgraduate studies in areas which are of no relevance to existing national situations. Increased responsiveness, sensitivity and responsibility to local situations in this respect is called for.

Comprehensive planning procedures involve intersectoral co-operation and integration, frequently involving ministries of health, education, finance and social-welfare, universities, schools for oral therapists, oral

associations, professional interest groups and international bodies. The planning group must involve all these structures as appropriate.

Equally important however, the planning activity must involve finding out the needs of the community in relation to oral health. Hence the planning of personnel training for oral health cannot be carried out without being built up from a comprehensive community diagnosis. As such it is best if the planning group is as broadly representative as possible and that it should be able to call on the professional skills of a range of specialist advisers particularly those in the social sciences.

There should be support and encouragement for local initiatives in developing oral health services, including assistance with the development of appropriate technology in the form of personnel, equipment and materials.

The role of international bodies in calling the attention of the oral profession to their responsibility in providing for the oral health needs of the whole population rather than selected groups needs to be emphasized.

IV COMMUNITY INVOLVEMENT

Genuine partnership between oral health personnel and the community depends upon mutual respect and trust. To build such a partnership is often difficult and may require a variety of contacts with different groups of the community as there are likely to be different levels of participation and interests. It is clear that it will be necessary to approach the community through local leaders and involve them in the planning, implementation, and evaluation of the community-oriented service as a first step. Later this should lead to the development of a fully community-based service, which is perceived by the community as their service.

There may be existing health workers in the community. It is important to include them and to define and develop their advocacy and facilitator roles within the group.

Neither the scientific basis of community based oral health programmes nor the capacity of the community to participate in such programmes on a scientific basis should be undervalued.

For many deprived communities health is defined in terms like caring, sharing, belonging and being able to look forward to tomorrow. A

HEALTH FOR THE MILLIONS

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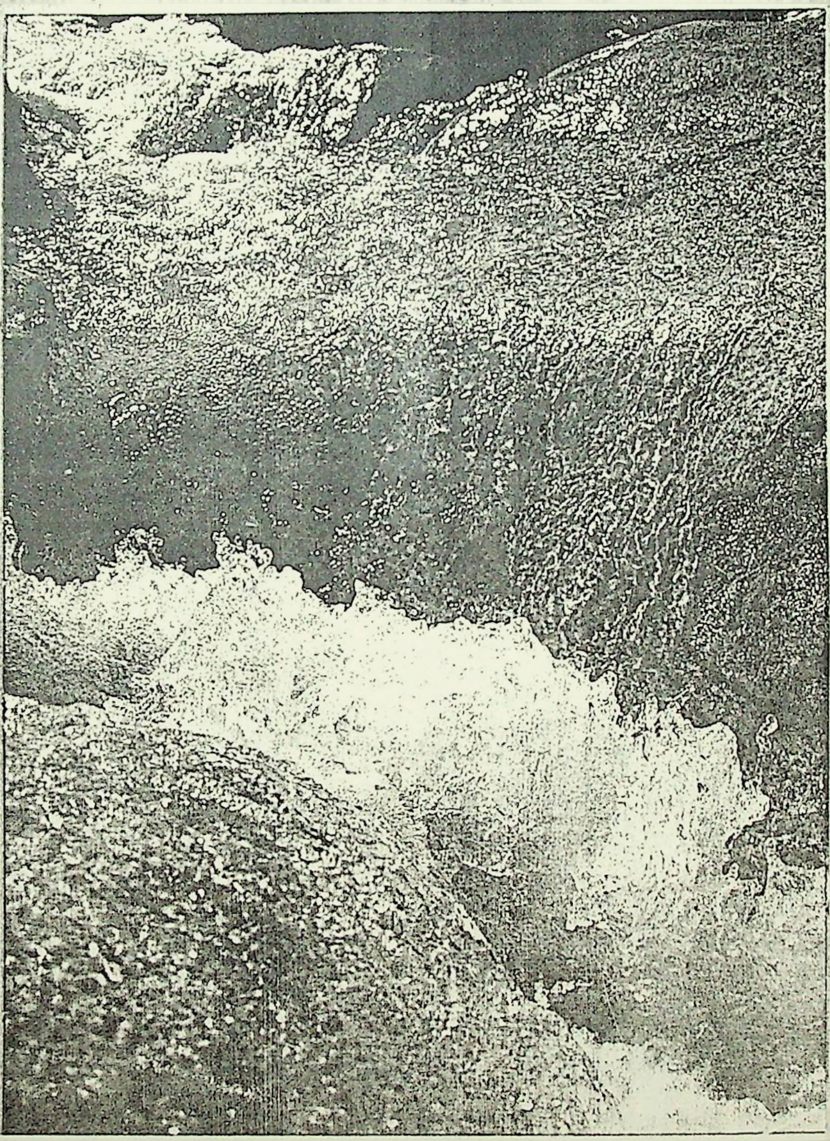


VOLUNTARY HEALTH ASSOCIATION OF INDIA

Water & Sanitation

and miles to go...

5

Fluoride : Too Much Can Cripple You

Excess flouride in drinking water can lead to fluorosis, a disease that surfaces in a variety of symptoms. The sub-mission planned for its control had as its key thrust awareness-raising, disease identification through village level epidemiological surveys and launching preventive measures essential for a disease which has no cure. Dr. A.K. SUSHEELA and G.GHOSH describe the submission's strategy and warn against misleading publicity recommending fluoride-based toothpaste for better dental health.

Although India has had the problem of fluorosis for over 50 years, very little had been done to mitigate the sufferings of the afflicted until the National Drinking Water Mission was launched in 1986-87. It is estimated that nearly 25 million people are afflicted with fluoride poisoning due to environmental pollution.

In India, the magnitude of environmental pollution with fluoride unlike in many parts of the world, is greatly enhanced by use of fluoride contaminated water, food, cosmetics like toothpastes and even drugs. Due to the earth's crust being extremely rich in fluoride-bearing minerals, the water is naturally fluoridated. The maximum fluoride content so far detected is 38.5 ppm (mg/litre) in drinking water.

Drinking water containing fluoride ranging from 1.5 to 38.5 ppm can cause severe health problems. Initially, the health problems were known to exist in only four states -- two in the Southern and two in the Northern parts of India and nowhere else. But to-day, 13 states, nearly fifty per cent, are declared endemic for fluorosis and associated health problems as a result of drinking fluoride contaminated water. It has become an urban as well as a rural health problem, affecting the poor and rich alike.

Government efforts

What did the Government of India do to deal with this public health problem? Since the early 1930s, the major focus has been to promote basic researches, epidemiolgical studies and clinical research to understand fluoride action on body tissues and on health in its totality. Researches in this field are still being promoted by various national and international agencies.

Inspite of the fact that India has substantial fresh water resources, there is a chronic shortage of "safe drinking water" in the country. The shortage may vary from mild to acute, depending upon geographical, topographical, climatic and other factors. The spread of the disease from four to 13 states over a period of 50 years is mainly due to population overgrowth necessitating the use of more and more water; people digging tube wells indiscriminately anywhere and everywhere; and total unawareness of the importance of water quality for human consumption.

To tackle the drinking water problem on a war footing, the Government of India during 1986, set up a Technology Mission on Drinking Water and related water management with the Department of Rural Development as the nodal agency. The strategy adopted for visible results is to focus on Sub-Missions, countrywide. The major Sub-Missions operating are:

1. Control of Fluorosis
2. Eradication of Guineaworm
3. Removal of Excess Iron
4. Removal of Salinity and Brackishness and
5. Source Finding and Water Management.

The methodologies adopted are:

1. Purification of water
2. Improvement of technical methods
3. Improvement of materials and designs
4. Improvement of maintenance methods
5. Computerized management information system
6. Scientific source finding
7. Continuous monitoring and evaluation
8. Community involvement and
9. Awareness programmes.

This article focusses on the activities in the Sub-Mission on "Control of Fluorosis" and the programmes which are on in the various states to remove fluorosis and associated health problems. The health problems have by now attained a magnitude, which is roughly estimated to have afflicted 25 million people, perhaps with an equal number on its way to being affected. The task assigned and the funds earmarked by the "Water Mission" to achieve the objective of the Sub-Mission on "Control of fluorosis" viz. to provide safe drinking water, prevent and control fluorosis within a time frame is by no means a casual task. A total commitment on the part of the Public Health Engineers, doctors, paramedical workers, village level functionaries and voluntary organizations to work in a co-ordinated manner, is a unique feature of the programme. The best scientific talents and expertise available in the field of public health engineering, defluoridation of water and health science have been identified and brought together to work in an integrated manner, with the participation of the people themselves.

Control in three phases

The Sub-Mission on "Control of

Fluorosis" has a three phased programe specially designed keeping in view the limitations and the lacunae in educational programmes in the country. The programme has been implemented in 18 districts in eight states during the past three years, covering over 50,00,000 people. The activities of the various phases are based on the following objectives:

Phase-I To conduct training-cum-awareness camps, and update the information on fluorosis, drinking water quality and defluoridation procedures for doctors, Public Health Engineers, para-medical workers and governmental and non-governmental personnel.

Phase-II To launch epidemiological survey(s) in the affected villages, taluks and districts to assess the exact magnitude of the problem as well as to analyse the quality of every drinking water source in the area.

Phase-III To introduce appropriate measures to provide safe drinking water as well as to ameliorate, prevent and control fluorosis.

The Phase-I activities are essentially an update on fluorosis which includes information on clinical manifestations, diagnosis, early warning signs of fluorosis and procedures to identify subjects afflicted with fluorosis. Under field conditions, without having to carry out sensitive laboratory-based tests, besides educating the people on the importance of drinking safe water, water quality assessment and defluoridation procedures (both at the domestic and community defluoridation installations) are dealt with. A little over 5000 individuals which include doctors, Public Health Engineers and village level functionaries have been trained in this Sub-Mission over the period of three years. The need for the up-date is essential, as doctors and engineers during their training lay emphasis on western concepts and practices. It may not be out of place to mention here that some of the text-books on Public Health Engineering in India still describe procedures to *fluoridate* drinking water rather than to *defluoridate* it. Besides, the doctors graduating from medical schools in the country are not taught about fluorosis in great detail. Fluorosis is considered a disease which has no treatment or cure and preventive aspects are often neglected. It is also true in India that the disease was very often misdiagnosed as arthritis, spondylosis or ankylosing spondylitis. We are not surprised to note the statement in Chemical Engineering News, (Page 37, August, 1988) that most doctors in the U.S. have not studied the disease and do not know how to diagnose it. It therefore emerges that even in U.S. the early warning symptoms of fluoride toxicity/poisoning may not be understood at all.

During Phase-II, the endemicity of the villages/districts for fluorosis is identified by visits to schools and from dental check-up. White, yellow, brown and black tooth enamel discolouration, either in spots or in horizontal streaks, is the sign of occurrence of dental fluorosis. In such cases, the water is probably contaminated with fluoride and the location endemic for fluorosis. During the past three years, several epidemiological surveys along with water quality testing with a focus on fluoride have been conducted by professionals, after attending the update camps. Based on the most recent data, the following observations and corrective measures both for providing safe water and preventing the disease have been introduced during the Phase-III activities:

- In an area endemic for fluorosis, it is not necessary that every source of water is contaminated with fluoride. The good sources are identified, labelled and the people are educated to drink from the good sources only; the fluoride-contaminated sources are used for washing and cleaning purposes. If the yield of water for consumption is low, possibilities are also explored for mixing the water from two sources, thereby diluting the concentration of fluoride to permissible levels.
- Presently in the National Drinking Water Mission, we are accepting contamination upto 1.00 ppm as the permissible upper limit as we have no other alternative. However, we do create awareness among the people, that the less the fluoride in water, the better the health.
- We also have data in India to suggest that 0.4 ppm of fluoride in drinking water is causing mild, moderate and severe forms of dental fluorosis and therefore we are looking for water with less and less fluoride contamination.
- It is also a fact that due to atrophy of the body's muscle fiber and connective tissue including the oral cavity caused by the use of fluoride, people living in endemic areas lose their teeth at an early age; they look much older and those who can afford it, resort to the use of dentures.
- Muscular weakness, loss of muscle power and neurological manifestations leading to excessive thirst, a tendency to urinate more frequently, although the volume of urine is not too large, are not uncommon among the afflicted individuals.
- Severe and widespread gastro-intestinal problems viz. anorexia, pain in the stomach, intermittent diarrhoea, chronic constipation, gas formation and a bloated feeling in the stomach (non-ulcer dyspepsia) caused due to drinking fluoridated water have been confirmed. Changing the source of water with low levels of fluoride (below 1 ppm) provides relief from the gastro-intestinal problems within a period of two to three weeks. In areas endemic for fluorosis, gastro-intestinal problems alert suspicion of fluoride toxicity and are used as early warning signs for preventive measures to be introduced. The

A profile of a few districts in India revealing the number of people ailing due to fluoride toxicity are shown

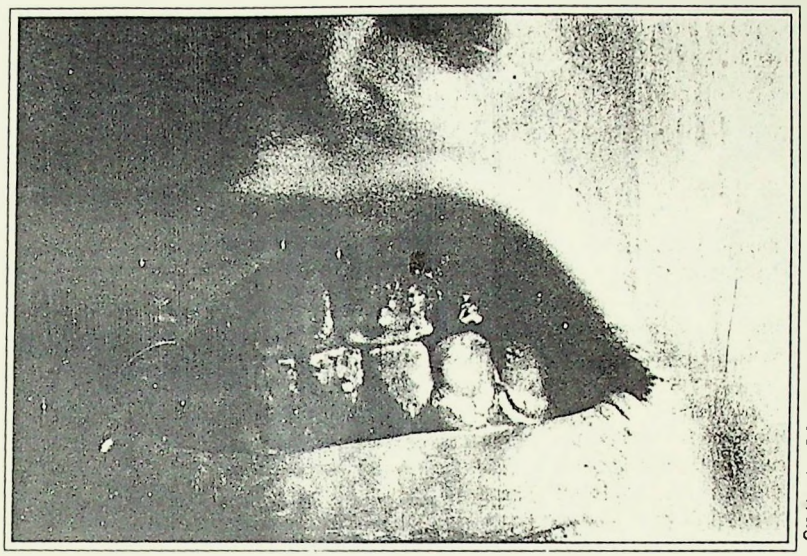
Name of the districts and state	Total population and the number of people afflicted with different complaints			Range of fluorid in drinking water
Dharwad (Karnataka)	Total population examined	--	72,744	0.3-150 ppm
	Afflicted with:			
	Dental Fluorosis	--	12,266	
	Skeletal Fluorosis	--	3,747	
	Gastro-intestial problems	--	6,304	
Raichur (Karnataka)	Total population examined	--	1,50,215	0.2-7.5 ppm
	Afflicted with :			
	Dental Fluorosis	--	12,933	
	Skeletal Fluorosis	--	2,999	
	Gastro-intestinal problems	--	1,882	
Amreli (Gujarat)	Total Population examined	--	56,189	1.8-11 ppm
	Afflicted with :			
	Dental Fluorosis	--	9,919	
	Skeletal Fluorosis	--	6,762	
	Gastro-intestinal problems	--	245	
Gurgoan (Haryana)	Total Population examined	--	85,792	0.2-19.6 ppm
	Afflicted with :			
	Dental Fluorosis	--	6,970	
	Skeletal Fluorosis	--	600	
	Gastro-intestinal problems	--	200	
Kurnool (Andhra Pradesh)	Total population examined	--	14,91,791	0.1-9.6 ppm
	Afflicted with :			
	Dental Fluorosis	--	43,927	
	Skeletal Fluorosis	--	8,833	
	Gastro-intestinal problems	--	30,400	

damage caused to the human intestinal mucosa, due to drinking fluoridated water is extensive. Under the Water Mission, in the affected areas where there are no good source(s) of water, domestic defluoridation procedures are being popularized among lactating mothers as well as pregnant (expectant) mothers, besides erecting community defluoridation tanks for the public. Alternative source(s) of safe drinking water, either by bringing it in from a distance through pipelines (if economically viable) or through alternate under-ground sources are also being considered for implementation.

The reports published from India, by earlier investigators, have been substantially up-dated during the last 10-15 years. It is strange to note that the west invariably quotes Indian reports on fluorosis which are outdated. For example, an Indian report which appeared in 1970 in a WHO publication entitled "Fluoride and Human Health" indicates that one can get afflicted with fluorosis by drinking water contaminated with 20 ppm of fluoride if consumed for 10 years; this is most conveniently quoted by the pro-fluoridation experts of the western world suggesting that water with 2 ppm is safe to drink. The fact remains that even 2 ppm of fluoride contaminated water can cause crippling fluorosis, if the calcium content of the water is low and alkalinity high and if consumed for even a couple of years, leaving aside 10 years.

Harmful publicity

Intermittently, we also face problems in the country due to the World Health Organization's publicity for use of fluoride in the name of prevention of caries as "teeth are for a life time" (as though other organs are not!). WHO's recommendation to drink fluoridated water, to use fluoride toothpaste, use fluoride salts in cooking and have it available on the table for use with food which has been promoted as educational material for the past several years, has come under severe criticism in India. With the initiative taken by the Ministry of Health and Family Welfare (Govt. of India) during 1989, WHO has been asked to refrain from such publicity in India as it causes more damage than good to the people. WHO has also been asked to add a footnote to their pamphlets indicating that it is not applicable to India and other countries where excess fluoride is the problem. WHO headquarters in Geneva have also been informed through their Regional Office at New Delhi of the Water Mission's efforts to defluoridate water as well as control fluorosis in India at massive expenditure. It would be worthwhile if such UN organizations would adopt a bifurcated policy in dental care appropriate to developing countries without imposing fluoride everywhere. In the developing countries, to prevent dental caries the need of the hour is not fluoride but adequate awareness of the importance of oral health and hygiene besides improving adequate calcium and vitamin C in the diet.



Credit UNICEF

Dental fluorosis in child

Some developing nations are now reporting on the unsuitability of WHO guidelines for fluoride concentration in drinking water. A recent report from Netherlands, based on a study in Senegal has shown dental fluorosis in children where fluoride in water ranged from 0.1 to 7.4 ppm, and prevalence of mild dental fluorosis is 68.5 per cent at 1 ppm of fluoride in drinking water. When fluoride exceeded 4 ppm, the prevalence of dental fluorosis reached 100 per cent. It has been suggested that the WHO guidelines for fluoride concentration in drinking water in Senegal are unsuitable and the upper limit should be reduced to 0.6 ppm (Lancet : 11, 223-225, 1988).

It is now quite gratifying to note that in India, WHO has extended support to the Water Mission activities, particularly the Sub-Mission on "Control of Fluorosis". WHO, during the early 1970s, was fully prepared to fluoridate India's water, but, because of the opposition they faced from Indian scientists, the scheme could not be implemented. Perhaps the support that is now forthcoming may be the beginning of a new era, when WHO may consider a totally different strategy for prevention of dental caries

in India and other developing countries instead of promoting the use of fluoride.

Toothpaste and fluorosis

It is unfortunate that the use of fluoride for prevention of caries, although formulated and brought out 50 years ago by U.S.- based dentists, is still being promoted the world over without questioning the rationale nor the health hazards which have been emerging due to fluoride poisoning. It has been show by Indian dentists that by use of fluoridated toothpaste for brushing teeth, in young and old, the serum fluoride levels are enhanced within minutes (Rajan et al, Fluoride in toothpaste : Cause for Concern, Fluoride 21:4, 1988; Rajan et al, Serum and Urine Fluoride in Tothpaste Users, J.Ind. Dent. Assoc. 59:137-142, 1987). The oral mucosa, rich in blood vessels absorb fluoridations rapidly. The sub-lingual blood vessels (the ones below the tongue) drain the stuff directly to the superior venecava and then to the heart. It is not true that toothpaste never enters the body unless it is swallowed/ingested. Fluoride does enter circulation directly from the oral cavity through the fine blood vessels of the mouth. Fluoride being a persistant bioaccumulator, even small amounts that enter through fluoridated toothpaste, are guaranteed entry, not only in children but even among adults and the cumulative or additive effects of fluoride are causing serious concern.

It has been discovered in India recently that there is no toothpaste marketed which is free of fluoride, whether labelled or otherwise. The amount of fluoride arising as a contaminant from the raw materials used viz. chalk, talc and calcium carbonate, may be as high as 800 ppm. It has also been observed in India as a result of extensive laboratory investigations that the fluoride is not mixed homogenously in the paste. When the paste is squeezed out from different depths of the tube, the amount of fluoride in the fluoridated brand of paste is highly variable. In none of the so-called fluoridated brands of the toothpaste, the quantity of fluoride in the paste is revealed on the carton or the tube. The expiry date of the toothpaste is never revealed either. These we consider unethical practices.

In order to ensure quality control procedures in manufacturing, a maximum contaminant rate of 800 ppm may be permitted in Indian toothpastes, but we insist on having a warning inscribed on the carton which should read: "Excess fluoride is injurious to health". This would alert the consumer to look for a paste with least fluoride contamination. We are also aiming at curtailing the false publicity for promoting the use of fluoride in the name of prevention of caries. Although children below the age of six years are not supposed to use fluoridated toothpaste, as per the recommendation of the Indian Council for Medical Research, invariably the advertisements are aimed at children, misguiding the public.

Perhaps due to the brain-washing publicity promoting fluoride for prevention of caries on television, radio and other print media, people do not quite realize the damage that excess fluoride can do to them. It has been shown that excess ingestion of fluoride leads to the accumulation of a particular chemical substance viz. dermatan sulphate, both in bone and teeth. The substance, on accumulation, tends to demineralize the area around, both in teeth as well as in bone. Such demineralized zones in the teeth get pitted and perforated in dental fluorosis besides being discoloured (Susheela, et al, Arch. Oral Biol., 33 10,765. 1987). The belief that cavity formation occurs only in dental caries is outdated. In fact, cavity formation may be aggravated in some due to excess ingestion or use of fluoride, leading to dermatan sulphate formation and ensuing demineralization of the tooth matrix.

In reality, by using fluoride for preventing caries, believing that it only makes the enamel strong, no longer holds good because fluoride also causes demineralization of the teeth which get pitted, perforated and chipped. In other words, the damage it causes to the teeth is never taken into account as it nullifies the age old concept that "fluoride is good for teeth."

Towards heart disease and cancer?

In addition to the above, fluoride induces cholesterol production in males and can also lead to blocking/calcification of blood vessels, specially in the region of the main vessel leading from the heart viz. the aorta, causing cardiac problems. This certainly means that the toxic effects of fluoride far outweigh its benefits in the Indian situation.

There are reports on high incidence of cancer due to fluoride in the U.S. (Cancer Mortality in Relation to Fluoridation and Population Changes, Data from 140 largest U.S. Cities from 1940-1980, Burgstahler, Int. Conference of the Fluoride Society, Utah, 1986). Data based on animal experiments from Japan and other parts of the world, also suggest that fluoride is cancer-causing (Mutation Research, 139, 193-941, 1984; Science of Total Environment 68, 79-76, 1988). These are serious problems that a nation ought to take into account before we start adding fluoride in massive doses to drinking water and toothpaste just because a few dentists, and of course the manufacturers, promote outdated concepts to promote the use of fluoride.

-- Dr. A.K. Susheela is National Co-Ordinator for Health Sector Activities, Sub-Mission on 'Control of Fluorosis', Technology Mission on Drinking Water. Dr. G. Ghosh is Director of the National Drinking Water Mission.

FLUORIDATION: ACTION REPORT

January 1986

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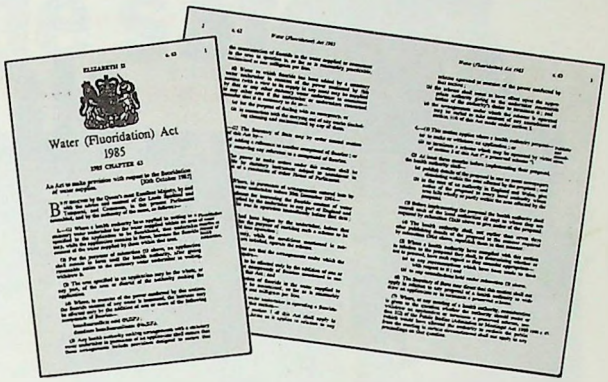


Fluoride Act gets Royal Assent: major boost in fight against tooth decay

THE best news for dental health in Britain for years – that is how many health authorities are responding to the Water (Fluoridation) Act, which received its Royal Assent in October, 1985.

The Act makes it clear that it is perfectly legal for health authorities and water suppliers to introduce fluoridation schemes to prevent tooth decay. Millions of children in England, Scotland and Wales stand to benefit throughout their lives.

Up to now about ten per cent of Britain has had water supplies fluoridated. Fluoridation means adjusting the natural fluoride up to the controlled level of one part of fluoride per million of water – a safe and highly effective procedure which has generally halved tooth decay among children drinking fluoridated water from birth.

A court case in Scotland two years ago, whilst totally vindicating fluoridation as a benefit to public health, cast doubt on its legality. That doubt hinged on the precise legal definition of words in previous Acts of Parliament concerned with the management of water supplies.

So the government decided to set the record straight with a new Act which specifically dealt with the powers of public bodies to fluoridate water.

Now that the Act is finally on the statute book, there is no doubt whatever. Health authorities are free to ask water suppliers to add fluoride to water as soon as detailed guidance has been issued by the DHSS and the Department of the Environment.

Most health authorities regard fluoridating water as the single most important thing they can do to prevent the misery and pain of toothache. They can now go ahead with their plans.

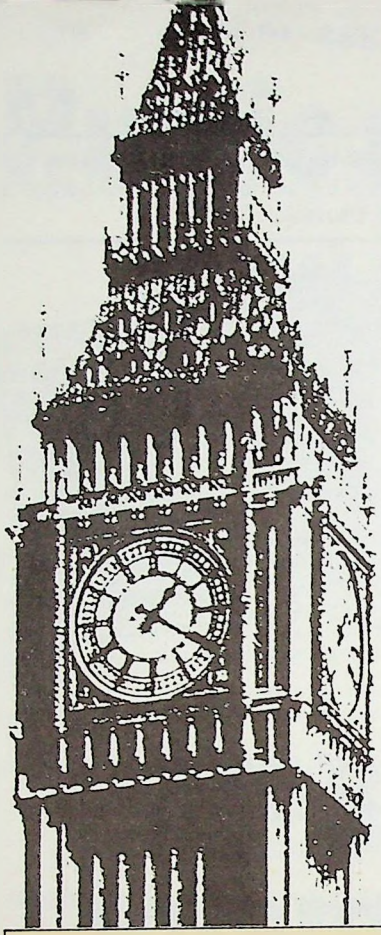
The new Act confirms that it is up to health authorities to decide whether to fluoridate local water supplies in the interests of preventing dental caries – one of the commonest diseases in the country and one of the most expensive for the NHS in treatment costs.

As before, health authorities still have to request the cooperation of water suppliers, some of which have in the past been reluctant to comply because of possible legal problems. Those problems no longer apply, thanks to the Water (Fluoridation) Act.

Once a health authority has decided to request a particular scheme, it must publish details in the local press and consult with local authorities at least three months before implementing the proposal.

It must do the same if, for any reason, seeking to discontinue a fluoridation scheme.

Separate legislation has yet to be introduced to cover Northern Ireland.



Parliament has done its job:

NOW IT'S UP TO THE NHS

HEALTH authorities now have the best opportunity they have ever had to improve the dental health of the community. That is the view of the British Association for the Study of Community Dentistry, which has long advocated fluoridation as the most effective way of tackling dental caries.

But the Association's President, Professor Aubrey Sheiham, points out that health authorities must be prepared to campaign vigorously for their fluoridation policies at local level.

"The new Act reaffirms that it is legal to fluoridate but it does not deliver fluoridation on a plate", said Professor Sheiham. "Health authorities still need to receive the cooperation of water suppliers and will no doubt find that opposition, though from a tiny minority, is vociferous and well-organised".

Professor Sheiham commends health authorities generally to follow the examples already shown in the West Midlands and North West, where regional coordinating groups have been set up to ensure that NHS policies are well publicised and effectively implemented.

Both groups are led by District Health Authority Chairmen. John Charlton (South Birmingham) chairs the West Midlands group and Gordon Legat (South Manchester) chairs the North Western group.

The West Midlands group, established in 1978, has had remarkable success. Since its inception, fluoridation schemes have been introduced to serve half a million people in Coventry, Solihull, Warwickshire and Worcestershire. Health authorities have also concluded agreements with water suppliers for schemes which will benefit a further 1¾ million consumers in many parts of the region – schemes which can proceed without delay thanks to the Water (Fluoridation) Act.

"While each District has to decide locally on the principle of whether to fluoridate, experience shows that they need to get together, usually with their Regional Health Authority, to stand any serious chance of putting policies into practice", said John Charlton.

Hot on the heels of the West Midlands are health authorities in the North West, a region with no fluoridation at present but a record of poor dental health.

"We are determined to make sure children in Greater Manchester and Lancashire benefit from the new legislation", said Gordon Legat. "Just three schemes could cover the vast majority of our population. We must not miss the opportunity created by the new legislation".

Other regions are known to be actively considering the next steps to take. Professor Douglas Jackson, Chairman of the British Fluoridation Society, said that the Society would do everything it could to see that the vast amount of campaign expertise built up in the West Midlands was made available to health authorities in the rest of the country.

Political backing across the board

SPEAKERS of all political parties in both Houses of Parliament expressed their strong support for fluoridation during the successful passage of the Water (Fluoridation) Bill.

Here are just a few of the important statements made during the debates in the Commons and in the Lords:

Mr Kenneth Clarke (former Minister of State for Health)

"Water fluoridation has advantages over all these (other) methods in that it is highly cost-effective, does not require the use of trained personnel or conscious effort by the recipient and confers a benefit on the whole of a given community".

Mr Michael Meacher (Opposition Spokesman on Health)

"Dental caries is one of the most costly diseases . . . Its prevention or reduction, in terms of the individual's health and to the nation as a whole, is a prize worth striving for. How can one justify not giving the child who lives in Wolverhampton or Salford the same protection as the child who lives in Birmingham or Watford already enjoys?"

Mr Michael Meadowcroft (Liberal Spokesman on Health)

"The most telling fact is that during twenty years or more since the argument has been contested in and out of the public arena . . . no professional health body has changed its mind about the efficacy of fluoridating water".

Lord Winstanley (a former general medical practitioner)

"As a doctor, and as a person who has looked into the issue with great care, I can say that I am wholly convinced that no public health measure has ever been subjected to such exhaustive and close scrutiny and ever been found to be so totally free of dangerous side effects or risks of any kind".

Lord Colwyn (a practising dentist)

"With the addition of one part per million of fluoride to water, we have the safest, most widely available method of bringing prevention to patients who do not seek first-hand advice from the dental surgery as well as the regular attenders".

Yet another 'thumbs up' on fluoride safety

YET another major report on fluoridation has given it a clean bill of health on safety. A special working party set up to investigate allegations by opponents of fluoridation about possible links with cancer found no evidence of hazard.

This conclusion confirms earlier reports by the Royal College of Physicians, the US National Cancer Institute and many other scientific bodies that fluoride in the water – whether present just naturally or at artificially supplemented levels – does not cause cancer.

Like the others, the Knox Report found no cause for concern, either in terms of cancer in general or cancer of any specific part or organ of the body.

The working party reached this view after exhaustive inquiries into previous allegations and after giving full consideration to the work of scientists from all over the world.

The report – "Fluoridation of Water and Cancer: A Review of the Epidemiological Evidence" – was commissioned by the DHSS.

The working party was made up of individuals eminent in epidemiology, cancer research, pathology, statistics and water treatment, and was led by Professor E.G. Knox of the Universtiy of Birmingham.

After four years' study the working party published a report in January, 1985. In its preface, Dr E.D. Acheson, Chief Medical Officer at the DHSS, said: "The wealth of evidence which has been gathered during this period (i.e. of 40 years' experience of water fluoridation) including that presented in this report, justifies the conclusion that fluoridation is a safe and effective method of reducing dental decay".

CANCER ALLEGATIONS FIRMLY REJECTED IN KNOX REPORT

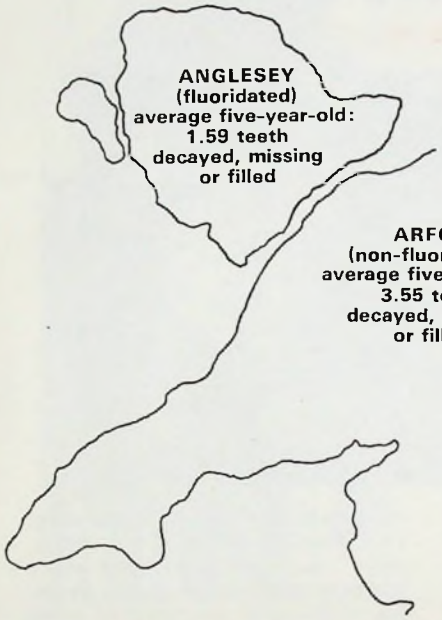
THE Knox team had carefully examined the statistics on cancer deaths produced by two American anti-fluoridationists but rejected their work on the following grounds:

1. ***A major weakness in the method used by the two Americans is their failure to make comparisons between cancer death rates in different populations as fair as possible – in other words to compare like with like.***
2. ***They made many mistakes and inconsistencies in the handling of data.***
3. ***They failed to conduct acceptable tests of statistical significance.***

The cancer scare was also firmly knocked on the head recently by Lord Jauncey, the judge presiding over a court case in Scotland where anti-fluoridation groups were trying to stop Strathclyde Regional Council from introducing a scheme.

Lord Jauncey dismissed their arguments, concluding in his verdict that fluoridation is safe and beneficial.

The AMAZING ANGLESEY story



ONLY a narrow strip of water separates the children of Anglesey from those of mainland Wales. But where dental health and tooth decay are concerned, the children of mainland Arfon could be an ocean away.

For in a study of dental decay in both places, the evidence points conclusively to the fact that children on fluoridated Anglesey have many fewer decayed, missing or filled teeth than their counterparts in non-fluoridated Arfon.

The study was carried out by researchers from Gwynedd Health Authority and the Universities of Leeds and Birmingham, who had noted that tooth decay among English children appeared to be falling. They wanted to find out if a similar phenomonon had been experienced in Wales.

Taking Anglesey – where the water is fluoridated – and comparing it with the adjacent mainland area of Arfon – where the water contains only relatively low natural levels of fluoride at 0.1 parts per million – seemed a good way of finding out.

As the diagrams of this page show, the results were significant. The research team compared the dental health of children aged 5, 12 and 15 who had been born and bred in the two areas. The five year-olds in Anglesey had, on average, 1.59 teeth decayed, missing or filled while in non-fluoridated Arfon the figure was over twice as high at 3.55.

By the age of 12, the Anglesey child had 2.59 teeth decayed, missing or filled. In Arfon the figure was much higher at 4.46.

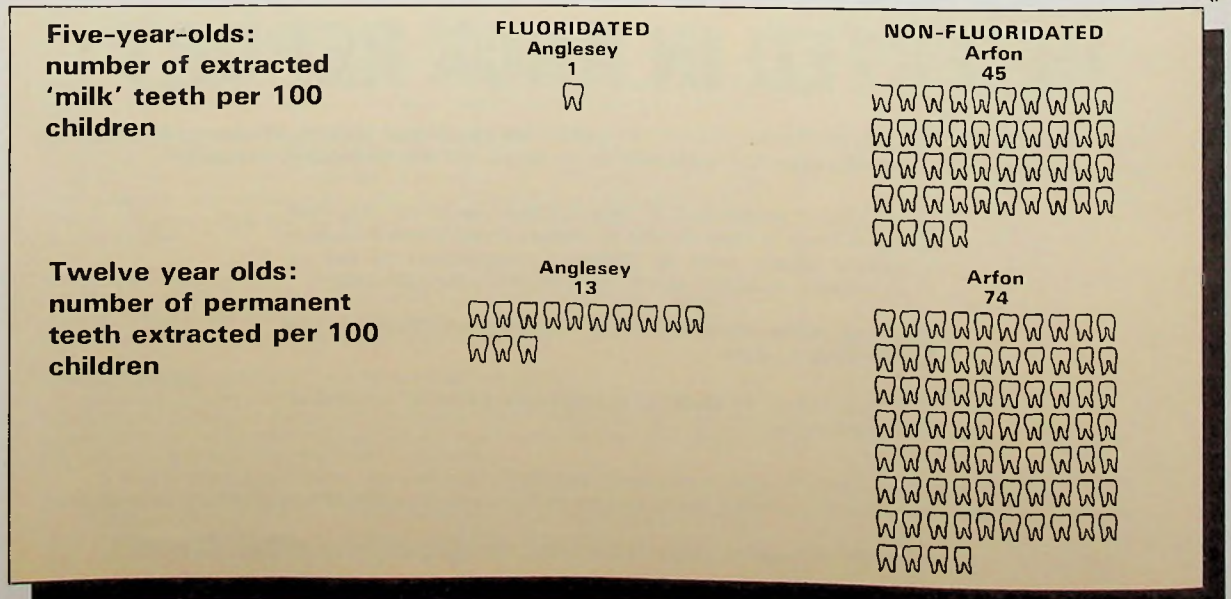
And for teenagers at 15 the comparative figures were 4.73 for Anglesey and 7.69 for Arfon – still a very big difference.

Other comparisons of dental health also put Anglesey in a favourable light. For every 100 five year-olds in Anglesey, only one deciduous tooth had been extracted . . . the figure for Arfon was 45.

The comparative figures for permanent teeth of the 12 year-olds were 13 and 74 respectively, and for the 15 year-olds 48 and 108.

As in England, the state of the children's teeth is improving in Wales – but with fluoridated areas showing a better track record.

Between 1974 and 1983 there was a drop in tooth decay among five year-olds of 44 per cent in the fluoridated area compared with 22 per cent in the non-fluoridated community.



NEWCASTLE knocks the teeth off MANCHESTER

MANCHESTER and Newcastle are both large industrial cities in the north of England. You would have no reason to suppose that children living in one or the other enjoy much better or worse dental health.

But 5-year old Manchester children have two and a half times as much tooth decay as 5-year-olds from Newcastle. Why should that be? The answer is fluoridation.

In July, 1985 a random sample of 245 children in North Manchester was examined, together with a similar sample from Newcastle.

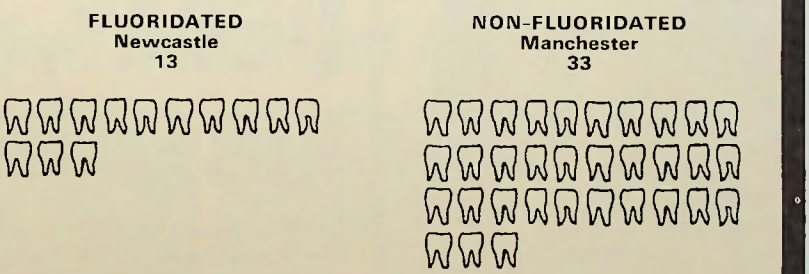
The differences were considerable. Manchester 5-year-olds had on average 3.3 teeth decayed, missing or filled. Those from Newcastle had only 1.3 teeth decayed, missing or filled.

Whereas only 30 per cent of Manchester children had never experienced dental decay, as many as 62 per cent of Newcastle children had never experienced it.

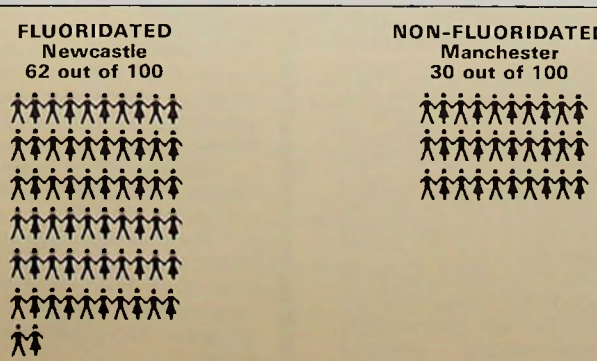
Newcastle receives fluoridated water. For the moment, Manchester does not.

Commenting on the results of the survey, the District Dental Officer for North Manchester, Mrs Jacqueline Duxbury, said: "Fluoridation must remain a major objective for Districts like ours, where dental health of young children still needs to be improved".

Five-year-olds: number of teeth decayed, missing or filled per 10 children



Five-year-olds % free of tooth decay



Putting dental mottling into perspective

OPPONENTS of fluoridation, as if clutching at straws to try to find something to complain about, claim that fluoride causes dental fluorosis, a severe form of dental mottling. This is not true.

Dental mottling, the white or brown flecks which occur mainly on second teeth, is usually caused by damage to the teeth while they are still developing in the gums. It results in certain areas of the tooth enamel being under-calcified.

Most of the mottled areas are not detectable except to the professional eye. They have a specific distribution not only on the tooth surface but between the teeth.

The number of children with this form of mottling varies from one community to another, whether or not

the water is fluoridated. In fact, it has been found that in areas where drinking water is fluoridated to a level of one part per million, the number of children with dental mottling is no greater than elsewhere and in some places is less than would be expected.

Dental fluorosis is a far more severe form of dental mottling which may appear if the natural level of fluoride is much higher than that maintained in fluoridation schemes. It is far more noticeable than dental mottling, has a different distribution, affects all the teeth, and is considered to be the first sign of possible fluoride toxicosis.

So in their over-enthusiasm to condemn fluoridation, those who are opposed to it on principle have wrongly confused dental mottling – which is found in its mild forms in all communities – with dental fluorosis.

ERODING TOOTH DECAY, not LIBERTY

LORD Avebury, Chairman of the Parliamentary Human Rights Committee, believes that fluoridation of water supplies is perfectly consistent with personal liberty.

Writing last year in the "British Dental Journal", he dismisses as invalid the so-called libertarian arguments used by opponents of fluoridation.

He said: "Fluoride is a natural constituent of water supplies, as indeed it is of many foods. The adjustment of the quantity to an optimum level cannot be compared with the addition to water of a substance not found there ordinarily.

"Nor can it be described as 'mass medication', a term frequently used by opponents, since it is not a means of curing a disease. A substance which has the effect of maintaining medical or dental health is more in the nature of a food or nutriment than a medicine.

"The individual liberty arguments against fluoridation are invalid, as can be judged from the fact that the issue has never been taken up by the National Council for Civil Liberties.

"No consumer has the right to dictate the chemical composition of the water supply, a recipe for anarchy. What is at stake is not the erosion of liberty but, in the words of a former Minister of Health, 'the erosion of millions of teeth and the resultant suffering and misery of thousands of children which fluoridation would go far to prevent'."

DISEASES IN THE WORLD TODAY

the foods that prevent them

all kinds of meat, but especially liver

fruits, especially citrus fruits, guava, mango, pineapple, berries

green vegetables

sea foods, such as fish, shellfish and algae

iodized salt

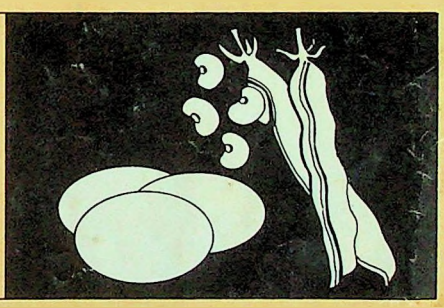
milk and cheese

meats and fish

eggs

pulses, such as beans, peas and lentils

groundnuts



breast milk for infants

cereals, such as rice, wheat or maize

roots and tubers (potatoes, yams or cassava)

fats and oils

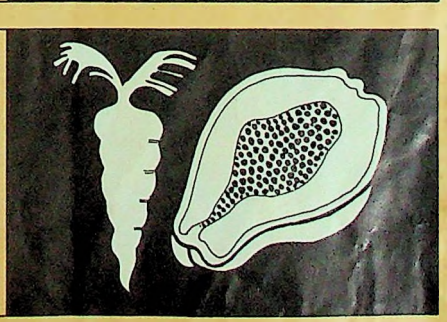
whole milk, butter and cheese

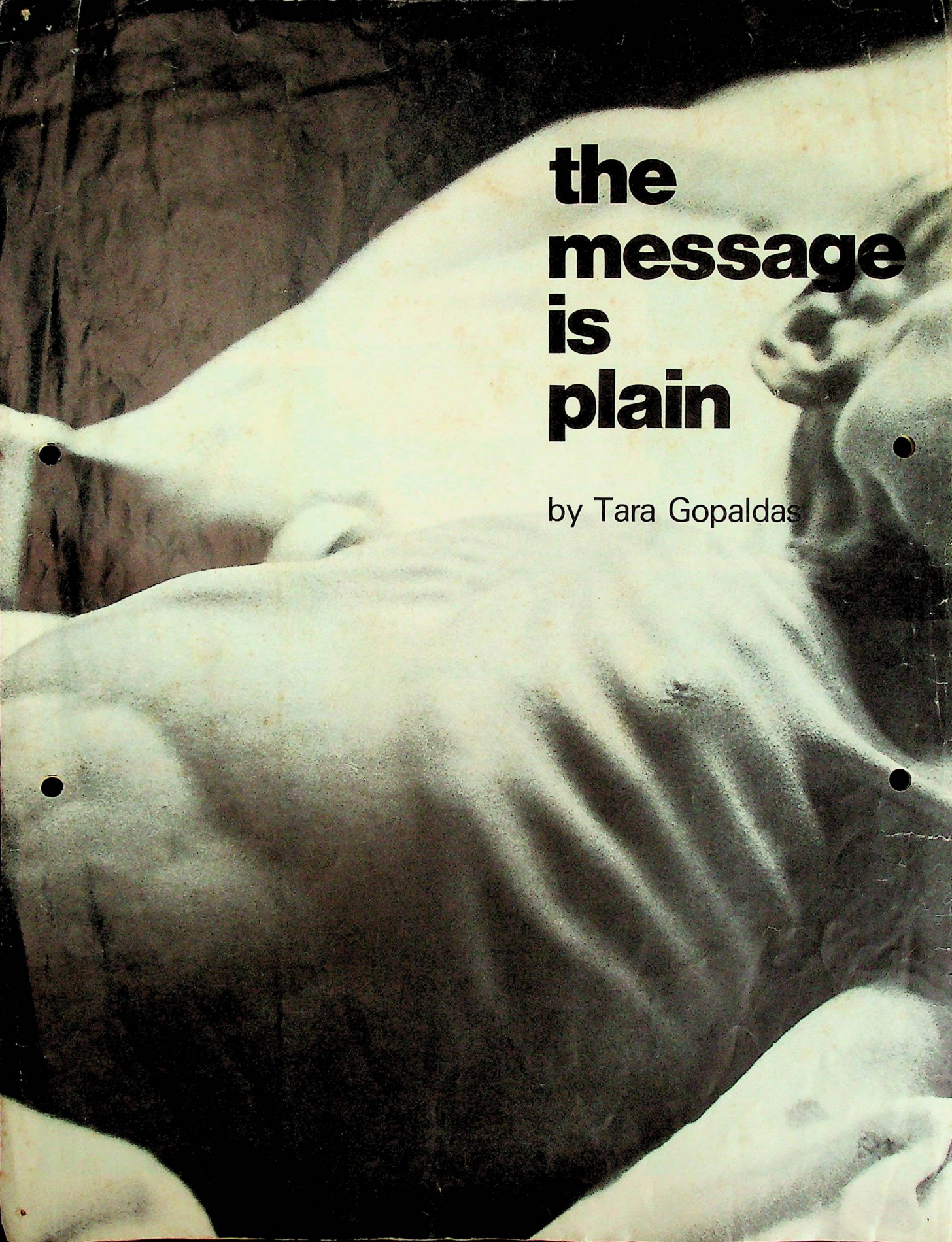
yolk of eggs and liver

vegetables, especially carrots and "greens"

yellow fruits, such as mango and papaya

red palm oil





the message is plain

by Tara Gopaldas

Japan. Yields, at 1.6 tons per hectare, would be less than half those achieved in Japan, Korea, Taiwan, Western Europe and the United States.

Most developing countries will need substantial external assistance to finance the heavy investments required for agricultural development. The recent alarm about world food prospects has been useful in focussing attention on this problem. In the past four years, total commitments for agricultural projects by the IBRD (International Bank for Reconstruction and Development) and other multilateral organizations have more than tripled, from US$ 724 million to $ 2,440 million. Bilateral assistance programmes of OECD (Organization for Economic Cooperation and Development) countries contributed an additional $ 800 million annually in 1973 and 1974. Progress is being made toward the establishment of an International Fund for Agricultural Development, proposed at the World Food Conference, in which the oil-exporting countries are pledged to contribute about half of the target of $ 1,000 million. A major expansion of the international agricultural research effort, sponsored by the World Bank, FAO and UNDP (United Nations Development Programme), is under way. The developing countries themselves are channelling more of their own resources into food production.

There are other encouraging signs. Quite apart from the countries of OPEC (Organization of Petroleum Exporting Countries), many developing countries have been successful in expanding their exports and thus improving their ability to pay for needed food imports. Total exports of the developing countries, excluding oil, have been growing during the past two decades at an annual rate of about 7 per cent, in real terms, reaching $63,000 million in 1973. Their exports of manufactured products have been rising at twice that rate and now represent more than one-third of the total. While the poorest countries have been lagging behind the more advanced developing countries, their exports have also been growing. For example, the volume of exports of manufactured products from South Asia is currently rising by over 8 per cent annually. At this rate, they should increase from $ 3,000 million at present to $ 6,500 million in 1985.

Finally, there is increasing evidence that population growth is beginning to slow down in the developing countries as birth rates continue to fall while mortality rates are levelling off. Here again, progress has been greatest in some of the more advanced developing countries; but there are indications that the population growth rate in India may also have passed its peak.

These developments are coming none too soon; but taken together they give reason for hope that hunger and malnutrition may finally be banished from this earth. ■

◁ *An Indian scientist demonstrates the dramatic contrast between ordinary rice and a new high-yield variety. Modern technology has brought about spectacular improvements in food productivity through the development of hybrid strains. (Photo WHO/FAO/D. Mason)*

Farmers on a communal farm in Peru applying fertilizer to cabbages. Most developing countries could increase their food production several fold if only they had access to the technical advances that have been made in agriculture. (Photo WHO/FAO/C. Sanchez)

▽

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THE FIVE PRINCIPAL DEFICIENCY

what are they – how to detect them

anaemia

Insufficient red pigment (haemoglobin) in the blood (mainly iron deficiency)

Main symptoms: pallor of skin and mucous membranes, general fatigue, breathlessness after exertion, palpitation, loss of appetite (anorexia), indigestion (dyspepsia)

endemic goitre

Enlargement of the thyroid gland resulting from iodine deficiency

Main symptoms: deformity of the neck, mental retardation and deaf-mutism; may occur in children born to mothers with goitre

kwashiorkor

Severe protein deficiency

Main symptoms: swelling (oedema), apathy and irritability, "flaky-paint" skin, sparse, straight and dyspigmented hair

marasmus

Severe calorie deficiency

Main symptoms: growth retardation, wasting, no subcutaneous fat, atrophied muscles – "all skin and bone"

xerophthalmia

Severe Vitamin A deficiency

Main symptoms: dryness of the eyes (xerosis), night-blindness (nyctalopia), corneal ulcerations, leading to blindness

Atmospheric pollution causing dryness of eyes

The Hindu, March 6, 1998

By Our Staff Correspondent

BANGALORE, March 5.

Rising levels of atmospheric pollution is cited as one of the most common factors that cause dryness of the eyes. With the number of vehicles increasing annually, air pollution is on the rise, and the eyes are made to wade through a sea of highly polluted air leading to evaporation of the tear film that surrounds the eye.

What is this eye disorder referred to as dryness of the eyes? "This is typically characterised by itching or burning sensation in the eye or redness of the eye and sometimes heavy tired and watery eyes," says Dr. Ram Mirlay, consultant ophthalmologist. Dryness is a common problem worldwide, and he is of the view that all steps should be taken to protect the eyes from the dust. "If dryness is not checked, it will lead to chronic eye disorders", he cautions.

One of the ways to protect the eyes is to wear dust protective goggles. "Don't you wear slippers or shoes when you walk on the road, similarly use this goggles to safeguard your eyes", he says. However, even this, he points out, may not give total protection as the eyes are not totally shielded.The problem of dryness occurs when the tear film, which is vital for clear vision and for the eye to remain healthy, begins to evaporate on account of constant exposure to the various pollutants including the pollen dust, an environmental condition peculiar to Bangalore.

Even airconditioned setting and long hours spent in front of the computer screen can cause this dryness, ophthalmologists say. According to them, airconditioners provide cooled dry air which evaporates the tear film, and those who work in such surroundings in cities that are dry and have low levels of humidity, are likely to be affected most.

As for the computers, research indicates that up to 75 per cent who work on computers experience one or more reversible eye problems. This is because when a person focuses hard on a computer screen, his blinking rate drops and this facilitates dryness. It is learnt that when people converse they blink between 18 to 25 times per minute on an average, but when they read or study the rate drops. When viewing a computer screen, it drops to about seven times a minute, it is said. The decreased frequency of blinking causes the tear film on the eye to evaporate faster resulting in dryness.Dryness could be treated by the use of tear substitute or an artificial lubricant. The artificial lubricant supplements the natural moisture of the eye and offers immediate relief." Polyvinyl alcohol based solution is the closest substitute to natural tears," says Dr. Ram Mirlay. However, it is essential to consult a doctor first before using the tear substitutes. According to him, in the U.S. 50 per cent of all eye drops are tear substitutes.

Here is some information that could help reduce eyestrain for computer users. Take breaks by just looking around the room; wear glasses; keep the screen free from dust; adjust lighting in the work area to minimise reflections and glare; adjust position of computer screen so that the top of the screen is slightly below eye level, position your screen so that you are looking down at it.

DIS - 1A. 25

As regards computer setting, black characters on a white background are usually the best, but other combinations are allright, if there is plenty of contrast between characters and the background. Dark backgrounds should be avoided. Lastly, blink more often while working at your computer screen to check dryness of the eyes.

NSAIDs Cause 1 in 5 Heart Failures

This recent study found that the use of nonsteroidal anti-inflammatory drugs (NSAIDs) caused a greater than 10-fold increase in the risk of congestive heart failure (CHF) in patients with a history of heart disease. In those without a history of heart disease, the use of NSAIDs still increased the risk by 60%.

Although previous studies have shown that the use of NSAIDs could lead to the development of congestive heart failure (CHF) in some patients, the magnitude of the association was not fully understood.

The authors speculate that if their results are correct, it means that NSAIDs are responsible for 19% of all hospital admissions for CHF.

CHF, commonly referred to as simply "heart failure", is a disorder in which the heart loses its ability to pump blood efficiently, thereby failing to meet the demands of the body. As pumping action is lost, blood may back up into other areas of the body, including the liver, gastrointestinal tract, and extremities (right-sided heart failure), or the lungs (left-sided heart failure). Some other potential causes of CHF include:

- high blood pressure
- heart valve disease
- congenital heart diseases
- cardiomyopathy
- anemia
- irregular heartbeats (arrhythmias)
- hyperthyroidism
- kidney disease
- infections

In addition to NSAIDS, other drugs such as beta-blockers and calcium channel blockers have also been shown to increase the risk of CHF.

Free Newsletter

Read this first

Archives of Internal Medicine June 2000; 160: 777-784.

COMMENT: Another reason that we need to be careful about avoiding drugs. It is important to recognize here that aspirin, Advil and Motrin type drugs are available without a prescription and may cause these serious side effects. It is possible that the widespread use of these drugs is contributing to the "epidemic" of heart failure going on (see article below). Is the temporary pain relief provided by these medications worth the risk of this life-threatening adverse reaction?

Related Articles:

NSAIDS Reduce Effectiveness of Antihypertensive Therapy

US Experiencing Heart Failure "Epidemic"

NSAIDS Report

Arginine and exercise improve heart failure

NSAIDs May Harm Kidneys of Elderly

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ALLERGIES, INFECTION & STRESS

The following information is abstracted from

Dr. Kenneth Seaton and the book ***Prostate Health in 90 Days*** by Larry Clapp, Ph.D. J.D.

Respiratory infections and allergies constitute the greatest stress on the people in most developed societies. Approximately 80% of visits to doctors offices are for upper respiratory problems. There are two common myths associated with allergies and common respiratory infections that provide strong clues to their management: The average adult has 5 colds per year.

Allergies are not caused by allergens such as pollens. house mites. dust or other such substances. While it is true that these types of things trigger allergic symptoms, the underlying cause is a malfunctioning immune system; "Immunity gone wrong". The allergen simply triggers an incorrect immune system response. In a sense, the immune system identifies the harmless allergen and tries to kill it. The allergic symptoms many of us are so familiar with, runny noses, watery eyes, etc., are direct signs of our immune systems trying to kill these harmless substances. This type of immunological mayhem cannot be separated from the stress put on our immune systems from infections (in many cases sub-clinical infections). Many auto-immune diseases can be viewed as a severe form of allergy where the immune system attacks its owner.

Dr. Seaton's studies over the past years demonstrate that the constant auto-inoculation of the nose and eyes by the fingertip area, leads to an overloaded immune system and most of the infections of the upper respiratory tract. This overload of the immune system through the contact of the fingertips to the eyes and nose is maintained by our congested urban areas, an environment rich in man made toxic substances (pollution) and fast, mass transportation which move microbes from one part of the world to another each day. All of which impacts us each time we touch our fingertips to the sensitive membranes of our eyes and nose, overloading our immune systems.

Germs Are Transferred Through Your Fingernails

When we think of diseases being transferred from person to person, we conjure up images of people sneezing or coughing on each other. We picture germs hurtling out of people's mouths and noses and rocketing through the air right into our own noses or mouths to infect us. A frightening picture, but one that we need not worry about, because relatively few germs become airborne, and fewer still

actually bother us.

Germs aren't hurtled at us by coughs and sneezes. They're handed to us during routine-and intimate -physical contact. Germs are handed to us because human hands-especially under the nails-are a cornucopia of germs. If you take samples from various parts of the hands you'll see that while there are tens, hundreds, thousands of germs on the backs and palms of the hands and on the fingers, there are tens of millions, sometimes hundreds of millions of germs under the fingernails.

The fact that the undersides of the fingernails are a breeding ground and a safe haven for germs is double trouble. First, the fingernails are not protected by the "horny" layer of skin, making it easier for germs to enter the body via the fingernails. Once inside they can get into the many blood vessels that feed the remarkably sensitive fingertips. Second, it is with our fingertips that we commonly touch each other-and ourselves. Fingertips touch flesh when we shake hands, when we caress a lover, when we grab hold of someone's arm to steady them. Fingertips are involved when we hand someone a pencil or a dollar, when we touch a computer keyboard or telephone. Fingertips come into play when we scratch ourselves, floss our teeth, prepare and eat our food.

Once on the hand germs accumulate under and around the fingernails, from where they hitchhike to the damp membranes of the eyes and nose (autoinoculation). Surprising, this is the process by which many germs and allergens affect our metabolism... through the contact of our fingertips to the eyes and nose.

Autoinoculation

The average person touches their nose, mouth, and eyes many times a day. If you watch a group of adults for an hour, you'll find that 1 out of every 3 touches their nose, and 1 in every 2.7 touches their eyes. Even this small amount of innocent touching is enough to carry germs from the mouth, where they were probably unable to penetrate the body's defenses, to the nose or eyes, where they have an easier time causing damage. This passing of germs from one part of your body to another, unknowingly carrying them to the places the germs like the best, is called autoinoculation. But whether the germs come directly from others, arrive indirectly via items we touch, or move from one part of our body to another via autoinoculation, they're bad news.

Lowering Albumin: the "Hidden" Problem with Infection and Disease

If germs entering the body simply did their damage, that would be serious enough. But they unwittingly do far more. In order to fight off invading bacteria, viruses, fungi and other invaders, the body sends the immune system into action. In no time at all, T-cells, B-cells, macrophages, eosinophils, and other immune system soldiers are engaged in battle with the enemy. Here's how poor hygiene can lead to health disasters:

- The battle against germs is a protein-based battle, because the immune system utilizes many protein-based substances to fight off the invasion.
- The immune system rapidly produces more protein-based substances to defend the body.
- There can only be a certain concentration of all proteins in the body.

- When the concentration of immune system proteins goes up, the concentration of other proteins must fall.
- One of the proteins that decreases when the immune system is engaged in battle is albumin, and that's where the trouble lies.

Albumin is an important protein that is found in most animal tissues. Medical doctors know about albumin, and often measure the amounts found in the blood with a simple blood test. Standard blood work-ups include albumin, depicting anything between 3.0 and 5.2 as "normal." Albumin has not been a focus of traditional medicine despite the fact that albumin levels are the single most important indicator of health status. If the level of albumin in your blood drops, your risk of contracting a serious possibly deadly-disease shoots way up. Statistical reports demonstrate that albumin levels correlate closely with age, but are not truly age-related. That is, albumin levels tend to drop as we grow older, but don't necessarily have to.

Albumin is assembled in the liver from more than 500 amino acids. It is the most abundant protein in the bloodstream, and has many important duties. It:

- Protects easily damaged tissues from the free radicals that can destroy cells and cause cancer by altering cellular DNA.
- Guards against heart disease by transporting the antioxidant vitamins that help keep the coronary arteries clean, binding up fatty acids that tend to clog arteries and stabilizing the ratio between HDL ("good" cholesterol) and LDL ("bad" cholesterol).
- Binds up waste products, toxins, and dangerous drugs that would otherwise damage the body and encourage disease. It also detoxifies the fluids surrounding cells.
- Protects the biological terrain by buffering the blood against pH changes.
- Helps to keep the blood flowing smoothly by preventing red blood cells and other substances from clumping together.
- Is essential for transporting vitamins, magnesium, copper, zinc, bilirubin, uric acid, sex hormones, thyroid hormone, other hormones, and fatty acids throughout the body. It regulates the movement of nutrients between the blood and the body's cells.
- Stabilizes red blood cells and growth hormones.
- Plays a major role in controlling the precise amount of water in various bodily tissues.
- Plays an important role in transporting and circulating reservoirs of thyroid hormones.
- Purifies the cerebrospinal fluid, nourishes brain cells, and maintains the blood-brain barrier.
- Helps to ensure that there are adequate amounts of certain key minerals in the bones.
- Binds and transports the "stress hormone" known as cortisol, reducing stress-induced damage to the thymus gland, brain, and connective tissue.

Albumin has been described as a "portable liver" because the liver is the body's chief mechanism for disarming toxins and other dangerous substances, and because albumin, which is made in the liver, does the same throughout the body. It's as if the liver has sent millions of tiny pieces of itself to every single little cell, to round up and destroy harmful substances and organisms.

Albumin plays an indispensable role in maintaining the delicate chemical balance

of the nourishing fluids (interstitial fluids) that surround and support the trillions of cells in the human body. If these fluids are healthy, the cells will flourish. But if the fluids become polluted, or depleted of certain substances, the cells cannot help but fall ill, and disease will sweep through the body. Albumin is like a filter that removes toxins from water, like the net that scoops debris out of a swimming pool, like the dispenser that squirts extra vitamin D into milkalbumin ensures that bodily fluids are clean, filled with nutrients, and properly balanced. And when you're filled with health-giving fluid, you cannot help but be healthy.

When Albumin Levels Fall

Ideally, there should be 5.0 g/d1 (grams per deciliter) of albumin in the blood. (Some doctors and laboratories state this figure as 50 grams per liter, which is the same thing.) Lower levels, around 3.5 or so, are commonly seen in long-time vegetarians and people suffering from malnutrition, kidney disease, cancer, severe infections, Crohn's disease, pancreatitis, and other diseases. However, levels don't have to fall as low as 3.5 before trouble appears.

Low levels of albumin have been linked to all cancers, with the risk of developing cancer rising as albumin falls. Albumin is low in all people suffering from cancer. You can track the progress of the disease by looking at the patient's albumin level-the lower the albumin, the more rampant the cancer. In fact, a low albumin level may actually cause cancer. Albumin normally neutralizes aflatoxin, nitrosamines, and other powerful carcinogens (cancer-causing substances). Without sufficient albumin to keep these and other carcinogens under control, cancer can flourish.

Back in 1775 we learned that at least one form of cancer could be caused by "dirt." Chimney sweeps exposed to soot were more likely to develop scrotal cancer than other people. Simply washing themselves carefully every day reduced their risk to normal. Few of us are anywhere near chimney soot these days, but we are exposed to many airborne chemicals, especially if we live in polluted cities or near certain kinds of factories and plants. We also know that some forms of cancer are caused by viral infections. That's why it's vital that we regularly and carefully wash away any germs or chemicals that can cause cancer, as well as those that can indirectly cause the disease by overwhelming our immune systems and reducing our albumin, allowing cancer to sneak in the "back door."

Declining albumin also indicates an increased risk of heart disease. A long-term British heart study found that a low albumin level was a good predictor of heart disease. Another study stated that the odds of suffering from coronary artery disease doubled when the albumin level fell to 4.4 (that's only about 10% below the ideal level Of 5).

Low levels of albumin are associated with several other diseases, including Hodgkin's disease and HIV, the precursor to AIDS. Indeed, you can use low albumin levels as a "predictor" of mortality. People with albumin levels below 3.5 g/dl are approximately twenty times more likely to die from all causes than those with albumin levels of 5.0 g/dl. Many studies have confirmed this fact. Nursing home residents with albumin levels of 3.5 g/dl had a death rate of about 50%, compared with an 11% rate found in those with higher levels (around 4.0 g/dl). Among men and women over the age of 70, death rates were 40% lower in those with albumin levels of 4.4 g/dl, compared to those with levels of 4.2 g/dl. Hip fractures are a serious problem for the elderly, who often weaken and die

after the break. Among hip-fracture patients whose albumin levels were low (3.0), the death rate was 70%. But among those with albumin levels 4.0, the death rate was only 11%."

Why Does Albumin Fall?

Albumin levels drop when the immune system engages in a battle with invading bacteria, viruses, fungi, and other germs. It also falls when the body is forced to deal with an onslaught of toxins and other dangerous substances that we inhale, drink, or eat, or that get into the body through the skin, respiratory system, or other ports of entry. The fact that the body mounts a powerful offensive is good, for otherwise we would die. But the unintended consequence, the shortfall in albumin, is harmful in the long run.

Temporary drops in albumin levels are necessary, and not a problem. It's as if we miss a credit card payment one month because of unexpected medical expenses, then make the missed payment, with interest, the next month. Our credit takes a small "hit," then quickly recovers before any permanent damage is done. The problem comes when we're continually battling infections, parasites, and toxins. Then our albumin runs low for months on end and, like a person who doesn't make a credit card payment for several months, our "credit" is eventually ruined. Now we're facing serious illness, and we never seem to have enough resources to beat the disease and "get ahead."

Increasing Your Albumin Level

It's important to monitor your albumin level and make sure it stays high, since low albumin has been linked to an increased risk of death from all causes in both sexes and among all races and age groups." The ideal albumin level is 5.0, but the average level in the United States is only about 4.2 -too low for optimal health and low enough to encourage disease. Albumin is produced in the liver, but there's no way to encourage the liver to make more-no drugs, no diet, and no form of exercise that will spur production.

Doctors can infuse albumin into the body, and may do so for patients suffering from cancer and other serious diseases associated with low albumin. Unfortunately, "albumin shots" don't work. When albumin is infused into the body, it upsets the carefully calibrated concentration of proteins (osmotic pressure). The liver attempts to get the concentration back to normal levels by halting its own production of albumin. And if that doesn't work quickly enough, it starts destroying albumin in a frantic attempt to get things back to normal in the body. The liver doesn't understand that the extra albumin may be helpful; it only knows that something is out of balance, and balance must be restored.

The only way to keep albumin levels high is to stay healthy. I know that sounds like circular reasoning-low albumin causes you to be sick, and the only way to increase albumin is to not get sick-but it's not. When we have an infection or have been injured, the total number of antibodies and certain other proteins rise. Since there can only be so many proteins in bodily fluids at one time, if the antibodies and other proteins associated with infection or injury rise, then the albumin must fall. Remaining healthy is the only way to keep the antibodies and other proteins at normal levels, leaving "room" for plenty of albumin.

Fortunately, we can lower the risk of infection by paying scrupulous attention to

personal hygiene. Infection is often caused by the foreign bodies (germs) which enter our body through the mouth, nose, eyes, and fingernails, as well as through cuts in our skin. Good hygiene won't prevent cuts, but it can eliminate many of the germs that are lurking around our eyes, mouth, nose, and fingernails. By preventing the germs from getting into our bodies and causing infections, we keep our antibodies at a normal level, allowing our albumin level to rise.

Other Problems That May Be Helped or Prevented with Good Hygiene

Prostate problems, including cancer, are known to be linked to high testosterone, which is kept in balance by albumin. But it's not just men with prostate cancer who can be helped by proper hygiene. A great many ailments are caused or worsened by poor hygiene and the inevitable drop in albumin, including:

Acne. Bacteria can grow in the sebum that abounds in glands just underneath the skin. Scratching, rubbing, or pinching acne can introduce even more germs to the infected areas.

Aging. Decreased albumin levels are probably the major cause of accelerated aging and contribute to most of the dysfunction we see with increasing age. Decreased albumin levels will increase free cortisol which causes most of the damage.

Allergies and asthma. Many of the substances carried in the fingernails, including staph aureus and worm eggs, can trigger allergic reactions or asthma when we autoinfect ourselves. The facial dips in the hygeine system literally stop most allergic pollen, dust or mold reactions in minutes.

Alzheimers. Immune system dysfunction seems to be the single most important reason we develop progressive dementia.

Athlete's foot. Our fingernails can pick up from our feet the fungi that cause athlete's foot, and then deposit it to other areas of skin, spreading the infection.

Arthritis. Infectious and rheumatoid arthritis can both be caused by invading organisms which can be kept out of the body with proper hygiene.

Candida. A common problem, especially for women, the fungi and yeast that cause candida can produce annoying to serious infections in the vagina, gastrointestinal tract, and elsewhere. There are women who had suffered with chronic vaginal yeast infections (for as long as 20 years) who had their problems disappear in one week on the hygiene program, especially from the baths.

Colds and flus. Although we instinctively flinch when someone with a cold coughs or sneezes on us (or anywhere near us), most of the moisture expelled from the mouth during a cough or sneeze does not contain many germs. That's because cold viruses do not routinely "hang out" in the saliva pools which comprise most of the droplets shot from the mouth by coughing and sneezing. Instead, the viruses congregate in the nose, which has the temperature and other conditions they prefer. Not only do cold viruses favor the nasal environment (the nasal passages are more hospitable for cold viruses), they also have a better chance of getting into the body and its cells via the nose. Since we touch our noses many times during the day, the chance of carrying germs to and from the nose is great.

Diabetes. Recurrent infections are a problem that plagues diabetics. Stopping the infection autoinfection cycle will help many diabetics avoid needless infection and allow their bodies to concentrate on healing.

Measles. Although generally harmless when it strikes in childhood, measles can be a very serious disease for adults. It's very difficult to get people, especially children, not to scratch or touch themselves when infected, raising the likelihood of contagion. Excellent hygiene of the hands, especially the fingernails, may help to keep the disease contained.

Pneumonia. Often called the senior citizen's "best friend" because it leads to a relatively quick and painless death, pneumonia often strikes after or in conjunction with other respiratory ailments. This means that a cold or flu can prepare the way for pneumonia-the same cold and flu that can be prevented with impeccable hygiene.

Worms. Almost everyone plays host to these tiny invaders at some point in their life-and perhaps many times. Worm eggs are easily picked up by our fingernails when we touch various surfaces or other people, and then enter our body via the mouth, nose, or eyes when we touch ourselves. Worms can be draining and debilitating, damaging body tissue and robbing of us nutrients and energy. It's best to wash away worm eggs before they get inside and force us to employ stronger measures to get rid of them.

And that's not all. AIDS, chicken pox, circulatory problems, cystic fibrosis, dandruff, ear and eye infections, fat metabolism disturbances, herpes, multiple sclerosis, sex hormone imbalances, skin diseases of various kinds, and thyroid disorders can also be helped or avoided by good hygiene, which helps prevent germs from entering the body and keeps albumin levels up.

Too Simple to Be True?

This sounds like a simple idea, perhaps too simple to be worthwhile. However, it is no more than an extension of the work of the great Austrian doctor, Ignaz Semmelweis, who created an incredible uproar in the medical community in the 1840s when he insisted that doctors wash their hands before examining women in the maternity ward. (The doctors often came right to the maternity ward from the morgue, where they had been dissecting cadavers, without stopping to wash their hands.) Pregnant women used to plead to be cared for by the midwives rather than doctors, because those who were cared for by the midwives (who did not cut up dead bodies) had a much higher survival rate. But the doctors refused to heed Dr. Semmelweis' plea. They were insulted by the very idea that they might be carrying germs to their patients and killing them. So they drove Dr. Semmelweis out of the hospital and into the insane asylum, where he died.

Yes, washing your hands sounds like a very simple solution to an incredibly complex set of problems, but the simplest ideas are often the best. Sometimes changing just one thing produces dramatic results. Scurvy used to decimate the crews of ships sailing the high seas in the 15 , 16th, and 17th centuries. The problem was solved when sailors began eating citrus fruits or drinking lemon or lime juice. Pellagra was a terrible scourge for many centuries in various countries. It attacked many in the United States, leading to the "Four D's": diarrhea, dermatitis, dementia, and death. This mysterious and impossibly

complex disease was eradicated by adding niacin, one of the B vitamins, to the diet. A solution doesn't have to be complex. It simply has to work. And good hygiene works!

The Solution

As is often the case in Nature, the solution is astonishingly simple and reliable! Advanced personal hygiene of the fingertip area, especially under and around the fingernails, and by the daily cleansing of membranes in the front of the nose and around the eyes can remove the overload from our immune systems and reduce the number of clinical and sub-clinical infections of the upper respiratory tract to levels never before thought possible. This non-invasive, non-pharmaceutical solution is surprising simple and effective. Advanced hygiene is a more scientific approach to washing that keeps germs, environmental toxins and allergens away from our bodies before they can cause any stress or damage to our systems.

Further, once the overload is removed from our immune systems, many allergies are self correcting. Not only does advanced personal hygiene reduce the amount of auto-inoculation of allergens, but once the overload is removed from our immune systems, the allergen that used to trigger allergic responses no longer causes immunological mayhem to our systems. The allergy is gone. Even though people may have different genetic predisposition's to allergies, stress and frequency of illness, it is an individuals standard of hygiene that determines the differences between people, not differences such as race, social or income levels.

The Skin

The skin is not just a covering over our bodies. It is an organ, like the heart, liver or brain. In fact, it is the largest organ in the body, and one of the most complex. How would you wash your heart, liver or brain each day? In reality, the skin is an endocrine organ, part of our immune systems. It is a vital and integral part of the immune system itself T cells, the master cells of the immune system, after passing through the thymus, migrate to the skin's surface where, amazingly, they receive maturation hormones from skin cells. Even tiny traces of chemicals can interfere with the skin cell's ability to produce these complex hormones which are vital for maintaining the immune system throughout our lives.

Fingertips

The sense of touch and blood flow and sensitivity of the fingertips is so extraordinary that a blind person can read by feeling. The fingertips are a real extension of our brain. Dolphins, with very large brains, can never build any civilization or even weapons to protect themselves.

-Our hands, with their opposable thumbs, are perhaps the most remarkable design in all of nature.

The area under the fingernail has no dead horny skin layer and is very thin. The blood flow to the nail bed is about the most complex of the entire body. Constant use of chemicals under and around the fingernails can cause those chemicals to be absorbed directly into the blood stream. One dangerous side effect of constantly applying antiseptics/disinfectants to the fingertips is the introduction of chemicals into the delicate tissues of the eyes, nose, skin, hair and food through touch.

Further, the skin, particularly of the fingernail area, is teeming with a wide variety of germs, something like the natural flora of the rain forests in South America. These germs should be in perfect balance, competing and producing complex chemicals that prevent colonization by pathogenic microbes. Amazingly, many people become sick because they do not have enough of the correct type of germs on their skin. We call this the "natural disinfecting power" of the skin. Natural oils, waxes and secretions should be on the skin and perhaps are vital in the production of the hormones that cause T cells to mature.

Antiseptics, Disinfectants & Antibacterial Agents

Over the last 15 years, Dr. Seaton formulated and tested approximately 5,000 different soaps. For years he tried every type of antiseptic/disinfectant/ antibacterial agents available. They ALL failed for the following reasons: (1) Most are not effective against ALL types of germs, thus some overgrow. (2) The combination of several different types of these is highly toxic, and causes stress for the immune system. (3) ALL chemicals that kill germs will kill people, if used in sufficient concentrations or over many years. (4) These chemicals disturb the natural balance of healthy microflora. (5) Over a period of time, all substances applied to the skin are absorbed through it and affect the body's internal metabolism and (6) the emulsion of the soap, when tiny traces are introduced into the eyes and nose, sends the immune system into disarray and contaminates the delicate tissues.

Further, there is grave concern in the medical community about the overuse of antibiotic medications, which has given rise to all sorts of antibiotic-resistant bacteria and deadly new infections. Now the overuse of antibacterial soaps is causing us similar concern. Our common skin flora includes a wide range of germs that are normally harmless; in fact, they compete with disease-causing microorganisms and in many cases provide a natural immunity against disease. Too many germs may pose a threat; too few, and we have an artificially sterile biological ecosystem in which virulent new organisms may take hold and flourish.

High Performance Hygiene Facial and Body Soap, by trial and error, enormous research and scientific design, does NOT disturb the natural disinfecting power of the skin, does NOT disturb the immunologic function of skin cells producing these complex hormones and is NOT toxic, no matter how many times it is used, even on a newborn baby. Dr. Seaton is constantly striving to improve his product, especially in its emulsion so it does not stress the immune system's ability to recognize self from non-self.

His High Performance Hygiene soap is a natural product. That means it is NOT made from petroleum products. Many soaps today are. Many manufactures use the carbon chain from petroleum products to develop a hydrophobic and hydrophilic carbon chain. For example; many manufacturers of consumer soaps use a type of ingredient similar to brake fluid called Diethylene Glycol or Triethylene Glycol. These substances are used to give the soap a smooth non-drying look, however, they are completely unnatural to the skin. These detergent-type soaps made from petroleum products are completely unnatural and remove the essential skin lipids. It is important to note: The skin is an organ, like the heart and the liver. Ask yourself the questions: "Would you rub brake fluid all over your liver everyday for 50 years?" "Would you have a healthy liver? "

An important test for a soap is: Can you eat a quart of it with no effect? There is a scale of poisons normally used in the pharmaceutical industry. For example; Nicotine, heroin and cyanide are S6, this means only a few drops can kill. A natural soap should be S1, practically non-toxic, because no-one can eat enough of it to cause any problems. The High Performance Hygiene soap is so safe, that many people have reported to Dr. Seaton that their cats or dogs have eaten half a tub. I tell them not to worry. It will do them more good than harm. I have to make certain that my dog doesn't get near the soap because he tends to eat it as well. Fortunately, I keep my dog outside. Animals have remarkable sense of smell and taste and seem to know what is poisonous and what is not.

As many do, making soap with Diethylene, Ethylene, Triethylene Glycol, or even Propylene Glycol is completely against common sense. To give some idea of the side effects the world's most prestigious Pharmacopoeia (Martingale, 28th ed.), published by direction of The Council of the Pharmaceutical Society of Great Britain states the following about these chemicals:

'Diethylene and Triethylene Glycol; A colorless almost odorless hydroscopic syrupy liquid with a sharp sweet taste. The toxic effect following ingestion by mouth are; depression of the central nervous system, degenerative changes in the kidneys and liver. The symptoms and effects are those similar to those produced by Ethylene Glycol intoxication. Seven children, aged 6 to 31 months, died following ingestion of a preparation in which the solvent was found to be Diethylene Glycol.'

Because the skin absorbs most substances that are applied to it, the soap maker needs to pay careful attention to these matters. This is particularly important when substances are applied under and around the fingernails where the skin is very thin and has no dead skin horny layer and a very high blood flow. In addition, whatever you apply to the fingertips ends up getting into the eyes and nose because of the astonishing frequency of self-inoculation (the touching of the fingertips to the eyes and nose). Further, the skin must contain the oils, fats, waxes and enzymes plus germs that were designed by Nature. A soap not only must clean the skin, it must replace the oils that are washed away. It must replace the oils as close to those designed by nature. The genius learns from nature.

The soap chemist must have a knowledge in immunology, biochemistry, dermatology, physiology, as well as chemistry. Dr. Seaton spent over 16 years in full time research attempting to make the world's most scientific soap. His efforts resulted in a revolutionary Tub Soap. It is the easiest way to clean the fingernails, hands, skin and hair. Young children and old people cannot handle a bar of soap. The bar of soap and the liquid pump are totally useless In cleaning under and around the fingernail area in all ages, which is the most important area with the highest concentration of germs. Iif there are 10 million germs on the hands, it is estimated that 9.9 million are under and around the fingernails. The bar or liquid soap totally concentrates on cleaning the palm and back of the hands. This is equivalent to hunting crocodiles in the Sahara desert. There are none there! Under and around the fingernails where the germs are is equivalent to the Nile valley where all the crocodiles can be found.

Further, because we are an animal and have animal fats and oils on our skin, it is impossible to make a soap from pure vegetable oils that is going to restore the natural skin lipids, and ensure that the natural disinfecting power of the skin

remains in tact. It is essential to make soap from a combination of animal and vegetable oils. Many native races living under natural conditions have perfect skin, and use special soaps derived from plants and animal fats. Remember, you are as old as your skin! You areas attractive as your skin! Your immune system Is as good as your skin! Your health can be determined by your skin, and you are recognized by those who know you by your skin.

Instructions for the Hygeine System

Throughout history, people living in cleaner societies have had higher albumin levels and longer lives. Dirty fingernails, which are indicative of overall cleanliness, have long been associated with lower albumin levels. Cleanliness is certainly a path to better health and longer life! Regular washing of the hands and face-at least five times a day-is an excellent start, but it's only a beginning. Careful scrubbing for a full minute, which few of us do, can remove up to 90% of the germs from most of the body, but not the fingernails. Neither will regular hand and face washing eliminate the germs hiding in the nose and eyes. And scrubbing with regular soap is not enough. In fact, washing with some soaps can actually increase the numbers of germs. Something more is needed.

Here are the instructions for using the various soaps in the hygeine system:

Overall Hygeine

1. Try to use a bath and not a shower. You could use however shower in the morning but it would be best to have a bath at night. When you take your bath but one half to one full cup of salt in the tub. This will help clean the genital tract, rectum and skin. If you do not have well water and use municipal water it is likely your water has chlorine in it. In this case it would be best to fill your tub from the shower head after you have attached a chlorine filter.

2. If you are a man NEVER insert your fingers into a woman's vagina unless your fingernails are clean with this system. If you are a female do not allow digit penetration of your vagina. If you break this rule you will greatly increase your risk for acquiring a vaginal infection.

3. Do NOT bite your nails. This habit should be stopped immediately! One can go to any pharmacy and obtain a solution that is used to help baby's from sucking their thumbs. It is non toxic and will work in days to stop you from biting your nails.

4. If you are a woman with long nails cut them to regular size. It will be virtually impossible to clean your nails well if they are very long. The other, perhaps more important issue, is that long nails prevent stimulation of the fingertips. If this happens for a prolonged time you will actually get brain atrophy. The brain requires regular stimulation from the fingertips to stay healthy.

5. Try to get to bed by 9 PM (10 PM at the latest). This will maximize your peak cortisol levels.

Hand Soap and Facial Dips

1. This system of hygiene replaces bar, liquid, and hair soaps, as well as skin creams. You must follow instructions exactly to get the results.

2. If the soap dries out, put the lid back on.

3. Hand washing. It is essential to prod the thumbs and fingernails (with dry hands) into the soap several times for approximately three seconds. Make certain to stick your fingers so that the nails is completely covered with the soap. It is also helpful to rotate the fingers to make certain the soaps is inserted on the sides of the nail. Use the excess soap clinging to the fingertips to rub all over the rest of the hands. Then wet, create lather, and rinse. Wash hands at least five times a day.

4. Skin cream: These soaps contain their own unequaled moisturizer. For best results do not use any skin cream. If necessary, we recommend sunflower oil.

5. Face washing. Massage the soap into the skin, lather, then rinse. For those with acne or other skin problems, use frequently. The facial dip is explained below.

6. Bath/shower. Use the sponge supplied. Wet both body and the sponge. Then turn off water and create a rich lather using the sponge and soap. Rinse.

7. Hair. Wet hair, use plenty of soap, massage into the scalp for 30 seconds, then rinse. Use a conditioner only if necessary.

8. Facial Dip A & C. Use Facial Dip A (hydrogen peroxide/zinc/magnesium) most of the time, preferably first thing in the morning, because it's very soothing to the nasal passageway and eyes. Use Facial Dip C (iodine/HOI) especially at the first sign of any infection (usually if Facial Dip A isn't working). It is safe to use up to 4 half-eyedroppers of Facial Dip C during infections such as colds, sore throats, or sinus problems. Do not use Facial Dip C every day month after month.

With Facial Dip A, use a capful in a normal hand basin containing 5 to 8 quarts of very warm water. With Facial Dip C use between 1 and 4 half-eyedroppers in 5 to 8 quarts of very warm water. It is essential to add 1 to.2 tablespoons of salt (sodium chloride) at least 80% of the time, yet not all the time. Do not mix Facial Dip A and C together. Use the hand basin, not a separate bowl. (The bacteria in the hand basin are essential to activate the process.) Mix the solution with running water, never your fingers. Do not draw the water to the back of the throat. Simply allow the first two inches of the nasal passageway to be cleaned.

10. Bath: Add half a pound of table salt, Plus 5 halfeyedroppers of Facial Dip C when you are actually in the water. Supervise young children. This bath is good for very young children who cannot perform the facial dip and to clean the genital tract in males and females. Do not add any soap for lo minutes because it deactivates the iodine.

11. Foot soak. Add 4 to 6 half-eyedroppers of Facial Dip C to one gallon of hot water, plus a tablespoon of salt. Soak feet for 10 minutes, particularly if you have problems or fungal infections.

12. Vitamin supplement. This system works very well with 1,000 mcg (not sublingual) of vitamin B12, but not with multivitamins. It is a good idea to fast one day a week, perform sensible exercise, and go to bed early, around 8 p.m., about twice a week.

13. Please note: Cleaning the fingernails, nasal passageway, and eyes is very complex. Do not use any other chemicals. Follow these instructions exactly.

Dr. Seaton's soap can be obtained in our office at a reduced rate or you can contact the distributor directly.

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THE HIDDEN HAZARDS OF MICROWAVE COOKING

by Anthony Wayne and Lawrence Newell

Is it possible that millions of people are ignorantly sacrificing their health in exchange for the convenience of microwave ovens? Why did the Soviet Union ban the use of microwave ovens in 1976? Who invented microwave ovens, and why? The answers to these questions may shock you into throwing your microwave oven in the trash.

Over 90% of American homes have microwave ovens used for meal preparation. Because microwave ovens are so convenient and energy efficient, as compared to conventional ovens, very few homes or restaurants are without them. In general, people believe that whatever a microwave oven does to foods cooked in it doesn't have any negative effect on either the food or them. Of course, if microwave ovens were really harmful, our government would never allow them on the market, would they? Would they? Regardless of what has been "officially" released concerning microwave ovens, we have personally stopped using ours based on the research facts outlined in this article.

The purpose of this report is to show proof - evidence - that microwave cooking is not natural, nor healthy, and is far more dangerous to the human body than anyone could imagine. However, the microwave oven manufacturers, Washington City politics, and plain old human nature are suppressing the facts and evidence. Because of this, people are continuing to microwave their food - in blissful ignorance - without knowing the effects and danger of doing so.

How do microwave ovens work?
Microwaves are a form of electromagnetic energy, like light waves or radio waves, and occupy a part of the electromagnetic spectrum of power, or energy. Microwaves are very short waves of electromagnetic energy that travel at the speed of light (186,282 miles per second). In our modern technological age, microwaves are used to relay long distance telephone signals, television programs, and computer information across the earth or to a satellite in space. But the microwave is most familiar to us as an energy source for cooking food.

Every microwave oven contains a magnetron, a tube in which electrons are affected by magnetic and electric fields in such a way as to produce micro wavelength radiation at about 2450 Mega Hertz (MHz) or 2.45 Giga Hertz (GHz). This microwave radiation interacts with the molecules in food. All wave energy changes polarity from positive to negative with each cycle of the wave. In microwaves, these polarity changes happen millions of times every second. Food molecules - especially the molecules of water - have a positive and negative end in the same way a magnet has a north and a south polarity.

In commercial models, the oven has a power input of about 1000 watts of alternating current. As these microwaves generated from the magnetron bombard the food, they cause the polar molecules to rotate at the same frequency millions of times a second. All this agitation creates molecular friction, which heats up the food. The friction also causes substantial damage to the surrounding molecules, often tearing them apart or

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forcefully deforming them. The scientific name for this deformation is "structural isomerism".

By comparison, microwaves from the sun are based on principles of pulsed direct current (DC) that don't create frictional heat; microwave ovens use alternating current (AC) creating frictional heat. A microwave oven produces a spiked wavelength of energy with all the power going into only one narrow frequency of the energy spectrum. Energy from the sun operates in a wide frequency spectrum.

Many terms are used in describing electromagnetic waves, such as wavelength, amplitude, cycle and frequency:

Wavelength determines the type of radiation, i.e. radio, X-ray, ultraviolet, visible, infrared, etc.
Amplitude determines the extent of movement measured from the starting point.
Cycle determines the unit of frequency, such as cycles per second, Hertz, Hz, or cycles/second.
Frequency determines the number of occurrences within a given time period (usually 1 second); The number of occurrences of a recurring process per unit of time, i.e. the number of repetitions of cycles per second.
Radiation = spreading energy with electromagnetic waves

Radiation, as defined by physics terminology, is "the electromagnetic waves emitted by the atoms and molecules of a radioactive substance as a result of nuclear decay." Radiation causes ionization, which is what occurs when a neutral atom gains or loses electrons. In simpler terms, a microwave oven decays and changes the molecular structure of the food by the process of radiation. Had the manufacturers accurately called them "radiation ovens", it's doubtful they would have ever sold one, but that's exactly what a microwave oven is.

We've all been told that microwaving food is not the same as irradiating it (radiation "treatment"). The two processes are supposed to use completely different waves of energy and at different intensities. No FDA or officially released government studies have proven current microwaving usage to be harmful, but we all know that the validity of studies can be - and are sometimes deliberately - limiting. Many of these studies are later proven to be inaccurate. As consumers, we're supposed to have a certain degree of common sense to use in judgment.

Take the example of eggs and how they were "proven" to be so harmful to our health in the late 1960's. This brought about imitation egg products and big profits for the manufacturers, while egg farms went broke. Now, recent government sponsored studies are saying that eggs are not bad for us after all. So, whom should we believe and what criteria should we use to decide matters concerning our health? Since it's currently published that microwaves - purportedly - don't leak into the environment, when properly used and with approved design, the decision lies with each consumer as to whether or not you choose to eat food heated by a microwave oven or even purchase one in the first place.

Motherly instincts are right

On a more humorous side, the "sixth sense" every mother has is impossible to argue with. Have you ever tried it? Children will never win against a mother's intuition. It's like trying to argue with the arm - appearing out of nowhere - that pinned you to the back of the seat when your mother slammed on the brakes.

Many of us come from a generation where mothers and grandmothers have distrusted the modern "inside out" cooking they claimed was "not suitable" for most foods. My mother refused to even try baking anything in a microwave. She also didn't like the way a cup of coffee tasted when heated in a microwave oven. I have to fully agree and can't argue either fact. Her own common sense and instincts told her that there was no way microwave cooking could be natural nor make foods "taste they way they're supposed to". Reluctantly, even my mother succumbed to re-heating leftovers in a microwave due to her work schedule before she retired.

Many others feel the same way, but they're considered an "old fashioned" minority dating back to before the 1970's when microwaves first overwhelmed the market. Like most young adults at the time, as microwave ovens became commonplace, I chose to ignore my mother's intuitive wisdom and joined the majority who believed microwave cooking was far too convenient to ever believe anything could be wrong with it. Chalk one up for mom's perception, because even though she didn't know the scientific, technical, or health reasons why, she just knew that microwave ovens were not good based on how foods tasted when they were cooked in them. She didn't like the way the texture of the microwaved food changed either.

Microwaves unsafe for baby's milk

A number of warnings have been made public, but have been barely noticed. For example, Young Families, the Minnesota Extension Service of the University of Minnesota, published the following in 1989:

"Although microwaves heat food quickly, they are not recommended for heating a baby's bottle. The bottle may seem cool to the touch, but the liquid inside may become extremely hot and could burn the baby's mouth and throat. Also, the buildup of steam in a closed container, such as a baby bottle, could cause it to explode. Heating the bottle in a microwave can cause slight changes in the milk. In infant formulas, there may be a loss of some vitamins. In expressed breast milk, some protective properties may be destroyed. Warming a bottle by holding it under tap water, or by setting it in a bowl of warm water, then testing it on your wrist before feeding may take a few minutes longer, but it is much safer."

Dr. Lita Lee of Hawaii reported in the December 9, 1989 Lancet:

"Microwaving baby formulas converted certain trans-amino acids into their synthetic cis-isomers. Synthetic isomers, whether cis-amino acids or trans-fatty acids, are not biologically active. Further, one of the amino acids, L-proline, was converted to its d-isomer, which is known to be neurotoxic (poisonous to the nervous system) and nephrotoxic (poisonous to the kidneys). It's bad enough that many babies are not nursed, but now they are given fake milk (baby formula) made even more toxic via microwaving."

Microwaved blood kills patient

In 1991, there was a lawsuit in Oklahoma concerning the hospital use of a microwave oven to warm blood needed in a transfusion. The case involved a hip surgery patient, Norma Levitt, who died from a simple blood transfusion. It seems the nurse had warmed the blood in a microwave oven. This tragedy makes it very apparent that there's much more to "heating" with microwaves than we've been led to believe. Blood for transfusions is routinely warmed, but not in microwave ovens. In the case of Mrs. Levitt, the microwaving altered the blood and it killed her.

It's very obvious that this form of microwave radiation "heating" does something to the substances it heats. It's also becoming quite apparent that people who process food in a microwave oven are also ingesting these "unknowns".

Because the body is electrochemical in nature, any force that disrupts or changes human electrochemical events will affect the physiology of the body. This is further described in Robert O. Becker's book, The Body Electric, and in Ellen Sugarman's book, Warning, the Electricity Around You May Be Hazardous to Your Health.

Scientific evidence and facts

In Comparative Study of Food Prepared Conventionally and in the Microwave Oven, published by Raum & Zelt in 1992, at 3(2): 43, it states

"A basic hypothesis of natural medicine states that the introduction into the human body of molecules and energies, to which it is not accustomed, is much more likely to cause harm than good. Microwaved food contains both molecules and energies not present in food cooked in the way humans have been cooking food since the discovery of fire. Microwave energy from the sun and other stars is direct current based. Artificially produced microwaves, including those in ovens, are produced from alternating current and force a billion or more polarity reversals per second in every food molecule they hit. Production of unnatural molecules is inevitable. Naturally occurring amino acids have been observed to undergo isomeric changes (changes in shape morphing) as well as transformation into toxic forms, under the impact of microwaves produced in ovens.

One short-term study found significant and disturbing changes in the blood of individuals consuming microwaved milk and vegetables. Eight volunteers ate various combinations of the same foods cooked different ways. All foods that were processed through the microwave ovens caused changes in the blood of the volunteers. Hemoglobin levels decreased and over all white cell levels and cholesterol levels increased. Lymphocytes decreased.

Luminescent (light-emitting) bacteria were employed to detect energetic changes in the blood. Significant increases were found in the luminescence of these bacteria when exposed to blood serum obtained after the consumption of microwaved food."

The Swiss clinical study

Dr. Hans Ulrich Hertel, who is now retired, worked as a food scientist for many years with one of the major Swiss food companies that do business on a global scale. A few years ago, he was fired from his job for questioning certain processing procedures that denatured the food.

In 1991, he and a Lausanne University professor published a research paper indicating that food cooked in microwave ovens could pose a greater risk to health than food cooked by conventional means. An article also appeared in issue 19 of the Journal Franz Weber in which it was stated that the consumption of food cooked in microwave ovens had cancerous effects on the blood. The research paper itself followed the article. On the cover of the magazine there was a picture of the Grim Reaper holding a microwave oven in one of his hands.

Dr. Hertel was the first scientist to conceive and carry out a quality clinical study on the effects microwaved nutrients have on the blood and physiology of the human body. His small but well controlled study showed the degenerative force produced in microwave ovens and the food processed in them. The scientific conclusion showed that microwave cooking changed the nutrients in the food; and, changes took place in the participants' blood that could cause deterioration in the human system. Hertel's scientific study was done along with Dr. Bernard H. Blanc of the Swiss Federal Institute of Technology and the University Institute for Biochemistry.

In intervals of two to five days, the volunteers in the study received one of the following food variants on an empty stomach: (1) raw milk, (2) the same milk conventionally cooked; (3) pasteurized milk; (4) the same raw milks cooked in a microwave oven; (5) raw vegetables from an organic farm; (6) the same vegetables cooked conventionally; (7) the same vegetables frozen and defrosted in a microwave oven; and (8) the same vegetables cooked in the microwave oven. Once the volunteers were isolated, blood samples were taken from every volunteer immediately before eating. Then, blood samples were taken at defined intervals after eating from the above milk or vegetable preparations.

Significant changes were discovered in the blood samples from the intervals following the foods cooked in the microwave oven. These changes included a decrease in all hemoglobin and cholesterol values, especially the ratio of HDL (good cholesterol) and LDL (bad cholesterol) values. Lymphocytes (white blood cells) showed a more distinct short-term decrease following the intake of microwaved food than after the intake of all the other variants. Each of these indicators pointed to degeneration. Additionally, there was a highly significant association between the amount of microwave energy in the test foods and the luminous power of luminescent bacteria exposed to serum from test persons who ate that food. This led Dr. Hertel to the conclusion that such technically derived energies may, indeed, be passed along to man inductively via eating microwaved food.

According to Dr. Hertel,

"Leukocytosis, which cannot be accounted for by normal daily deviations, is taken very seriously by hemotologists. Leukocytes are often signs of

pathogenic effects on the living system, such as poisoning and cell damage. The increase of leukocytes with the microwaved foods were more pronounced than with all the other variants. It appears that these marked increases were caused entirely by ingesting the microwaved substances.

This process is based on physical principles and has already been confirmed in the literature. The apparent additional energy exhibited by the luminescent bacteria was merely an extra confirmation. There is extensive scientific literature concerning the hazardous effects of direct microwave radiation on living systems. It is astonishing, therefore, to realize how little effort has been taken to replace this detrimental technique of microwaves with technology more in accordance with nature. Technically produced microwaves are based on the principle of alternating current. Atoms, molecules, and cells hit by this hard electromagnetic radiation are forced to reverse polarity 1-100 billion times a second. There are no atoms, molecules or cells of any organic system able to withstand such a violent, destructive power for any extended period of time, not even in the low energy range of milliwatts.

Of all the natural substances - which are polar - the oxygen of water molecules reacts most sensitively. This is how microwave cooking heat is generated - friction from this violence in water molecules. Structures of molecules are torn apart, molecules are forcefully deformed, called structural isomerism, and thus become impaired in quality. This is contrary to conventional heating of food where heat transfers convectionally from without to within. Cooking by microwaves begins within the cells and molecules where water is present and where the energy is transformed into frictional heat.

In addition to the violent frictional heat effects, called thermic effects, there are also athermic effects which have hardly ever been taken into account. These athermic effects are not presently measurable, but they can also deform the structures of molecules and have qualitative consequences. For example the weakening of cell membranes by microwaves is used in the field of gene altering technology. Because of the force involved, the cells are actually broken, thereby neutralizing the electrical potentials, the very life of the cells, between the outer and inner side of the cell membranes. Impaired cells become easy prey for viruses, fungi and other microorganisms. The natural repair mechanisms are suppressed and cells are forced to adapt to a state of energy emergency - they switch from aerobic to anaerobic respiration. Instead of water and carbon dioxide, the cell poisons hydrogen peroxide and carbon monoxide are produced."

The same violent deformations that occur in our bodies, when we are directly exposed to radar or microwaves, also occur in the molecules of foods cooked in a microwave oven. This radiation results in the destruction and deformation of food molecules. Microwaving also creates new compounds, called radiolytic compounds, which are unknown fusions not found in nature. Radiolytic compounds are created by molecular decomposition - decay - as a direct result of radiation.

Microwave oven manufacturers insist that microwaved and irradiated foods do not have any significantly higher radiolytic compounds than do broiled, baked or other conventionally cooked foods. The scientific clinical evidence presented here has shown that this is simply a lie. In

America, neither universities nor the federal government have conducted any tests concerning the effects on our bodies from eating microwaved foods. Isn't that a bit odd? They're more concerned with studies on what happens if the door on a microwave oven doesn't close properly. Once again, common sense tells us that their attention should be centered on what happens to food cooked inside a microwave oven. Since people ingest this altered food, shouldn't there be concern for how the same decayed molecules will affect our own human biological cell structure?

Industry's action to hide the truth

As soon as Doctors Hertel and Blanc published their results, the authorities reacted. A powerful trade organization, the Swiss Association of Dealers for Electro-apparatuses for Households and Industry, known as FEA, struck swiftly in 1992. They forced the President of the Court of Seftigen, Canton of Bern, to issue a "gag order" against Drs. Hertel and Blanc. In March 1993, Dr. Hertel was convicted for "interfering with commerce" and prohibited from further publishing his results. However, Dr. Hertel stood his ground and fought this decision over the years.

Not long ago, this decision was reversed in a judgment delivered in Strasbourg, Austria, on August 25, 1998. The European Court of Human Rights held that there had been a violation of Hertel's rights in the 1993 decision. The European Court of Human Rights also ruled that the "gag order" issued by the Swiss court in 1992 against Dr. Hertel, prohibiting him from declaring that microwave ovens are dangerous to human health, was contrary to the right to freedom of expression. In addition, Switzerland was ordered to pay Dr. Hertel compensation.

Who invented microwave ovens?

The Nazis, for use in their mobile support operations, originally developed microwave "radiomissor" cooking ovens to be used for the invasion of Russia. By being able to utilize electronic equipment for preparation of meals on a mass scale, the logistical problem of cooking fuels would have been eliminated, as well as the convenience of producing edible products in a greatly reduced time-factor.

After the war, the Allies discovered medical research done by the Germans on microwave ovens. These documents, along with some working microwave ovens, were transferred to the United States War Department and classified for reference and "further scientific investigation." The Russians had also retrieved some microwave ovens and now have thorough research on their biological effects. As a result, their use was outlawed in the Soviet Union. The Soviets issued an international warning on the health hazards, both biological and environmental, of microwave ovens and similar frequency electronic devices.

Other Eastern European scientists also reported the harmful effects of microwave radiation and set up strict environmental limits for their usage. The United States has not accepted the European reports of harmful effects, even though the EPA estimates that radio frequency and microwave radiation sources in America are increasing at 15% per year.

Carcinogens in microwaved food

In Dr. Lita Lee's book, Health Effects of Microwave Radiation - Microwave Ovens, and in the March and September 1991 issues of Earthletter, she stated that every microwave oven leaks electro-magnetic radiation, harms food, and converts substances cooked in it to dangerous organ-toxic and carcinogenic products. Further research summarized in this article reveal that microwave ovens are far more harmful than previously imagined.

The following is a summary of the Russian investigations published by the Atlantis Raising Educational Center in Portland, Oregon. Carcinogens were formed in virtually all foods tested. No test food was subjected to more microwaving than necessary to accomplish the purpose, i.e., cooking, thawing, or heating to insure sanitary ingestion. Here's a summary of some of the results:

Microwaving prepared meats sufficiently to insure sanitary ingestion caused formation of d-Nitrosodienthanolamines, a well-known carcinogen.
Microwaving milk and cereal grains converted some of their amino acids into carcinogens.
Thawing frozen fruits converted their glucoside and galactoside containing fractions into carcinogenic substances.
Extremely short exposure of raw, cooked or frozen vegetables converted their plant alkaloids into carcinogens.
Carcinogenic free radicals were formed in microwaved plants, especially root vegetables.
Decrease in nutritional value
Russian researchers also reported a marked acceleration of structural degradation leading to a decreased food value of 60 to 90% in all foods tested. Among the changes observed were:
Deceased bio-availability of vitamin B complex, vitamin C, vitamin E, essential minerals and lipotropics factors in all food tested.
Various kinds of damaged to many plant substances, such as alkaloids, glucosides, galactosides and nitrilosides.
The degradation of nucleo-proteins in meats.

Microwave sickness is discovered

The Russians did research on thousands of workers who had been exposed to microwaves during the development of radar in the 1950's. Their research showed health problems so serious that the Russians set strict limits of 10 microwatts exposure for workers and one microwatt for civilians.

In Robert O. Becker's book, The Body Electric, he described Russian research on the health effects of microwave radiation, which they called "microwave sickness." On page 314, Becker states:

"It's [Microwave sickness] first signs are low blood pressure and slow pulse. The later and most common manifestations are chronic excitation of the sympathetic nervous system [stress syndrome] and high blood pressure. This phase also often includes headache, dizziness, eye pain, sleeplessness, irritability, anxiety, stomach pain, nervous tension, inability to concentrate, hair loss, plus an increased incidence of appendicitis, cataracts, reproductive problems, and cancer. The chronic symptoms are

eventually succeeded by crisis of adrenal exhaustion and ischemic heart disease [the blockage of coronary arteries and heart attacks]."

According to Dr. Lee, changes are observed in the blood chemistries and the rates of certain diseases among consumers of microwaved foods. The symptoms above can easily be caused by the observations shown below. The following is a sample of these changes:

Lymphatic disorders were observed, leading to decreased ability to prevent certain types of cancers.
An increased rate of cancer cell formation was observed in the blood.
Increased rates of stomach and intestinal cancers were observed.
Higher rates of digestive disorders and a gradual breakdown of the systems of elimination were observed.

Microwave research conclusions
The following were the most significant German and Russian research operations facilities concerning the biological effects of microwaves:

The initial research conducted by the Germans during the Barbarossa military campaign, at the Humbolt-Universitat zu Berlin (1942-1943); and,

From 1957 and up to the present [until the end of the cold war], the Russian research operations were conducted at: the Institute of Radio Technology at Kinsk, Byelorussian Autonomous Region; and, at the Institute of Radio Technology at Rajasthan in the Rossiskaja Autonomous Region, both in the Union of the Soviet Socialist Republics.

In most cases, the foods used for research analysis were exposed to microwave propagation at an energy potential of 100 kilowatts/cm3/second, to the point considered acceptable for sanitary, normal ingestion. The effects noted by both German and Russian researchers is presented in three categories:

Category I, Cancer-Causing Effects
Category II, Nutritive Destruction of Foods
Category III, Biological Effects of Exposure

CATEGORY I
CANCER-CAUSING EFFECTS

[The first two points of Category I are not readable from our report copy. The remainder of the report is intact.]

3. Creation of a "binding effect" to radioactivity in the atmosphere, thus causing a marked increase in the amount of alpha and beta particle saturation in foods;

4. Creation of cancer causing agents within protein hydrolysate compounds* in milk and cereal grains [*these are natural proteins that are split into unnatural fragments by the addition of water];

5. Alteration of elemental food-substances, causing disorders in the digestive system by unstable catabolism* of foods subjected to microwaves [*the metabolic breakdown process];

6. Due to chemical alterations within food substances, malfunctions were observed within the lymphatic systems [absorbent vessels], causing a degeneration of the immune potentials of the body to protect against certain forms of neoplastics [abnormal growths of tissue];

7. Ingestion of microwaved foods caused a higher percentage of cancerous cells within the blood serum [cytomas - cell tumors such as sarcoma];

8. Microwave emissions caused alteration in the catabolic [metabolic breakdown] behavior of glucoside [hydrolyzed dextrose] and galactoside [oxidized alcohol] elements within frozen fruits when thawed in this manner;

9. Microwave emission caused alteration of the catabolic [metabolic breakdown] behavior of plant alkaloids [organic nitrogen based elements] when raw, cooked, or frozen vegetables were exposed for even extremely short durations;

10. Cancer causing free radicals [highly reactive incomplete molecules] were formed within certain trace mineral molecular formations in plant substances, and in particular, raw root-vegetables; and,

11. In a statistically high percentage of persons, microwaved foods caused stomach and intestinal cancerous growths, as well as a general degeneration of peripheral cellular tissues, with a gradual breakdown of the function of the digestive and excretive systems.

CATEGORY II

DECREASE IN FOOD VALUE

Microwave exposure caused significant decreases in the nutritive value of all foods researched. The following are the most important findings:

1. A decrease in the bioavailability [capability of the body to utilize the nutriment] of B-complex vitamins, Vitamin C, Vitamin E, essential minerals and lipotropics in all foods;

2. A loss of 60-90% of the vital energy field content of all tested foods;

3. A reduction in the metabolic behavior and integration process capability of alkaloids [organic nitrogen based elements], glucosides and galactosides, and nitrilosides;

4. A destruction of the nutritive value of nucleoproteins in meats;

5. A marked acceleration of structural disintegration in all foods.

CATEGORY III

BIOLOGICAL EFFECTS OF EXPOSURE

Exposure to microwave emissions also had an unpredictably negative effect upon the general biological welfare of humans. This was not

discovered until the Russians experimented with highly sophisticated equipment and discovered that a human did not even need to ingest the material substance of the microwaved food substances: that even exposure to the energy-field itself was sufficient to cause such adverse side effects that the use of any such microwave apparatus was forbidden in 1976 by Soviet state law.

The following are the enumerated effects:

1. A breakdown of the human "life-energy field" in those who were exposed to microwave ovens while in operation, with side-effects to the human energy field of increasingly longer duration;

2. A degeneration of the cellular voltage parallels during the process of using the apparatus, especially in the blood and lymphatic areas;

3. A degeneration and destabilization of the external energy activated potentials of food utilization within the processes of human metabolism;

4. A degeneration and destabilization of internal cellular membrane potentials while transferring catabolic [metabolic breakdown] processes into the blood serum from the digestive process;

5. Degeneration and circuit breakdowns of electrical nerve impulses within the junction potentials of the cerebrum [the front portion of the brain where thought and higher functions reside];

6. A degeneration and breakdown of nerve electrical circuits and loss of energy field symmetry in the neuroplexuses [nerve centers] both in the front and the rear of the central and autonomic nervous systems;

7. Loss of balance and circuiting of the bioelectric strengths within the ascending reticular activating system [the system which controls the function of consciousness];

8. A long term cumulative loss of vital energies within humans, animals and plants that were located within a 500-meter radius of the operational equipment;

9. Long lasting residual effects of magnetic "deposits" were located throughout the nervous system and lymphatic system;

10. A destabilization and interruption in the production of hormones and maintenance of hormonal balance in males and females;

11. Markedly higher levels of brainwave disturbance in the alpha, theta, and delta wave signal patterns of persons exposed to microwave emission fields, and;

12. Because of this brainwave disturbance, negative psychological effects were noted, including loss of memory, loss of ability to concentrate, suppressed emotional threshold, deceleration of intellective processes, and interruptive sleep episodes in a statistically higher percentage of individuals subjected to continual range emissive field effects of microwave apparatus, either in cooking apparatus or in transmission stations.

Forensic Research Conclusions

From the twenty-eight above enumerated indications, the use of microwave apparatus is definitely not advisable; and, with the decision of the Soviet government in 1976, present scientific opinion in many countries concerning the use of such apparatus is clearly in evidence.

Due to the problem of random magnetic residulation and binding within the biological systems of the body (Category III:9), which can ultimately effect the neurological systems, primarily the brain and neuroplexuses (nerve centers), long term depolarization of tissue neuroelectric circuits can result. Because these effects can cause virtually irreversible damage to the neuroelectrical integrity of the various components of the nervous system (I. R. Luria, Novosibirsk 1975a), ingestion of microwaved foods is clearly contraindicated in all respects. Their magnetic residual effect can render the pyschoneural receptor components of the brain more subject to influence psychologically by artificially induced microwave radio frequency fields from transmission stations and TV relay-networks.

The theoretical possibility of psycho telemetric influence (the capability of affecting human behavior by transmitted radio signals at controlled frequencies) has been suggested by Soviet neuropsychological investigations at Uralyera and Novosibirsk (Luria and Perov, 1974a, 1975c, 1976a), which can cause involuntary subliminal psychological energy field compliance to operative microwave apparatus.

FORENSIC RESEARCH DOCUMENT
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Ten Reasons to Throw out your Microwave Oven

From the conclusions of the Swiss, Russian and German scientific clinical studies, we can no longer ignore the microwave oven sitting in our kitchens. Based on this research, we will conclude this article with the following:

1). Continually eating food processed from a microwave oven causes long term - permanent - brain damage by "shorting out" electrical impulses in the brain [de-polarizing or de-magnetizing the brain tissue].

2). The human body cannot metabolize [break down] the unknown by-products created in microwaved food.

3). Male and female hormone production is shut down and/or altered by continually eating microwaved foods.

4). The effects of microwaved food by-products are residual [long term, permanent] within the human body.

5). Minerals, vitamins, and nutrients of all microwaved food is reduced or altered so that the human body gets little or no benefit, or the human body absorbs altered compounds that cannot be broken down.

6). The minerals in vegetables are altered into cancerous free radicals when cooked in microwave ovens.

7). Microwaved foods cause stomach and intestinal cancerous growths [tumors]. This may explain the rapidly increased rate of colon cancer in America.

8). The prolonged eating of microwaved foods causes cancerous cells to increase in human blood.

9). Continual ingestion of microwaved food causes immune system deficiencies through lymph gland and blood serum alterations.

10). Eating microwaved food causes loss of memory, concentration, emotional instability, and a decrease of intelligence.

Have you tossed out your microwave oven yet?

The use of artificial microwave transmissions for subliminal psychological control, a.k.a. "brainwashing", has also been proven. We're attempting to obtain copies of the 1970's Russian research documents and results written by Drs. Luria and Perov specifying their clinical experiments in this area.

Click here for another article on Microwave Cooking

BROCCOLI SPROUTS

Small quantities of fresh broccoli sprouts contain as much cancer protection as larger amounts of the mature vegetable sold in food markets, according to researchers at Johns Hopkins University. Just 5 grams (0.17 ounces) of sprouts contain concentrations of the compound glucoraphanin equal to that found in 150 grams (5.2 ounces) of mature broccoli. The compound is a precursor to sulforaphane, proven in animal studies to boost cell enzymes that protect against molecular damage from cancer-causing chemicals.

Sulforaphane has been shown to mobilize, or induce, the body's natural cancer protection resources and help reduce the risk of malignancy. Broccoli is the best source of the chemical precursor to sulforaphane -- glucoraphanin. Now, broccoli sprouts are an "exceptionally rich source" of inducers of cellular enzymes for "detoxifying" chemical carcinogens -- cancer causing compounds. Some of these compounds are potent enhancers of phase 2 enzymes, which speed the detoxication of electrophiles and reactive oxygen metabolites. Therefore, they say, induction of phase 2 enzymes by these compounds can "...protect cells against mutagenesis and neoplasia."

The researchers attempted to calculate how much broccoli one would have to eat in order to produce a significant degree of protection against cancer, based on epidemiologic evidence. They found that one would have to eat about two pounds of an average broccoli a week in order to reduce, say, one's risk of colon cancer by about 50%.

An additional complication is that it is impossible to determine by the looks of this cruciferous vegetable alone or even with knowledge of how and where it was grown whether you are buying the 'high-inducer' or 'low-inducer' broccoli. The enzyme-inducing abilities of samples taken from 22 varieties of fresh and 7 brands of frozen mature broccoli vary greatly. Only ~~sophisticated scientific measurements can determine the~~ concentrations.

But fresh broccoli sprouts offer an alternative. One can get away with eating 10 to 100 times lower quantities. Three day old sprouts have the additional advantage that they're far more uniform in their potency. Broccoli sprouts look and taste something like alfalfa sprouts, according to the researchers. The report also notes that small quantities of broccoli sprout extracts markedly reduced the size of rat mammary tumors that were induced by chemical carcinogens.

The researchers refer to the concept of "chemoprotection" -- deliberate efforts to increase the body's own defense mechanisms to reduce susceptibility to carcinogens by administration of substances that can be precisely identified, and ideally, delivered in the diet. The interesting aspect of chemoprotection strategies is that they're almost never organ-specific. Chemoprotection produces a general cancer

protective effect which blocks multiple steps -- a cascade of steps -- that are common to cancer formation.

INSIGHT: At the present time, broccoli sprouts are not being grown commercially. However you can grow them yourself quite easily. You can purchase organic broccoli seeds from Johnny's at 207-437-4301. Item number 148, four ounces are $9.00 or a pound for $12.00. Non-organic seeds can probably be purchased through farm supply stores or other seed catalogs. You can also call Jaffe Brothers at 619-749-1133 for instructions on how to sprout. They also sell sprouting lids to apply to Ball jars which make the entire process quite convenient. The library or health food store may also have some instructions on sprouting seeds.

A small amount of spouts go a long way. A pound of sprouts will probably make over ten pounds of sprouts which from the researchers calculations translates up to as much cancer protecting phytochemicals as 1000 pounds (half a ton) of broccoli! The other major benefit is that the sprouts don't smell as you don't have to cook them. They are eaten raw, usually as an addition to salad. I have already ordered my sprouts. I suspect that there are similar benefits for many of the other vegetables when eaten as sprouts. From the Proceedings of the National Academy of Sciences (1997;94:10367-10372)

Sprouting Instructions:

Sprouting Time 5-7 days Temperatures 60-80
Yield 1 Tbs seed=1-1 1/2 cups sprouts

Place seeds in the jar and cover with cheesecloth or other porous material. Secure the cloth with a rubber band. Fill the jar full with filtered water and let soak overnight. In the morning drain off the water. The cloth will keep the seeds in the jar. Rinse seeds with water again and let drain. You can also use reusable plastic sprouting lids (available from Jaffe Brothers at 619-749-1133) which attach to Ball jars. This avoids having to use new cheesecloth each time. Put the container on its side in a dark location that stays about 70 degrees. Rinse twice each day with water and drain. Continue the rinse cycle until harvested. When sprouts are 1-2 inches long you can put them in the light to green them up. You can rinse out the hulls or skim them of the top of the water with a spoon. They are not harmful to eat but removing them minimizes spoilage when sprouts are stored. Sprouts are best stored in an airtight container in the refrigerator and keep for about one week.

DIS - 1A-27

Free Newsletter

Read this first

Carbohydrates

Lower Your Grains and lower your insulin levels.
Over the past fifteen years, our dietary establishment has made a virtual industry of extolling the virtues of carbohydrates. We're constantly told that carbohydrates are the good guys of nutrition, and that, if we eat large amounts of them, the world should be a better place. In such a world, the experts tell us, there will be no heart disease and no obesity. Under such guidance, Americans are gobbling breads, cereals, and pastas as if there were no tomorrow, trying desperately to reach that 80 to 85 percent of total calories advocated by the high-carb extremists.

Scientific Evidence of the value of eating few grains
Unfortunately, the debate over the validity of this concept has primarily been waged in the media and lay publications and not in the scientific journals. Many of the popular books which support this position are gimmicky, and often, lack adequate scientific referencing. Yet, at their core is very important concept -- limiting the intake of carbohydrates, (especially as cereal grains and starches), will improve human health.

CONSTITUTIONAL SKIN DISEASES

CONSTITUTIONAL SKIN DISEASES

Constitutional skin diseases is a common problem. Left untreated these idseases have a tendency to be chronic. Often infection enters due tc scaratching or due to medications producing pus discharge and swelling of the affected part.

Usually the patient goes from doctor to doctor and switch from one system of medicine to another. Often patient gets relief for varying periods of time. The chief reason for their persistence is because the affected person doesn't understand why these skin diseases occur.

Basically these types of skin diseases are constitutional and the person is born with a tendency to develop it. But only a small percentage of the people who has this tendency actually develop the skin disease and only in a minority it persist and becomes troublesome.

Some of the factors which aggravate or precipitate these conditions are dry skin, climate, working enviornment and psychological stress. Psychological factors usually mean person's inability to adjust well with his enviornment which includes family, friends, work, finances etc.

Very often persons who suffer from chronic skin diseases become suspicious that they have certain diseases like cancer, V.D., leprosy, leucoderma etc. Though a competent doctor can easily recognise these conditions, he may ask for some investigationsto confirm, but more often to reassure the patient.

One of the major symptom associated with constitutional skin disease is itching. Scratching is socially embarrasing and the patient feels very conscious and guilty about it. Itching is an intolerblesymptom and the patient scratches so that the sensation gets converted to tolerable pain or burning. Repeated scratching produces skin damage and allow the skin to become thick and dark. The nerves underneath the skin will become irritable and produces itching on slight provocation. The best stimulus for itching is scratching itself and the skin will be irritable for quite sometime after a bout of scratching. So unless the scratching stops the skin disease will always be present.

Often the person touches the affected part without his knowledge. Once an irritable area is touched the area bcomes very itchy and the person will have to scratch. Most of the time the affected area is touched as a matter of habit, usually when the person is immersed in some mental activity like reading or thinking. (Usually long spells of regretting the past or worrying about the future).

If the person can ask someone to remind him frequently he will be aware of the habit. Then he could prevent the itching by applying the prescribed medications and doing something more active like going for a walk etc. Sometimes the person realises this too late and will be forced to scratch. But such episodes will become rarer and stop if he concentrates on his problem.

The medications are to reduce the itching. But medications cannot help the patient unless the patient understands the effect of scratching and makes an effort to avoid it. Applying the prescribed ointment 10-15 minutes before the usual times of itching, eg: after undressing, bed time, etc will help to reduce the itching. The person must make very effort to achieve a cure at the shortest possible time.

As mentioned earlier the person with constitutional skin diseases has the tendency to get them. So even after a cure it is important to avoid the provocating factors which produces the disease. If relapse actually takes place it is better to take treatment again as early as possible.

LEUCODERMA (VITILIGO)

Leucoderma is a common skin disease. It produces white patches due to loss of colour producing substance known as melanin. The exact nature of the disease is still not known.

It is not contagious and usually produces no health problems except for the appearance. It has nothing to do with leprosy. In India it is definitely a social disease since many believe that leucorderma is contagious and is a form of leprosy.

The effect of treatment vary. In majority very satisfactory results can be obtained if treatment is taken regularly.

Leucoderma is unpredictable in its course. It can appear at almost any age. Only a small percentage of the patients get widespread patches. In the majority, the patches are confined to the covered parts of the body and in such cases doctors rarely recommend treatment unless the patient is very anxious.

It is important to remember that even after a complete clearance the white patches can come back any time, and the patient may have to take the treatment again.

The usual treatment recommended involves exposing the patches to sunlight after giving local or internal medication. The ideal time for the treatment is when the sunlight is brightest ie. between 10 AM and 4 PM. The following precautions are necessary while taking the treatment.

1. STRICTLY adhere to the recommended duration of sun exposure. Over exposure can produce severe reactions. Under exposure will delay the response.

2. Not to exceed the time limit even if the sun is not very bright for example on a cloudy day.

3. The patches must be covered to protect them from sunlight if patient is going out in the sun on the same day.

4. Protect the eyes either by covering or with dark glasses during sun exposure.

5. If any reaction like itching, burning pain or blister formation occurs, STOP the treatment and contact the doctor as early as possible.

COMMON BALDNESS

Common Baldness or loosing hair during adulthood is a universal problem. This tendency is purely hereditry and produced by the influence of hormones by certain glands in the body which becomes active after the perosn becomes matrue.(Puberty). So common baldness starts only after puberty. Though the glands and the hormones are normal the effect on the hair roots is different in different individuals and is purely hereditary. But hereditary factors does not manifest like a mirror image of the parents. Often a wide variations are observed and sometimes the genes carrying the trait can even skip generations.

In common baldness which affects every one, some of the hairs which enter the resting phase are not replaced. The percentage of the hair could be so low that some will never have noticable hair loss. But on the other hand there are people with strong hereditary tendency to lose hair rapidly and become almost totally bald in their twenties.

The common baldness follows certain well defined patterns in men. But in women it is more diffuse, and obvious thinning confined to certian areas of the scalp is rare but seen occassionally.

Since dandruff occurs only on hairy areas and spares bald areas many will think dandruff is the cause of baldness which is not true.

TREATMENT

Since common baldness is a physiological response to the sex hormones, at present there is no modality which will arrest this process. Removal of the glands (Castration) will certainly arrest the further hair loss but obviously cannot be recommended.

Hair transplantation gives satisfactory results if done by experts. This is very expensive and time consuming. Well fitting wigs are good remedy for many. A new drug - MINOXIDIL is under investigation and also being marketted in United States, we will have to wait and watch about its safety and effectiveness in common baldness.

cont..3..

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NORMAL HAIR GROWTH AND COMMON BALDNESS

The hair goes through a cycle known as hair cycle. These are:

1. Growing Phase (ANAGEN):

During this phase the hair grows continously. The growth rate for scalp hair is about 1 cm/month. The length of the hair depends on the length of the growing phase which is largely determined by heredity. This varies from few months to sometimes 20 to 30 years, which explains why some have unusually long hair. During the growing phase if the hair is pulled out, hair root will be balck in colour.

2. Resting Phase (TELOGEN):

During this phase the root degenerates slowly and become loose at the attachment. This last for 1-2 months. Towards the end of this phase the hair can be pulled out with gentle pressure. The hair root will be white in colour. About 20% of the scalp hair will be in resting phase at any given time.

3. Falling Phase (CATAGEN)

During this phase a new hair start growing from the bottom of the hair root to replace the old hair. Old hair falls off. The hair is pushed out by the new hair which takes its place.

This is the normal hair cycle. The following points are are note worthy.

1. During falling phase hair will come out naturally without using any force - This should not be taken as loosing hair.

2. With moderate force the hair in the resting phase will come out easily. That is why during combing, lot of hair is seem to fall.

3. Usually most of the scalp hair comes out during bath. That is why people who take bath less often complain that they loose more hair while taking bath, since the accumulated number of hair is in the resting phase will come out while putting pressure on the scalp hair.

4. People who have thick hair notice more hair coming out since proportionately more hair enter the resting phase for them.

cont..2..

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PSORIASIS

Psoriasis is a very common skin disorder. About 1-3% of the people all over the world irrespective of country and race suffer from Psoriasis.

Posriasis is NOT CONTAGIOUS to others and usually does not affect the perosn's health.

Psoriasis usually affects the skin and occassionally the nails and very rarely the joints. On skin it manifests as scaly patches. Sometimes only the soles or palms are affetcted where it is seen as scaly patches and cracks which bleeds often. Itching is usually mild in Psoriasis. But in some it can be severe and disturbing and aggravated by physical and emotional stress.

Psoriasis has no relationship with food and the effect of certian food on Psoriasis is only coincidental.

Psoriasis is due to the production of excessive skin which is shed as scales. The basic cause of this excessive skin production, occuring only in certain individuals is not known yet. Extensive reasearch is going on all over the world, about Psoriasis.

Psoriasis is unpredictable in its course. Natural appearance and disappearance are observed, sometimes even without any medications. Sometimes the disease clear for many years. Some patients observe remarkable improvement and sometimes total clearance of Psoriasis during certian seasons particularly either in summer ar in winter.

Appearance of Psoriatic patients on the sites of injury is a common occurance. This also happen whereever patient scraches. So it is important to avoid injuries and avoid scratching.

Various medications are available for the treatment of Psoriasis. Though a guaranteed cure even after the stoppage of treatment is not available at present, in most patients satisfactory clearence of Psoriasis can be achieved with simple treatment.

The type of treatment recomended varies according to the severity of the disease, associated symptoms like itching, the location of the patches, patient's occupation, social needs etc.

Often various modalities of medicines have to be tried before coming to the conclusion of what suits one individual patient.

Some of the very effective medications are also sometimes inconvenient, expensive or toxic, so is usually reserved for people with severe disease (eg. PUVA, Methotrexate, Oral Retinoids etc). Such treatment should be taken only under strict medical supervision.

cont..2..

Psoriasis being a chronic skin disease produces much social and psychological problems. It is observed that those who have well adjusted personal and professional lives accept the disease and the treatment better.

The following points should be noted in regard to the treatment

(a) For best results apply the ointment immediately after bath.

(b) The ointment should be gently rubbed into the skin. After application ointment should not be present on the skin.

(c) All ointment should be applied to the minimum to sensitive areas like face, armpits, joints, between fingers etc.

(d) Areas like palms and soles where thick patches produces cracks, soaking the parts in warm water for 10-30 minutes before applying the ointment will be useful for faster relief.

(e) The ointment should be applied only to all scaly patches old and new and not to the healed patches with only colour change.

ATOPIC DERMATITIS

Atopic dermatitis (infantile Eczema) is a constitutional type of skin disease. It is not contagious to others. The skin is very itchy and the child scratches and the disease is the result of the scratching. Sometimes large areas of the body can be affected.

The disease is unpredictable in its behaviour. Though most children are free of the disease by the age of 1 year, many many continue to have the disease for a long period. Till the disease disappear on its own it has to be kept at under control.

The following measures are very useful.

1. Never tie the childs hands to prevent him/her from scratching. When the child scratches a lot take him and hold close to your body and talk to him. Divert his attention. Gently rub the area where he was scratching.

2. Many children have secere bouts of scratching when they feel insecure. For eg. when parents have a fight. Try to avoid such situations.

3. Breast feeding the child is a good practice. It gives the child a sense of security in addition to all the advantages of breast feeding.

4. Your child's skin is usually dry because of this disease, It is likely to become worse in a place like Bangalore where the climate is very dry. The following measures will help to reduce the dryness.

a. use minimum amount of soaps and detergents for bathing. Even better, avoid soap completely. Gram flour is better. (Bengal gram or green gram)

b. Immediately after bath when the skin is wet smear a vegetable oil (coconut oil, olive oil etc) or cold cream generously all over the body. The effect will be much less if applied when the skin is dry.

5. To cut the nails once in 3 days. It will be easier if it is done after bath.

6. Avoid putting woollen and other rough clothes directly over the skin. It will irritate the skin.

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7. Accept the condition and avoid indulging in long discussion with friends and relatives about the disease in the presence of the child.

8. The child should never be allowed to feel less acceptable or lovable because of the disease. It is quite possible that child will have problems of being accepted by others outside the house. But non acceptance in the home will have much worse effect.

9. Managing a child with atopic dermatitis is not easy. Good understanding between the parents is essential. The temptation of blaming each other for the childs plight should be avoided.

10. The medication should be correctly used. Often more than what is required is used to produce severe side effects and toxic effects. The medication shouls be used just enought for the satisfactory control of the disease.

11. Stop using the ointment once the child develop painful boils or pus filled boils. Ointment will make the infection worse.

12. Well managed children will only require medical attention rarely. But never hesitate to consult the doctor. Doctor is primarily meant to reassure you and help you to manage better.

DRY SKIN

The skin problems directly or indirectly due to dry skin are very common.

The following factors contribute to this condition:

1. Climate: Dry skin is common in a place like Bangalore where the atmospheric humidity is low throughout the year. In addition the dryness of the air becomes more pronounced during the winter months (November to March).

2. Heridity:

Dry skin runs in families and heriditary influence is pronounced.

3. Age:

Dry skin becomes worse with increasing age. Once a person gets symptoms due to dry skin it can only becomes worse without treatment year after year.

The dry skin can produce symptoms by itself or it makes many skin diseases worse.

Treatment:

The skin is dry because of lack of moisture. So unless the skin is made to retain more water dry skin will remain.

The most important step is to hydrate the skin. This can be done by soaking the body in water for a long time (water tub or long bath). Before the water is allowed to dry apply a vegetable oil or vaseline to all dry areas of the body. The oil has to be rubbed into the skin, preferably after mixing with a small amount of water. If done correctly the oil will enter the skin rapidly. (if applied on a dry skin the oil will not enter the skin and will dirty the clothes).

A little amount of Talcum powder can be used to remove the oily appearance on areas like face and neck.

If any ointment is prescribed for the associated skin disease, this can be applied over the skin, after applying the oil or vaseline.

Soap:

Soap and detergents should be used to the minimum by people who suffer from dry skin. Soaps with high alkali content and medicated soaps should be avoided. Gram flour (eg green gram or bengal gram) is a good substitute for soaps.

The problems associated with dry skin are part of the person's skin and so cannot be cured, but only be controlled. After few visits to the doctor for guidance the patient should know how to manage the skin throughout the year.

High mortality despite good care-seeking behaviour: a community study of childhood deaths in Guinea-Bissau

M. Sodemann,[1] M.S. Jakobsen,[1] K. Mølbak,[2] I.C. Alvarenga Jr.,[3] & P. Aaby[4]

The care-seeking behaviour of mothers of 125 children deceased aged 1–30 months was investigated by verbal autopsy in an urban area of Guinea-Bissau. A total of 93% of the children were seen at a health centre or hospital during the 2 weeks before death. In a previous survey covering the period 1987–90 we found that 78% of the children who died had presented for consultation (8); despite this increase in care seeking, infant mortality had not decreased. Comparison of elapsed time from disease onset to first consultation between children who died and matched surviving controls indicated that the interval was shorter for children who died than for those who survived (odds ratio (OR) = 0.7; 95% confidence interval (CI): 0.5–0.99). Of the 125 terminally ill children, 56 were hospitalized. A total of 20 children died on the way to hospital or while waiting in the outpatient clinic. Lack of hospital beds resulted in 15 mothers being refused hospitalization for their child. Of hospitalized children, 42% were discharged as improved or recovered during the 30 days preceding death. These results reveal a need for improved hospital admission criteria, improved recognition of the symptoms of serious illness, better discharge criteria, and the implementation of quality assurance systems for health services.

Introduction

Many studies have reported a significant decrease in child mortality following general improvements in primary health care (PHC) (*1, 2*). Such decreases are mainly the result of improvements in antenatal care and vaccination coverage: the effect of diarrhoeal disease programmes is less unequivocal. However, as vaccines and antenatal care cannot entirely eradicate the problem of excess childhood mortality in developing countries, better case management of severely ill children is clearly needed.

Improvements in the management of severely ill children are often based on audits of case histories with fatal outcomes (*3*). However, few studies in developing countries have investigated care-seeking behaviour prior to death in serious childhood illness in order to improve case management (*4–6*). Mortality surveys have found large variations in the proportion of children seen at a health facility before dying (*7–10*). It is important to explain why mortality remains high among under-5-year-olds in settings with easy access to health care facilities. For example, in a rural area of the Gambia, where 80% of children were fully immunized and PHC programmes had been active for 10 years, infant mortality was still 120 per 1000 live births in 1990 (*11*). In the Bandim suburb of Bissau, Guinea-Bissau, we previously reported that the infant mortality was 94 per 1000 and under-5-year-old mortality 215 per 1000, despite the presence of two health centres, a mother-and-child health clinic and an outpatient clinic (*8*).

We conducted the present study on patterns of care-seeking behaviour prior to a child's death to investigate child mortality on the basis of the mother's experience. The aim was to obtain individual case histories that could be used to improve patient management in primary health care programmes and thereby lower childhood mortality in developing countries.

Subjects and methods

Study area

The study was carried out in the suburbs Bandim 1 and Bandim 2 of the capital Bissau, Guinea-Bissau. The population of approximately 25000 persons

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is served by two local health centres (with senior nurses consulting), one mother-and-child health clinic (with physicians consulting) as well as an outpatient clinic at the paediatric ward of the national hospital (with hospital paediatricians consulting). All inhabitants live within 1 km of a health centre and within 3 km of the mother-and-child health clinic and outpatient clinic. Apart from a small one-time charge levied for a child's vaccination chart, no fees were charged at health facilities in Bissau during the study period. Since 1979, the area has had a demographic and health surveillance system that covers the following: registration of all pregnancies and births; and for children less than 5 years of age, routine collection, by means of 3-monthly visits to all houses, of information on vaccinations, infections, nutritional status, migrations, and deaths. Morbidity and care seeking are monitored by weekly household interviews. Traditional remedies for severe diseases are not generally the first treatment choice.

Study population

The cohort followed in the present study consisted of all 1347 children born in Bandim 1 or 2 between 1 May 1992, and 30 April 1993. Deaths were ascertained by means of the routine surveillance system. Two additional rounds of data collection were carried out in 1993 and 1994; furthermore, a census of the entire population was performed in 1994. Verbal autopsies were conducted by two of the authors (MS & ICA) and a specially trained Guinean midwife. Interviews were carried out from July 1992 to November 1994, by which time the youngest children in the study cohort were 18 months of age and the oldest children 30 months of age. Median time from death to interview was 7 months (25–75th percentile: 6–9 months). The immediate cause of death was determined by combining information from the verbal autopsy, the morbidity survey, and a register of hospital diagnoses. Morbidity information was considered valid if the child had been followed up until death, and hospital diagnoses were considered valid when the child had been hospitalized for more than 24 hours. Hospital records were available for 43 of 56 hospitalized children (76.8%). A probable cause was determined for 93% of all deaths. In this study, "hospitalization" was defined as hospitalization at any time during the 30 days preceding death, regardless of subsequent discharge from hospital. Households with deaths were divided into two socioeconomic status groups: group 1 (50 mothers) consisted of households with two or more of the following: corrugated iron roof, television, inside toilet, and electricity; group 2 (75 mothers) consisted of households with less than two of these characteristics. Socioeconomic information was obtained from the health surveillance system.

Statistical methods

Sample means were compared with the Student's *t*-test for normally distributed data, but the Kruskall–Wallis test was used when sample variances were significantly different. In bivariate analyses, background factors were controlled for by means of a Mantel–Haentzel stratified analysis of two-by-two tables. A nested case–control study was carried out by matching a control to each fatal case. The control was chosen from among children in the study population participating in a weekly morbidity survey, age-matched (±1 month), had experienced an episode of disease within the same month as the fatal case, was seen at a health centre or a hospital, but survived at least 3 months following the episode. Time to consultation was measured as the number of days between the onset of illness and the first consultation. The odds ratio was calculated as the ratio of discordant pairs, and 95% confidence intervals (CI) were calculated with Miettinen's test-based approach (*12*).

Results

Childhood mortality in the study group

All deaths of live-born children (248/1347) were investigated by verbal autopsies with the mother, or the nearest relative if the mother was absent. The circumstances, timing, and location of each contact with health care personnel during the fatal illness were recorded during the interviews. The proportion of stillbirths was 55 per 1000 births, perinatal mortality was 81 per 1000 births, and infant mortality 91 per 1000 live births. However, only post-neonatal deaths (125/1347) were included in this analysis.

Of the 125 such cases, 114 had verifiable information on care seeking. Of these, 106 children (93%) were seen by a health professional during the 14 days preceding death. Of the eight others, two died on the way to consultation, three died suddenly and unexpectedly, and three from diseases ascribed to traditional ceremonial causes. Compared with our previous mortality survey in the same city, the behaviour reported here represents a significant reduction in the risk of not being brought to a health facility (risk ratio = 0.3; 95% CI: 0.2–0.7) (*8*). Moreover, 33 (26.4%) of the deaths occurred in hospital, whereas in the previous survey 45% of deaths occurred in hospital. A total of 23 children (18.4% of deaths) died

Table 1: Cause of death and pattern of care seeking

	Median duration of illness (days)[a]	Median days to first consultation[a]	No. of children, by place of first consultation[b]						
			Health centre	Outpatient clinic	Other[c]	No information	No consultation	No information on care seeking[d]	Total
Acute diarrhoea	7 (3–22)	2 (2–4.5)	13 (56.5)	4 (17.4)	4 (17.4)	2 (8.6)	0 (0.0)	0 (0.0)	23
Fever	4 (3–25)	2 (1–5)	10 (31.3)	10 (30.3)	7 (21.1)	1 (3.0)	4 (12.1)	1 (3.1)	33
Pneumonia	10 (5–30)	3 (1–5)	9 (56.3)	7 (43.8)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	16
Malaria	8 (2–20)	3 (2–5)	3 (50.0)	3 (50)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	6
Other[d]	7 (2–30)	1.5 (0–15)	4 (11.4)	11 (34.3)	4 (11.4)	2 (5.7)	4 (11.4)	9 (25.7)	35
Chronic diarrhoea	30 (25–60)	14.5 (1–46)	8 (66.7)	2 (16.7)	1 (8.3)	0 (0.0)	0 (0.0)	1 (8.3)	12
Total			47	38	16	5	8	11	125

[a] Figures in parentheses are the 25th–75th percentiles.
[b] Figures in parentheses are percentages.
[c] Mother and child health clinic or regional hospitals in the interior.
[d] Malnutrition (n = 5), measles (n = 8), congenital diseases (n = 8), sudden death (n = 3), unknown (n = 11).

at home after being discharged from hospital. No information on the location of death was available for seven children.

Time to consultation

To determine whether mothers delayed consultation in fatal cases, we performed a nested case–control study. It was possible to match a surviving control for 93 cases. Equal delay occurred in 9 of the matched pairs. In 35 matched pairs, the case exhibited the longest delay and in 49, the control. Cases were more likely to have a shorter delay than controls, with an odds ratio of 0.70 (95% CI: 0.50–0.99).

First consultation and hospitalization

The place of first consultation according to cause of death is shown in Table 1. The median time from onset of symptoms to first consultation was independent of child's age, ethnic group, mother's education, socioeconomic status, and cause of death.

Of the 47 children first seeking care at a health centre, only 23 (49%) were admitted to hospital and, of these, 20 only after the mother sought consultation more than once. Of the 38 children first seeking care at the outpatient clinic at the paediatric ward, 22 (58%) were admitted to hospital. Immediate hospitalization was more likely among children presenting at the outpatient clinic first than for children presenting at the health centre (risk ratio = 5.0, 95% CI: 2.0–12.5). Children taken to a health centre survived longer (median, 8 days; 25–75th percentile: 2–24 days) following first consultation than children initially taken to the paediatric ward (median, 2 days; 25–75th percentile: 0–8 days; $P = 0.01$). No difference in survival time after first consultation was found between children hospitalized immediately ($n = 18$) and others ($n = 20$; $P = 0.8$). After controlling for mother's education, socioeconomic status, and child's age, there were no differences in place of first contact with the health system. However, children with diarrhoea were more likely to contact a health centre first. Of those children surviving at least 2 days from first consultation without hospitalization, 39 (84.8%) later reattended a health facility.

Hospitalization: reasons for refusal or discharge

Of the 114 deaths for which we have information, 103 children (90.3%) presented at hospital one or more times during their terminal illness. Of these, 10 children died on the way to hospital and 10 while waiting for treatment in the outpatient clinic (Table 2). Of the remaining 83 children, 56 (67.4%) were admitted either at the first visit ($n = 22$) or at a subsequent visit; of those admitted, 18 children died the day of admission. For the 61 children not admitted at first consultation, 15 of the mothers stated they were turned away from the outpatient clinic having been informed that their child should be hospitalized, but that there were insufficient beds. Refusal of admission did not depend on mother's education, socioeconomic status, or child's age ($P = 0.9$). Elapsed time since disease onset (>14 days) increased the risk of refusal (risk ratio, 2.4; 95% CI: 0.9–6.6).

Of the 56 children admitted to hospital, 23 (41.7%) were discharged before death, with the risk of discharge being independent of socioeconomic status ($P = 0.44$) and child's age ($P = 0.35$). Of these, 9 children were discharged as "cured", 10 as "improved" or "recuperating", 1 was discharged by the mother, and 2 had no status information at discharge.

Case histories

Six representative case histories depicting the management problems of severe childhood illness in the study area are shown in Table 3.

Discussion

Despite a high percentage of children with fatal illness obtaining treatment from a health professional, infant mortality in the study suburban area of Guinea-Bissau has continued to be high: (1987–90, 94 per 1000; 1992–93, 91 per 1000). The proportion of children who later die after presenting for consultation both at health centres and hospitals has increased significantly since our previous mortality survey (*8*).

Among physicians and other health care workers in developing countries, a common explanation for high childhood mortality is that, as mothers are believed to be incapable of caring for a severely ill child, children are brought to care too late. Moreover, they do not recognize severe symptoms and may seek traditional care first. However, the present study indicates that mothers sought care sooner in cases of fatal illness than in other cases.

Conceivably, seriously ill children would have a better chance of survival if they were treated at the outpatient paediatric ward than at a health centre. The ratio of mothers choosing a health centre as site of first consultation to mothers choosing the paediatric ward was the same, regardless of mother's education, socioeconomic status, child's age, and ethnic group. Moreover, the chance of being admitted im-

Table 2: Place of first consultation and subsequent care seeking

Place	Hospitalized: Directly	Hospitalized: After one or more reconsultations	Hospitalized: Total hospitalized[a]	Not hospitalized: Died at home[b]	Not hospitalized: Died on the way to outpatient clinic	Not hospitalized: Died while waiting for consultation at outpatient clinic	Not hospitalized: Total not hospitalized	No information on place of death	Total
Health centre	3	20	23 (11)	19 (11)	2	3	24	—	47
Outpatient clinic	18	4	22 (6)	7 (6)	3	6	16	—	38
Mother-and-child health clinic	0	2	2 (1)	2 (1)	0	1	3	—	5
Hospital or health clinic in the interior	1	4	5 (2)	3 (2)	3	0	6	—	11
No information on place	—	—	4 (3)	—	—	—	1	—	5
No consultation	—	—	—	6	2	—	8	—	8
No information on consultations	—	—	—	4	—	—	4	7	11
Total	22	30	56 (23)	41 (20)	10	10	62	7	125

[a] Figures in parentheses are number of children subsequently discharged who died outside hospital.
[b] Figures in parentheses are number of children dying at home after one or more reconsultations.

Table 3. Typical case histories

Girl, 9 months of age, presents at health centre with diarrhoea and vomiting after 2 days of illness; given oral rehydration salts and sent home; next day mother seeks care for child at another health centre; after 2 more days, mother presents child at outpatient paediatric clinic where the child is given oral rehydration salts and sent home; 20 days later child dies without receiving additional care.

Boy, 4 months of age, presents at outpatient paediatric clinic with diarrhoea and vomiting after 2 days of illness; given oral rehydration salts and sent home; 3 days later mother seeks care for child at outpatient paediatric clinic; child given oral rehydration salts and sent home; 10 days later, child collapses at home and mother takes child to outpatient paediatric clinic; child is sent home without treatment and dies same day.

Boy, 14 months of age, presents at outpatient paediatric clinic with high fever after 1 day of illness; given medication and sent home (mother told not enough hospital beds); child suffers generalized seizures at home, and next day mother returns to outpatient paediatric clinic with child comatose; after waiting 2 hours, child sent to laboratory for tests and dies without receiving additional care.

Boy, 9 months of age, hospitalized twice within 2 months for high fever and multiple boils; each time discharged as "cured" 2 weeks after last discharge child dies at home with fever and convulsions.

Girl, 7 months of age, presents at mother-and-child health clinic with high fever and chills after 2 days of illness; given chloroquine and paracetamol and sent home; same day child worsens, and mother seeks care at outpatient paediatric clinic; child dies before receiving additional care.

Boy, 12 months of age, presents at health centre with fever, vomiting, and constipation after 1 week of illness; child given lemon juice and sent home; after no improvement, mother seeks care same day at outpatient paediatric clinic; child admitted; after waiting more than 1 hour for a blood sample, and then waiting for the father to buy the necessary drugs (which were not in stock at hospital), child dies without receiving additional care.

mediately to the paediatric ward of the hospital was remarkably lower for children presenting initially at a health centre than for children presenting initially at the outpatient paediatric ward. It is therefore possible that health centres retard needed hospitalization. As health centers were visited more frequently than in our previous mortality survey, this could have serious implications.

Apart from five children dying suddenly at home or on the way to their first consultation, only five children were not presented for consultation at hospital, which is a marked decrease from our previous survey. However, it is significant that so few fatally ill children were admitted to hospital, even from among those who first presented at the outpatient paediatric ward. In many cases, the reason was a shortage of beds. There were nine terminally ill children attended by a paediatrician at the outpatient clinic 48 hours prior to death without being admitted. The risk of refusal of admission after consultation at the outpatient paediatric ward was higher if symptoms had a duration of more than 14 days, indicating that chronic illness was less likely to be seen as requiring hospitalization. This is significant, as the two most common chronic illnesses, persistent diarrhoea and malnutrition, both have a very high mortality (*8*). Since children not returning for a second consultation died rapidly (median survival, 1 day) mothers' lack of knowledge does not explain why children were not hospitalized. Children not hospitalized after a first visit to the outpatient paediatric clinic died as rapidly as those who were; it is therefore unlikely that refusal of admission was based exclusively on clinical criteria. Hospitalization was not influenced by socioeconomic status, mother's education, child's age, or ethnic group. However, verbal autopsies suggested that hospitalization was obtained more easily if a mother knew a staff member in the outpatient clinic.

A considerable proportion of children died waiting for consultation at the outpatient clinic or laboratory. Some of these deaths could probably have been avoided by means of a revision of clinic procedures and by training health personnel to recognize children requiring immediate care.

A major problem is the high proportion of children dying at home after discharge from hospital. In light of discharge status, this could not have been caused by mothers fleeing the hospital with dying children. No sociocultural factors were related to risk of discharge, suggesting that inadequate recognition by medical staff of the potential consequences of illness or nosocomial infections may have been responsible. If so, this problem has been aggravated since our previous mortality survey, since a larger proportion of hospitalized children later die at home. The number of beds in the paediatric ward remained constant between the two surveys. Hence, demand for limited bed space may have contributed to some premature discharges. Hospital beds can be occupied for long periods of time by chronically ill children suffering from malnutrition, persistent diarrhoea, complications resulting from cerebral malaria, or tetanus. A clearer distinction between acute and long-term illness in terms of management and the need for care, as well as a more strict set of rules for admission and discharge could potentially lower demand for bed space.

The present mortality survey consists of case histories with a fatal outcome. Since medical consultations for terminally ill children represent only a small fraction of all consultations for sick children, this survey may be biased as an evaluation of the adequacy of health system procedures. However, the present study does point to a number of specific problems in case management. Previous studies ana-

lysing care-seeking behaviour have focused particularly on traditional beliefs and practices that prevent mothers from seeking proper medical care (*5, 6, 9*). However, our experience suggests that it may be equally important to examine the quality of the medical care provided. This is supported by a Mexican study using verbal autopsies in a similar way. In an area where no household was farther than 30 minutes from a health facility, 60% of the deaths in children occurred at home and 80% of these children had received qualified medical care within 3 days of death (*4*).

For dealing with problems associated with severely ill children WHO/UNICEF recommends "integrated management of the sick child", which combines the principles learnt over the past 15 years in disease-specific health programmes into a unified approach to managing childhood illness (*13*). This initiative focuses on improvements in health-worker performance and changing family behaviour in relation to sick children. Training courses for the inpatient case management of sick children have also been developed.

The present analysis clearly supports the need for such initiatives. Surveys analysing fatal cases can be a valuable tool, and can serve as a cost-effective means for health care workers to identify areas for improvements in the case management of severely ill children. Improving the management of such children may be as important for decreasing childhood mortality as vaccination and antenatal care programmes have been, especially in countries with poorly educated and badly paid health care workers. Inadequate supplies, physical facilities, and equipment may also contribute to the persistence of high childhood mortality in Bissau. Such constraints emphasize improved health system management as a means of better using available resources.

A key step in improving case management should be the establishment of an effective triage system that singles out seriously ill children as soon as they come to a health facility and ensures that appropriate action is taken. More formal criteria for admission, referral, and discharge are also needed, accompanied by clinical and system-management training of staff. Finally, measures should be taken to assure the quality of services provided by health care workers, e.g. by medical audit or the use of epidemiological methods such as those used here. These findings could be extended to the health services of other developing countries; however, important differences may exist depending on available resources, personnel, and payment systems. Hence, additional studies investigating the case management of severe illness at the primary health care and hospital levels in other countries are warranted.

Acknowledgements

We are indebted to Angelina Da Silva and Queba Djana for assistance during interviews and identification of mothers. This study was supported by the Science and Technology for Development Programme of the European Community (contract No. TS3-CT92-0060) and by the Danish Council for Development Research (grant No. 104.DAN8/535).

Résumé

Une mortalité élevée malgré la recherche de soins appropriés: résultats d'une enquête communautaire sur les décès d'enfants en Guinée-Bissau

Bien que de nombreuses études aient fait état d'un déclin significatif de la mortalité infantile à la suite d'améliorations générales des soins de santé primaires, la vaccination et les soins prénatals ne peuvent supprimer à eux seuls la surmortalité infantile dans les pays en développement. Une prise en charge plus efficace des enfants gravement malades est donc nécessaire. Rares sont les études qui ont été faites dans les pays en développement sur la recherche de soins appropriés comme moyen d'améliorer la prise en charge des enfants gravement malades. La présente enquête sur la demande de soins avant le décès a été conduite pour analyser le problème de la mortalité infantile en fonction de l'attitude de la mère. Il s'agissait de recueillir des antécédents médicaux individuels en vue d'améliorer la prise en charge des cas dans le cadre de programmes de soins de santé primaires et ainsi, de réduire la mortalité infantile dans les pays en développement.

La demande de soins par les mères de 125 enfants décédés entre 1 et 30 mois a été étudiée au moyen d'autopsies verbales dans une zone urbaine de Guinée-Bissau. Tous les habitants sont à moins de 3km d'un centre de santé. Au total, 93% des enfants avaient été reçus dans un centre de santé ou un hôpital dans les deux semaines ayant précédé leur décès. Notre précédente enquête, sur la période 1987–1990, avait montré que 78% des mères des enfants décédés les avaient montrés en consultation; toutefois, malgré cette augmentation de la demande de soins, la mortalité infantile n'a pas baissé. Si l'on compare le temps écoulé entre l'apparition de la maladie et la première consultation pour les enfants décédés et des témoins survivants appariés, il apparaît que cet intervalle avait été plus court pour les enfants qui sont décédés que pour ceux qui ont survécu (odds ratio = 0,7; intervalle de

confiance à 95% = 0,5–0,99). Sur les 125 enfants qui étaient en phase terminale, 56 ont été hospitalisés. Vingt sont décédés lors du transport à l'hôpital ou en attendant d'être vus en consultation dans un dispensaire. Faute de lits disponibles, 15 mères se sont vu refuser l'hospitalisation de leur enfant. Sur les enfants hospitalisés, 42% ont été déclarés en meilleure santé ou guéris et renvoyés chez eux dans les 30 jours ayant précédé leur décès. Ces résultats montrent qu'il est nécessaire d'améliorer les critères d'hospitalisation, la reconnaissance des symptômes des maladies graves et les critères de sortie et de mettre en oeuvre des systèmes d'assurance de la qualité des services de santé.

La solution préconisée par l'OMS et l'UNICEF pour le traitement des enfants gravement malades est la "prise en charge intégrée de l'enfant malade" qui associe les principes acquis depuis 15 ans dans le cadre de programmes de lutte contre des maladies déterminées en une approche uniforme et cohérente de la prise en charge des maladies de l'enfance. La présente étude montre très clairement que de telles initiatives sont nécessaires. Les enquêtes sur les cas mortels peuvent être un instrument précieux et fournir aux agents de santé un moyen d'un bon rapport coût/efficacité de déterminer les domaines dans lesquels des améliorations doivent être apportées à la prise en charge des enfants gravement malades.

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MEMORANDUM

TO: **Health Authorities and Community Health Councils**

SUBJECT: **Fluoridation of Water**

FROM: **Professor John Lenihan**

Issued by The Fluoridation Society Ltd
63 Wimpole Street
London W1M 8AL

June 1984

This Memorandum has been prepared by Emeritus Professor John Lenihan, who until recently was the Professor of Clinical Physics at the University of Glasgow and the Director of the Department of Clinical Physics and Bio-Engineering, West of Scotland Health Boards.

He directs this Memorandum to all those who need to make a decision about the fluoridation of water supplies and warns them against accepting the spurious evidence of the anti-fluoridation lobby.

Issued by The Fluoridation Society Ltd
63 Wimpole Street
London W1M 8AL

First, I want to say something about poison – about a material which we are obliged to take into our bodies every day, whether we like it or not. This material, which has not been approved by the Committee on the Safety of Medicines, is made of substances which cause painful illness leading to agonizing death. I am speaking, of course, of sodium chloride – common salt, the salt that we sprinkle in our soup. This substance is added by manufacturers, and without consultation, to a great variety of foods and drinks. It is composed of two most horrible poisons – sodium and chlorine. A tiny quantity of sodium, if taken into the body, produces severe and painful burns. Chlorine is the original poison gas. These two elements are present in almost all of our food. You may think that there is something absurd here – and so there is. Every substance essential for human life can also act as a poison – water, oxygen, calcium, phosphorus, and many more. The idea that some substances are always wholesome and others always poisonous is a favourite war cry of 'pure-water' enthusiasts – but it has no foundation in medicine, science or common sense.

Each generation seems to reproduce a particular mould of people who resist the most obvious advances in public health. In an earlier generation they resisted vaccinations, they resisted the chlorination of water and they resisted the pasteurization of milk. Their modern-day prototypes resist the fluoridation of drinking water with the same venom and enthusiasm. At all times these protestors constitute a small proportion of the population, but they compensate by indulging in hysterical and wild verbosity. The inferiority of their arguments causes them to quote bogus experiments, falsified evidence and palpable untruths. They hope to win their battle by generating fear rather than by reasoned argument. The danger is that by this method they do influence some minds not in possession of the true facts, and in this respect they have achieved some success by delaying the progress of fluoridation in the UK. They have also caused more teeth to decay than need have, thereby increasing suffering and adding to the ever-increasing costs of the NHS.

The anti-fluoridation lobby thought that in their pack of cards they had an unbeatable Ace – this was the fluoridation-cancer scare. In their gambling game they thought that if they played this Ace frequently and with skill they could kill fluoridation once and for all. And this is what they attempted. Those who introduced the rogue Ace were two Americans respectively called Dr Burk and Dr Yiamouyannis but their evidence has not been published in any reputable scientific journal – not surprisingly, because their report is riddled with elementary errors. They compared cancer death rates in a number of American cities, some with fluoridated water and some without. The death rates were higher in the selected cities where fluoride was added to the water. Therefore, they said, fluoride causes cancer. The cancer death rate in Glasgow is higher than in East Kilbride. The consumption of beer per person is higher in Glasgow than in East Kilbride. But if I told you, on this evidence, that beer causes cancer, you would think that I was talking nonsense – and you would be right. The death rate is higher in Glasgow because the average age of the population is

higher. Burk and Yiamouyannis ignored the elementary precaution of correcting their death rates for age, sex and colour. When the corrections are made it is seen that fluoride has no effect at all on the cancer death rate. This same conclusion has been reached by similar studies in Britain and in hundreds of communities all over the world. The non-association of fluoridation and cancer has been established by the US National Cancer Institute (1972), the Royal College of Physicians (1976), The Royal Statistical Society (1977), The International Agency for Research on Cancer (1982), and the Institute of Cancer Research: Royal Cancer Hospital, London (1982). Of course, what Drs Burk and Yiamouyannis are really telling us is that people live longer in towns where the fluoride content of the water has been brought to the correct level. But if people live longer, more of them will die of cancer, simply because cancer is mainly a disease of old age; more than half of those who die of cancer are over sixty-five.

When challenged to answer these criticisms, Burk and Yiamouyannis put up a smokescreen, claiming that their latest report answers all the criticisms – or that they have a secret method of analysing the results (unknown to the rest of the world) which gives support to their theories. Sometimes their friends claim (as one of them did on Radio Scotland a little while ago) that the critics (including Sir Richard Doll, the world's foremost authority on cancer statistics) are now convinced by the arguments of Burk and Yiamouyannis. This is either a slip of the tongue or a barefaced lie.

In the Strathclyde fluoridation case Burk and Yiamouyannis attempted to win the game by playing this particular Ace card but they had no success. The judge, Lord Jauncey, in his report said, 'I found Dr Burk's evidence on this question of cancer deaths to be vague and unimpressive.' And of his partner, the judge commented, 'Dr Yiamouyannis displayed great ingenuity and a very fertile mind during his evidence . . . but I was driven to the conclusion that he not infrequently allowed his hostility to fluoridation to obscure his scientific judgement.' In this final denouncement one can be absolutely sure that this manufactured 'Ace' card is completely bogus.

The whole of this saga typifies the totally spurious approach of the anti-fluoride lobby in its accusation that fluoridation causes other diseases. The Pure Water Association circulates stories about work in Holland suggesting that fluoride causes instant asthma, skin rashes, and in fact all kinds of allergies. The doctor who made these claims refuses to give any scientific evidence for them and they have not been verified anywhere else in the world. If fluoride at one part per million causes so much trouble it is strange that no complaints have come from the millions of people who drink tea or bathe in sea water, since both of these liquids contain one part per million of fluoride . . . or perhaps it is not so strange, since the whole story is utter rubbish.

On the other hand the benefits of adjusting the fluoride level in water to the correct value of one part per million cannot be disputed, except by the mentally blind. The water supply in Birmingham has been fluoridated since 1964 and the

benefits have been spectacular. The number of children attending clinics for the relief of toothache has dropped from over 10,000 in 1965 to under 2000 in 1977. The number of children given general anaesthetics for the removal of teeth – a procedure with some danger – has dropped from 18,398 to 3851 – a reduction of 78 per cent. In Wolverhampton (not fluoridated), during the same period, the number of general anaesthetics actually *increased* by 30 per cent. In Birmingham last year sixty-two children were supplied with false teeth – representing one in 3500 of the school population. In Wolverhampton the corresponding figure was one in 280 of the school population – twelve times higher. Students at the Dental Hospital in Birmingham will soon have to go to other places to gain experience of tooth decay.

The benefits of fluoridation are not confined to children. They continue into adult life, so that natural teeth last longer. Attempts to belittle the benefits of fluoridation are often made by people who don't know – and don't want to know – how fluoride works. It is not a magic remedy for tooth decay fully established. To obtain the benefit, fluoride must be taken from the time that teeth are being formed – very early in life for the milk teeth and a little later for the permanent teeth. The benefits are not the same at all ages because the teeth most liable to decay don't all appear at once. So it is easy to take one figure out of a table of results and read a wrong conclusion.

But of course the anti-fluoride movement contains many people quite willing to resort to this kind of manoeuvre to obtain the answer that they want. Indeed it is a movement with some very odd people. In recent times Health Authorities and Boards have been bombarded with propaganda coming directly or indirectly from the National Health Foundation in California. This organization is well known for its efforts to promote bogus remedies for cancer and for other activities which have violated the law – and been suitably punished in the courts.

This sort of opposition is not new. Every major advance intended to improve the public health – food hygiene, anaesthetics, vaccination – has been achieved only in the face of ferocious opposition and hysterical abuse. The people who suffer while the argument goes on are not the people who do the arguing. It is the young, the poor and the under-privileged who suffer most.

It is sometimes suggested that fluoride should be given in other forms – in school milk, table salt, toothpaste, or tablets. But none of these methods would be effective. Protection by fluoride must begin at birth – not at the age of starting school – if children's teeth are to be saved. Babies and young children don't take much salt – and not all of them use much toothpaste, or can be persuaded to take a fluoride tablet every day from birth to fifteen. In a recent project in England, parents of 3500 children were invited to take part; 759 children started the scheme but after nine months only seventy-nine were still taking the tablets.

It is also said that even something so beneficial as fluoride should not be added to drinking water because that is mass medication. But there is already a lot of mass medication. Many substances are already added to water – chlorine,

copper sulphate, alum, and a lot more – to make it fit to drink. No doubt we could each take untreated water and boil it, chlorinate it and generally purify it for ourselves – but we don't. There are times when individual freedom must give way to the greater need of the community. That's why we have a Parliament – and local councils. It is sometimes claimed that fluoridation of water supplies is illegal.

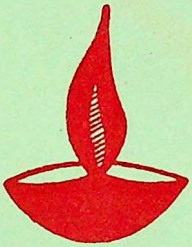
You may think, as you weigh the evidence, that the merchants of fear are quite right – that there is indeed a gigantic conspiracy involving the Royal College of Physicians, the DHSS, the World Health Organization, and every reputable body that has reported on the problem – that all of these people are eager to poison themselves and their children. Or you may agree that fluoride is simple, safe, cheap, and effective . . . that it has been tested more thoroughly than any other measure to improve public health – not only in communities all over the world but for thousands of years in nature's laboratory. You may prefer the evidence and experience of our own reputable experts on cancer and on general health including dental health *rather than that of imported scaremongers and carpetbaggers*.

In conclusion, the petitioners in the legal enquiries held in Eire and in Scotland had every financial and physical opportunity to bring evidence of the dangers and inefficacy of fluoridation, in the form of both documentation and witnesses. Independently both Mr Justice Kenny in Eire and Lord Jauncey in Scotland dismissed the pleas of the petitioners and declared fluoridation to be both safe and effective. Whereas Lord Jauncey ruled on a fine legal point that fluoridation was not legal in Scotland, HM Government have stated their intention to clarify the law for the whole of the UK. When this happens there can be no logical reason for any further delay in introducing this well-proven dental health measure and sounding the death knell of the anti-fluoride movement.

If you vote in favour of fluoridation you will have the admiration and gratitude of the vast majority of your people and of their children for all time to come.

DIS-11.

AN OPTIMISTIC HORIZON IN EPILEPSY



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14, J. Tata Road, BOMBAY - 400 020

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Foreward

The Indian Epilepsy Association Bangalore Chapter is very active in patient education and this booklet is one such venture.

The advances that have taken place in the diagnosis and treatment of seizures have made epilepsy a well controllable disorder, with the promise of a good and normal life for the patients. Information and knowledge regarding seizures is essential for patients and relatives so that fear, ignorance and anxiety may be expelled. To achieve this goal, Prof. Dr. K.S. Mani, a renowned neurologist and an international expert in Epilepsy has written a simple and informative book on seizures which will be most useful. A study of this short booklet will no doubt benefit a large number of people with seizures and encourage them to lead a normal life.

(Sd/-)
Prof. Dr. B. Ramamurthi
Immediate Past President
Indian Epilepsy Association

Madras - 600 004
February 2, 1995

AN OPTIMISTIC HORIZON IN EPILEPSY

General

Epilepsy is a common condition affecting 0.5 to 1% of the general population. No race, caste, community or gender is exempt, but it is probably a poverty-related illness affecting more often the people in the lower socio-economic strata of society. Its prevalence in certain parts of the world like some areas of Africa, Central and South America is very high. It is not caused by devils or evil spirits (fig 1). It is often a result of a scar in the brain - a legacy of difficult delivery, childhood infections, head injury or in the elderly, diffuse blood vessel disease. Less common causes include brain tumours. In certain parts of the world, especially India and Central and South America, worm infestation of the brain as in cysticercosis (fig. 2) and others are common causes. However, it must be stressed that in a vast majority of instances the cause is not known, at least during life or even if known does not alter the total management of the problem.

Seizure Types

Epilepsy is like fever and consists of several types of attacks or seizures. The common type called generalized tonic-clonic seizure (GTCS) or grandmal or major seizure is well known and consists of a sudden cry, fall - if erect - unconsciousness, stiffness and jerking of limbs with eye balls rolled upwards. Froth, tongue bite, passage of urine or injury are common, but not constant. The patients often go to sleep and on waking up have severe headache, vomits, soreness in muscles or tiredness.

Some patients experience a warning signal seconds or minutes before an attack. This may be in the form of jerking of fingers, toes, mouth, or eyes on one side or in the form of simple or elaborate subjective feeling or experience, not obvious to others. These tend to recur and have a constancy of pattern for that individual. These are called Simple Partial Seizures (SPS) and last 1-2 minutes during which the patient is fully alert. In another type called Complex Partial Seizure (CPS) the patients have a partial disturbance rather than total loss of consciousness. They have a vacant stare, champ the mouth, smack the lips, or move the hands and legs in a bizarre, strange

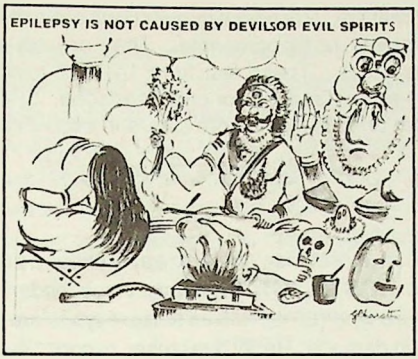


Fig: 1

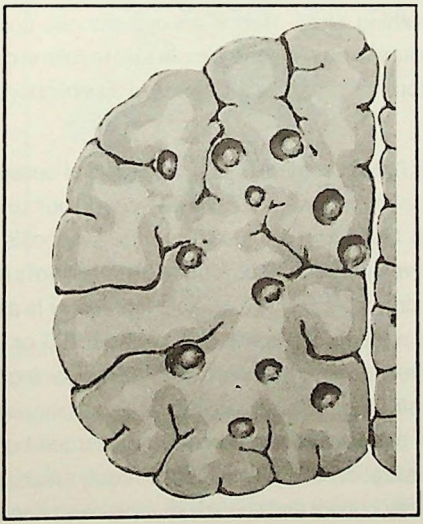
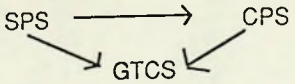


Fig: 2

fashion - automatism. Response to questions are either absent or if present incomplete or inappropriate. They are not aware of the content of an attack. These last from 1/2 to 2 minutes followed sometimes by sleep, tiredness or mild headache. SPS or CPS can remain as such or as is much more common evolve into a GTCS.



The latter can also arise straightaway without a preceding SPS or CPS-primary GTCS. GTCS-primary or secondary is the most common seizure type (80%). Mixed seizure types are often seen in those with brain damage. Repetitive lapses of consciousness lasting 5 - 15 seconds (absence seizures) or jerking of the limbs lasting 1 or 2 seconds only but on a background of full consciousness (myoclonic seizures) are relatively uncommon.

DIAGNOSIS

Epilepsy can be diagnosed only by an accurate description of the attack and nothing else. Hence an eye witness account is very essential. Failure to observe this simple rule results in overdiagnosis of this condition. 15% of cases diagnosed as epilepsy do not suffer from it !

EEG is of help in confirming the diagnosis of epilepsy - but only in about 30% in the tropics - and also its type. In our set up, in nearly two thirds the first EEG is unhelpful in the diagnosis of epilepsy. Likewise in developing countries routine estimation of serum levels of anti-epileptic drugs (AEDs) in a reliable laboratory is a luxury which most - not all - patients can very well do without. CT or MRI scan are of help not in the diagnosis of epilepsy but in determining the presence of underlying structural disease. It should be emphasized that these investigations cannot and should not be discarded but used intelligently with realization of their limitations, cost, reliability and ready availability. They cannot and should not replace an adequate history, follow-up evaluation and a practical common sense approach.

MANAGEMENT

The concept that epilepsy is a chronic recurrent disorder requiring life long medication is no longer true. We know today that in a vast majority of subjects, the attacks can be controlled, if not cured, by early treatment with Anti Epileptic Drugs (AEDs). There are certain rules of the game which must be followed scrupulously. Drug treatment is not for few days or weeks only but years - at least 2-5 years. Not a single dose must be missed and it should be taken at the correct time (fig 3).

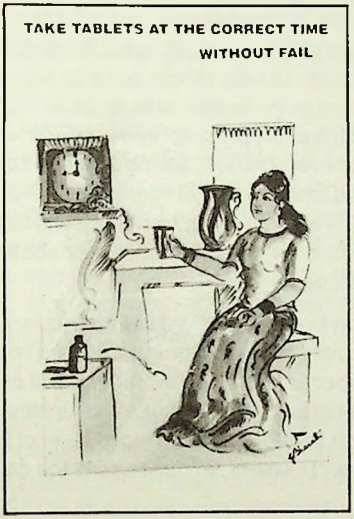


Fig: 3

The drug prescribed depends on the seizure type. In the most common type of seizure-GTCS-Phenobarbitone (PB), Phenytoin (PHT), Carbamazepine (CBZ), or Sodium Valproate (VPA) are equally effective as far as seizure control is concerned. This is a point of practical importance in rural epilepsy control program in developing countries as confirmed by several studies in India, Pakistan, Kenya, Tanzania, Turkey, and Ecuador (in South America). Sedative side

effects or restlessness do occur with PB but not in all cases nor are they considered disturbing in every community and socio-cultural milieu. We must learn to individualize and not generalize. VPA is the drug of choice in absence/myoclonic seizures-fortunately not common. Every parent/patient must learn to recognize his/her seizure type(s) and maintain an accurate diary of not only the attacks but also their types. If properly utilized this can help the doctor to identify the correct drug and dosage for that individual.

There is no fixed dose for AEDs. The drug should always be given as monotherapy (single drug). Polytherapy (more than one drug) more often than not is unnecessary, less effective, more expensive and potentially more toxic. The dosage of the drug chosen has to be stepped up gradually till side effects occur or seizures come under control. In the event of the former, slow replacement by another AED is necessary and the entire process repeated. This exercise may take weeks, months or even years, requiring patience from the doctor and the relatives. The limited value of serum AED level estimations has already been referred to. It is worth emphasizing that PB or PHT can often be given once daily, while CBZ or VPA need to be given atleast in two or three doses per day.

One question frequently asked is why a patient should have an attack at a particular time even if drugs are being taken. This may be from missed dose or time, change of brand, use of drugs beyond their date of expiry (never buy drugs loose), fever, vomits, diarrhea, use of other drugs which interfere with the blood level of the AED or poor/inadequate sleep. However, in most cases the cause remains unknown.

First Aid

A single tonic clonic seizure - the shaking or convulsive phase-lasts hardly 1-2 minutes, though the subject may be unconscious for longer periods. It does not call for any first aid measure. Do not insert any object into the mouth, hold the patient, turn the neck or body during a seizure or pour fluids down the throat. These are all potentially dangerous first aid measures. Leave the patient severely alone (fig.4). This maxim also applies to children who often develop

Fig: 4

seizures during fever. However, if the convulsive phase lasts beyong 10 minutes by the clock, if consciousness is not regained in 1-2 hours, or if there are recurrent (more than 3) GTCS, the patient must be taken to the nearest doctor. Rectal or oral diazepam (calmpose) - solution meant for injection - 1/2 to 2 ml - is a first aid measure at home, but should not be resorted to for single seizure for the simple reason it is unnecessary. Moreover tolerance to the drug tends to develop rendering it useless during a future emergency.

RESULTS

Provided the rules of the game are followed - correct diagnosis, proper identification of the seizure type, institution of the appropriate drug as monotherapy (single drug) and strict 100% drug compliance (not 99% !) - GTCS which are potentially harmful to life or limb can be controlled fully - (100%) - in about 70% of all patients. Half the remainder will have their attacks reduced by more than 50%.

In general, the outlook for full control of seizures tends to become less with each of the following parameters - presence of overt brain damage, often in the form of mental retardation (hardly in 15% of all epilepsies); CPS as the seizure type; poor drug compliance (irregular drug intake); unnecessary polytherapy (monotherapy will do in most cases); choice of wrong drug; failure to control the attacks early (generally life time total of GTCS should not eceed 30-50, when control

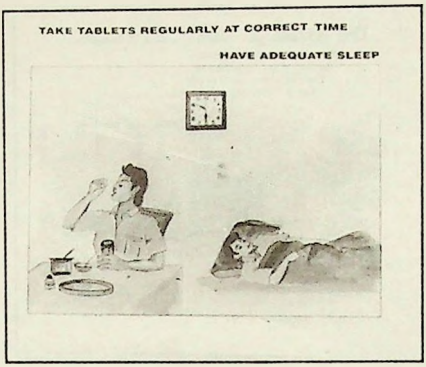


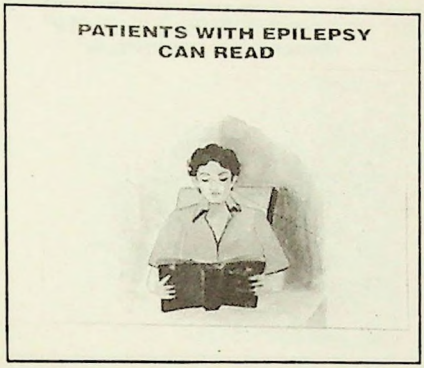
Fig: 5

becomes progressively more difficult) and poor life style compliance, especially with regard to adequancy and regularity of sleep (fig. 5).

In general, drug treatment can be withdrawn slowly over a period of 1-1 1/2 years if the patient is totally seizure free for 2-5 years and has no overt brain damage, though there are certain exceptions. Relapses occur in about 20% of all epilepsies, generally during the reduction phase or within one year of total cessation of AEDs.

LIFE STYLE

80% of patients with epilepsy have no mental or neurological handicap. The patients are normal except for the few minutes during a seizure which may be once an year or even daily - the latter an extermely uncommon event. These patients can study (fig. 6), play ("let the children fall, break their bones which can mend, but don't break their hearts" - Jeavons), work (fig. 7), eat what they like (the so called tonics are a total waste of money). They can marry (fig 8), have children, breast feed the infant (fig. 9) and have full participation in recreational and social activities including sports. (Jonty Rhodes the South African Test Cricketer is a legendary living example). They can cycle (fig. 10). Till the attacks come under control, they should avoid cooking near an open fire when they are alone (fig. 11). It may also be prudent not to lock the door while bathing (fig 12) or go near a tank or an open well without a wall (fig. 13). Otherwise they can lead a



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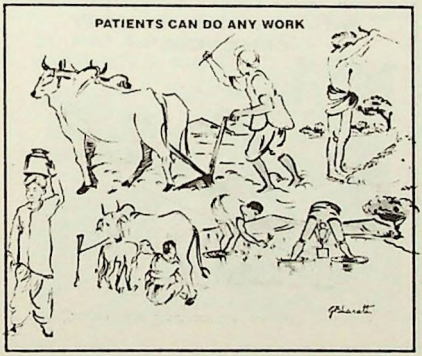

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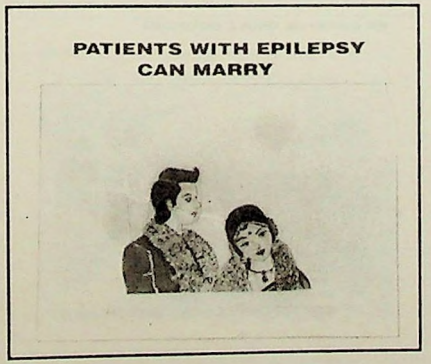

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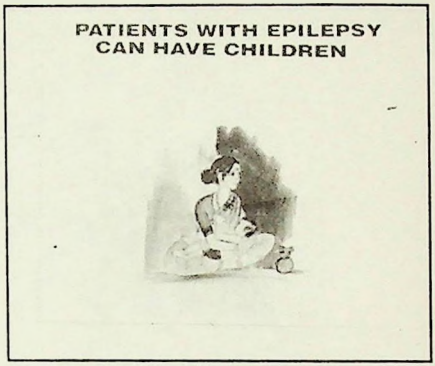

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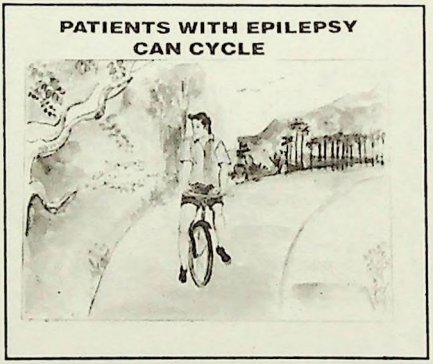


Fig: 10

Fig: 11

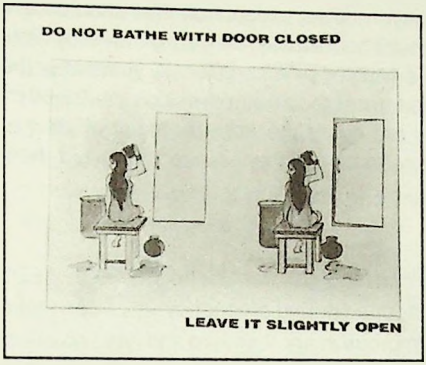
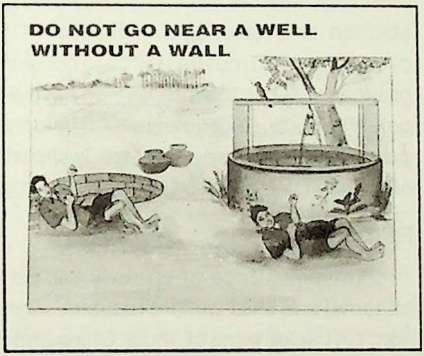


Fig: 12



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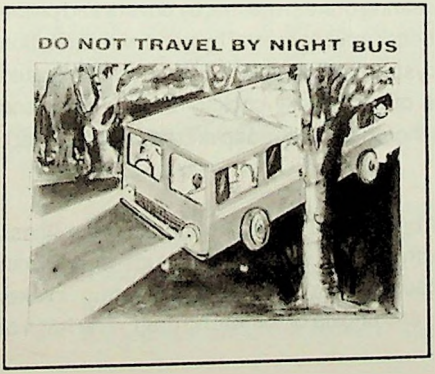

Fig: 14

perfectly normal life like anyone else, except that they should have regular hours of sleep (about 7 hours a day)-beware of travel by night bus (fig 14) - and keep off driving or swimming for 1-2 years after the last attack. Tobacco or moderate (social) intake of alcohol - 2 x 60 ml of whisky twice a week - are not taboo from the point of view of epilepsy, though there may be far weightier reasons to avoid them totally.

PREGNANCY

Women with epilepsy can have children like anyone else. AEDs should not be reduced or stopped during pregnancy which can make epilepsy worse in a third, improve in another third and have no effect in the balance. Repeated seizures during pregnancy can result in abortion or miscarriage. AEDs do have harmful effects on the developing child, but this tends to occur within the first 6 - 8 weeks of pregnancy, when the lady is often not even aware of it. This very low potential to cause developmental defects should not be blown out of proportion. There are other equally important causes. More than 95% of pregnancies exposed to AEDs result in normal bouncing babies.

PREVENTION

The prevalence of epilepsy can be reduced by better primary maternal and child welfare measures including universal immunization. Sri Lanka, parts of India, China and Cuba are classic examples. So also are compulsory wearing of crash helmet by drivers and pillion riders of 2-wheelers and better environmental hygiene and sanitation to reduce the scourge of cysticercosis (infestation from contaminated vegetables, consumption of imperfectly cooked 'measly' pork and failure to wash the hands thoroughly with soap and water after toilet).

CHRONIC EPILEPSY

Chronic epilepsy can be reduced by early treatment of the illness by monotherapy even with simple, inexpensive drugs rather than allow the epilepsy to become chronic by irregular drug intake, unnecessary polytherapy and choice of the wrong drug. The habit of

giving an additional dose by mouth for a single recurrence or change of dose, drugs or doctors for every recurrence deserves to be condemned. We will run out of drugs in no time, though not doctors! Effective peripheral decentralized management of epilepsy with a single drug at the rural level is the key towards prevention of chronic epilepsy in developing countries and can be effectively merged with other disease control programs like malaria, leprosy, tuberculosis etc., to make it cost effective.

What of the 15 to 20% of patients who unfortunately have chronic or drug resistant epilepsy ? The term chronic or drug resistant epilepsy should be reserved only for those who have not responded to monotherapy with PB, PHT, CBZ or VPA in adequate dosage or careful polytherapy with two drugs.

NEVER DRUGS / SURGERY

Newer drugs like Vigabatrin, Lamotrigine or Gabapentin have been introduced into the market in Western countries, mostly as add-on-treatment for years, if not life. They are used mainly in CPS and Lamotrigine also in Lennox-Gastaut Syndrome. The latter is an intractable type of epilepsy in children with brain damage characterised by mental retardation, restlessness and mixed-seizure types including myoclonic jerks and GTCS. Results in CPS show in general full control in 10 -15%, and more than 50% control in an additional 40 -45%. Surgery for epilepsy, especially in intractable complex partial seizures has yet to be placed on a firm footing in developing countries. It should be stressed that AEDs must be continued for at least 3-5 years after surgery and relapses occur in 10-20% of patients.

TEN COMMANDMENTS

These are as under :

1. Epilepsy is an eminently controllable problem.
2. Epilepsy is NOT a mental illness.
3. If you witness an attack, note the details. An accurate description prevents wrong diagnoses. First aid is rarely needed.
4. Early treatment is the secret of success.

5. The drugs prescribed have to be taken regularly.
6. The duration of treatment in general is 2 to 5 years after the last attack.
7. Restrictions for persons with epilepsy are very few; ensure adequate sleep.
8. Patients can study, work and enjoy life.
9. Women with epilepsy can marry and bear children.
10. Treat problems as you would in asthma, headache, diabetes. Do not overprotect nor ostracize.

CONCLUSION

To conclude epilepsy is not a dreadful disease as made out to be. It can be fully controlled in about 70 - 80% and totally cured in 50% of patients by early and proper treatment even with simple, inexpensive drugs. Children and adults can lead a perfectly normal life. The key to success is not drugs alone but attempt on the part of the physician to explain about the disease, drugs and their pros and cons to the patients / relatives. Patient education, often a neglected aspect can be achieved through pamphlets, booklets, discussions, television programs and lay Epilepsy Associations. After all patients / relatives have a right to know about their illness, don't they !

DIS-1

DIABETES

Health Committee
Lok Vidnyan Sanghatana, Maharashtra

What is diabetes? Why does it occur?

When sugar in our blood can not be utilised adequately by the cells in the body, the level of blood sugar increases beyond the normal limit and this is diabetes. In diabetes, not only does the sugar in blood increase but the metabolism of carbohydrates, proteins and fats gets disturbed and a complex disease process sets in.

The food we eat ultimately gets converted in our body into a sugar called glucose. This glucose provides the energy required for the body functions. A hormone called "insulin" is essential for the utilisation of the glucose by the cells. Insulin is produced by some special cells in the organ called "pancreas". In diabetes, this insulin is either not produced in adequate quantities or does not act properly, due to which the cells cannot utilise the glucose for energy. Instead, sugar keeps accumulating in the blood. This results in an increased level of glucose in blood, but the cells are starved of glucose.

There is no one single reason as to why insulin is not produced or utilised properly. To some extent diabetes is hereditary. However, obesity, lack of exercise and improper food habits are known to interfere in the action of insulin. This is seen from the fact that Indians settled abroad with affluent, sedentary, Western lifestyle show a higher prevalence of diabetes.

What are the ill effects of diabetes?

If the level of sugar in blood increases beyond normal limits, it has several ill effects. The fine blood vessels called "capillaries" get damaged due to high blood sugar. This may cause blindness due to bleeding from the capillaries in the retina of the eye. Diabetes increases the chances of cataract or glaucoma (increase in the pressure in the eye-ball).

Diabetes also affects the capillaries in the kidneys, heart, skin or nerves. It also increases the proportion of fatty substances in the blood which in turn leads to a condition called arteriosclerosis (hardening of blood vessels called the arteries). A fatty substance called cholesterol gets deposited on the inner lining of the arteries and thus increasing the resistance to the flow of blood through the arteries. This causes high blood pressure and thereby cause heart-attack.

Increased blood sugar increases the chances of infection as the sugar itself is a good nutrient medium for the infectious agents to grow. Therefore, the chances of fungal infection of external genital organs, or of urine infection or T.B. are more in diabetic persons. The prevalence of impotence in diabetic men is higher. In diabetic women, if the disease is not kept under check during pregnancy, the chances of still birth or of a deformed foetus are more. In fact a diabetic women should attain a good control on diabetes before she conceives.

When should diabetes be suspected?

Diabetes usually occurs after the age of 30 or 40 years. There is the other, more severe form of diabetes in which the production of insulin is grossly reduced. This can happen in childhood or in young age also. In this juvenile diabetes, the patient becomes weak, despite eating frequently. The person remains ever hungry because despite having lot of sugar in the blood, the body cells cannot utilise it (i.e. they remain starved). The muscles and fats in the body get broken down into sugar in an attempt to provide sugar to the cells. However, it is of no use due to lack of insulin. This break-down of muscles, fat causes loss in weight, tiredness and excess sugar gets filtered down into the urine. The sugar also absorbs more water from the blood into the urine. This results in frequent urination and persistent thirst. If this is not attended to, the young diabetic falls seriously ill and may develop coma.

The symptoms of diabetes in older age may often be relatively minor or even absent. These patients have frequent hunger and thirst, frequent urination and tiredness as in juvenile diabetes but in a milder form. The patient has to get up at night for urination. Delayed healing of wounds, tendency to develop pus, itching due to fungal infection, dimness of vision, feeling of tingling, numbness in hands and feet, impotency in men and unsuccessful pregnancy in women.... all these symptoms can be due to diabetes. If close blood relatives are/were diabetic, one should be more suspicious about these symptoms. However, absence of these symptoms does not necessarily mean absence of diabetes. Nearly 50% of diabetics do not show these symptoms. Diabetes can be diagnosed with certainty only through blood test. Therefore after the age of forty years, blood sugar should be tested at least once in five years.

How is diabetes diagnosed?

As mentioned above, diabetes can be correctly diagnosed only by blood sugar test. Test for only urine sugar does not give a certain diagnosis. This is because in some cases urine may show sugar with the blood sugar being normal or in some case blood sugar may be high but sugar may be absent in urine.

The blood sugar is to be examined after the person remains without food, drink (except water) for 10-12 hours. This is called as the "fasting" sugar. If it is more than 140 mg per 100 ml of blood it means the person has diabetes. However, if it is less than 140 mg it does not necessarily indicate absence of diabetes. Therefore in such persons, a test called post-glucose blood sugar is done. In this test the person is asked to take 75 gram glucose in water after fasting for 10-12 hours. The patient is advised not to take anything except water, not to smoke or to exert for two hours after taking glucose. Exactly after 2 hours another blood sample is taken to test the blood sugar level. If the person has diabetes then the blood sugar level would be more than 200 mg/100 ml of blood two hours after the glucose meal. Some time instead of giving glucose, the patient is asked to have normal food and the blood is tested two hours after. The test performed by giving glucose is more reliable for correct diagnosis because we are giving measured quantity of glucose. If the sugar level is found to be 115 to 140 mg in fasting sample or between 140 and 200 mg in the sample after glucose/food the diagnosis of impaired glucose tolerance or diabetes in the offing is made. Under such circumstances further testing or frequent testing or testing after controlling diet and doing exercises may be done on the advise of the doctor.

Besides blood sugar what other tests are required?

If diabetes is confirmed by blood-sugar test a thorough physical examination of the patient including weight, blood pressure, examination of the heart, blood vessels, nervous system, retina is carried out because diabetes can affect these systems. Any problem of kidney function can be detected by simple urine examination. If any problem is detected in this urine-examination, further test can be done to know its extent and severity. Urine sugar is also tested. Although, test for blood sugar is must for the diagnosis of diabetes, once it is diagnosed urine can be tested every week at home by the patient himself/herself to get a rough idea as to whether the disease is under control or not.

Initially the urine is also tested for the presence of substances called "ketones". The presence of ketones indicates that the persons is suffering from severe diabetes. This calls for immediate treatment. Once diabetes is detected tests for blood fats (lipids) and electrocardiogram of the heart are also done. Diabetes is known to induce abnormal changes in blood-fat and in the ECG. These tests are therefore helpful to decide the line of treatment.

TREATMENT OF DIABETES

Health Committee
Lok Vidnyan Sanghatana, Maharashtra

What is the treatment for diabetes? Can it be cured?

Diabetes can not be completely cured and once acquired it stays for life. However, with appropriate exercise, diet control and drugs it can be kept under check. Mild diabetes can be kept under control without taking medicines with the help of proper diet and exercise alone. In addition to diet control and exercise, some patients have to take antidiabetic drugs. In some, only tablets are needed, but in some patients, insulin injection is to be taken daily.

Diet: Indian, especially Maharashtrian traditional balanced diet is mainly based on cereals and pulses. There is no qualitative change required in this diet for a diabetic person. One has to avoid sugar/sugar meats consume only very limited quantities of oil, butter, ghee, etc., take only limited quantities of food to maintain weight within prescribed limits. The approximate formula for prescribed limit of weight is - height in cms minus one hundred = weight in kg. If your weight is more than this limit, with the help of following simple guidelines change your diet to reduce weight.

To reduce 1 kg weight in a month through dieting, one will have to reduce 7200 calories from diet (about 240 calories per day). One hundred calories are provided by the following food items and accordingly the calories can be controlled by appropriate reduction in consumption. Cooked rice - 1 cup - 30 grams of dry rice); one chapati of 6" diameter (made out of 30 grams of wheat); one sada dosa; two idlis; two slices of bread; one cup (katori) liquid dal (in the form of sambar, amti, etc.); one egg; one banana; 2.5 spoons of butter, 2 spoons of oil or ghee.

The following can be consumed in liberal quantities as they have low calories e.g. carrot, cucumber, onion, tomato, lavaki (dudhi bhopla), radish, clear vegetable soup, etc.

The following should be taken only in moderate quantities: (The calories contained in the oil used for cooking have not been taken into account. Oil should be used to the minimum). Vegetables like cabbage, cauliflower, ladies finger, brinjal, etc. Fruits such as papaya, sweet lime, oranges, figs, etc. Fluids such as butter milk and milk without cream, tea without sugar, fish, lean chicken, etc.

The following should be taken in very small quantities: potato, sweet potato, yam, sago, green peas, guava, banana, mango, custard apple, etc.

The following should be avoided as far as possible: Sugar, gud or jaggery, honey, sweets, ice-cream, jams, pastries, cream, ghee, butter, fried foods, oily pickles, groundnuts, almonds, cashew. They are calorie-rich substances.

Alcohol also has calories like sugar. It should be avoided. This is particularly so when the diabetes is severe and not under control or when other complications are present. Eat a little at a time, four times a day so that the sugar level in blood does not rise suddenly.

Use only limited quantity of oil for cooking. Daily 20 grams of oil per head i.e. about 0.6 kg per head per month is the limit. Other fats particularly animal fats such as ghee, butter should be avoided as they are much more likely to increase cholesterol in diabetics. Home made ghee may be used but it should be restricted (only one tea spoon in a day - 10 grams).

Diabetes may also result in other problems relating to heart and kidneys. If this happens, this would need further diet modification (salt restriction, etc), which should be done under appropriate medical advice. There is a general misconception that diabetic should not eat rice. As a matter of fact rice has less calories as compared to chapati or roti (weight for weight). However, since rice has less fibre content its digestion is quicker which may cause a spike in blood sugar after the meal. There is no harm in eating limited amount of rice, as part of a balanced meal.

Exercise: Exercise reduces the amount of insulin required to control blood sugar. With the help of proper diet and exercises it is possible for some diabetics either to avoid use of drugs altogether or reduce its dose. Therefore diabetics must do some light exercises. Many diabetics are overweight and exercise helps to reduce it. Walking is the simplest and the safest exercise. One should walk briskly for at least half hour to stay fit, and for longer time to reduce weight. To reduce 1 kg in a month one has to burn 7200 calories through exercise. Keeping in mind the need to reduce weight to the desired level within 3-6 months, the diet and exercises have to be planned.

Types of activities and calorie consumption per minute: Cleaning household utensils, mopping, gardening, painting - 2 to 5 cal; works like masonary - 4 to 5 cal; brisk walk, climbing stairs, cycling, tennis, carpentry - 5 to 10 cal.

In fact exercises are of three types and the exercise programme preferably should include all the three. Please refer to the separate booklet by the Health Committee on this subject. Diabetics should consult doctor before starting these exercises, and also observe the following precautions.

A patient taking insulin should eat 25 to 30 gms of carbohydrates (half a banana or guava or half cup milk) before exercise. Otherwise exercise may induce sudden drop in sugar level causing giddiness. If the person also has hypertension or heart ailment, only such exercises which can be easily tolerated should be done in consultation with the doctor. However there is no need to consult a specialist if one chooses only brisk walk as exercise. If there is a problem in the retina of the eye, exercises which include jerky movements and lifting of weights should be avoided since they may trigger bursting of already weakened capillaries. Exercise causes quicker absorption of insulin injected on arm or thigh. If the time of exercise coincides with that of the injection, the injection may be taken under the skin on the abdomen.

Medicines: The prescribed medicines should be taken at the right time in correct dosages and should not be changed without medical advice. Diabetes cannot be cured and therefore diet control should always be properly observed. In some cases insulin is needed only for temporary reasons such as during stress like pregnancy or during illness. Once the stress is over, insulin can be replaced with tablets.

Diabetes may cause symptoms such as tingling and numbness or burning or pains in hands and feet. Sometimes 'B' vitamin is prescribed to treat these symptoms but its utility has not yet been scientifically proved. However, due control on the blood sugar can reduce these problems in some patients.

How to know whether diabetes is under control or not?

Symptoms of diabetes recede as diabetes is controlled. The true indication of control of diabetes is level of blood sugar which to some extent can be gauged by testing urine sugar. Urine sugar can be tested at home. Its utility for a particular patient is determined by the doctor depending upon factors like the severity of diabetes and patient's attitude.

If the patient does not have to take insulin it is good to test the blood and urine sugar every month (however, if the patient cannot afford it, then at least every three months) to know whether diabetes is under control. If the patient is on insulin then more frequent testing for blood-sugar is required depending upon the severity of the disease.

The usual blood sugar test indicates whether the sugar has increased beyond normal limits on that particular day. A test called 'Glycosylated Hemoglobin' shows the control on blood sugar during the preceding 3 months. Since this test is more expensive costing about Rs. 150 to 200, doctors do not advice it to all the diabetics.

However, if the doctor suspects that the patient does not take the necessary precautions related to diet, exercise and medicines, this test becomes quite necessary.

In the initial stages of the treatment when there is a need to reduce weight, the patient should keep a note of his/her weight every 15 days for a few months and if the progress is satisfactory, a monthly check-up of weight is enough.

Once in a year it is necessary to do a general physical check up and examination of eyes, urine, blood cholesterol and other lipids, ECG as diabetes increases the possibility of certain problems in the eyes, kidneys, and the heart. These diseases are symptomless in the initial stages and hence they aggravate silently. These tests are therefore needed to detect these diseases at an early stage. This is more so for certain retinal problems which otherwise could cause blindness. This blindness can be prevented by timely check up and treatment of the disorder at an early stage.

What other care is required?

Any increase or recurrence of symptoms of diabetes should be reported to the doctor and be properly treated. Any infection should also not be ignored and a prompt, proper treatment for that infection is a must, as any infection can quickly become serious in a diabetic person.

Specific time table for for food and medicines should be strictly observed. An excess dose of insulin or a powerful oral drug in relation to diet may cause a sudden drop in the level of blood sugar. If this happens, the patient may get profuse sweating, giddiness, uneasiness and even unconsciousness. In this situation 4-5 spoonful of sugar should be taken. A patient on insulin or oral anti-diabetic drugs should carry a small plastic packet of sugar (about 5 spoonful) in his/her packet. Patients taking insulin should carry an identity card with a drug chart indicating the drug used and dosages.

Diabetics should avoid all forms of tobacco as it increases risk of heart diseases. They should avoid walking barefoot, should cut their nails properly and do proper early dressing for all kinds of injuries including small cuts as diabetics are more prone to get their wounds infected fast and develop a septic.

There is no scientific proof that diabetes is either cured or controlled with non-allopathic drugs (e.g. Ayurvedic or Homeopathic). However those who use these drugs on account of their faith in that system of medicine, should periodically keep a check on blood and urine sugar levels to avoid any possible threat to life.

Thus in brief proper care in the form of diet, exercise and medication can keep diabetes under control. This depends more on the patients themselves and the doctor's role is mainly of a guide.

Can diabetes be prevented?

Although diabetes is hereditary, at least to some extent it depends upon one's life style. Lack of exercise, excessive consumption of oily and rich foods, mental tension help to cause diabetes. This should be avoided. Persons who have history of diabetes amongst close blood relatives should be more careful about life style.

There is evidence that in children pancreas may get affected due to malnutrition. This can reduce insulin production resulting in diabetes. The high prevalence of malnourishment in India needs to be overcome. This will also help in reducing the prevalence of diabetes in India.

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CHANGING PARADIGMS OF INFECTIOUS DISEASES - IN DEVELOPING COUNTRIES

New Paradigms of Infectious Diseases

Microbes and man have been engaged in a continuing battle for ascendancy on this planet. In India whatever successes had been achieved on the public health front were largely in the field of infectious diseases. Small-pox is an excellent example. To this will soon be added elimination of polio, guineaworm disease, tetanus in the newborn and leprosy. Despite these successes, the microbial front remains disturbing and is a cause of anxiety. Thirty new diseases have appeared on our planet in the past 20 years. Infectious diseases continue to be the leading cause of death. What diseases were thought to have been conquered or nearly-conquered, have now staged a comeback. Examples are plague, malaria and kala-azar. Diseases such as tuberculosis thought to be controllable through available technologies and for which elaborate national plans of control were developed decades ago, still exist and are getting worse. What has proved to be a new and deadly disease of mankind throughout the world and despite its early recognition soon after its introduction into India in 1985-86, HIV infection in India is rising inexorably and there is a sense of despair. Diseases such as dengue and cholera which are endemic in India with periodic outbreaks from time to time, have now assumed more virulent characteristics. These examples represent a formidable array of emerging and re-emerging infectious diseases and a new paradigm of infectious diseases is now in evidence due to a multiplicity of factors. Recognition of these paradigms and the factors contributing to them is essential for developing effective control programmes. The increasing phenomenon of drug resistance is a part of the paradigm.

Amongst the factors influencing the new paradigms of infectious diseases, there is the age-old factor of poor living conditions which in many areas have worsened for certain sections of the community, often in spite of growing overall economic prosperity.

Other factors include the unintended effects on ecology, at both macro and micro levels, resulting from human activities under the overall rubric of "development". These developmental activities include:

(i) Dams and irrigation projects which are necessary and bring about prosperity may also tend to create ecological conditions, if not foreseen, conducive for re-emergence of old diseases, especially vector-borne diseases.

(ii) Industrialisation and energy-producing initiatives which are necessary for economic growth but also lead to environmental degradation with health consequences.

(iii) Changes in land use patterns and human encroachments of forest areas, a common phenomenon today, exposing human populations to infections with which they have had no previous encounter.

(iv) Unplanned urbanisation and expessive population growth creating optimal conditions for the entry and spread

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of infectious diseases in congested squatter settlements. The essential problem here is the breakdown of infrastructure and services in slum areas creating conditions conducive to the resurgence of diseases once brought under control. Urban health is a matter of overwhelming urgency without much time left to correct the prevailing deficiencies and hazards.

(v) Growing trade, tourism and trucking accompanying increased international travel leading to greater intermingling of people today than ever before creating conditions for the spread of infectious diseases. The speed of transmission of infections increased enormously as mankind transited from sailing ships to jet planes.

The changes in climatic conditions now reveal significant health effects. Atmospheric pollution, deforestation and ozone depletion play a key role in global warming. This in turn, would lead to higher surface water evaporation rates with greater rainfall and heavier monsoons in key areas of the planet. This would alter everything from migration of birds, habitat ranges of insect vectors of disease and the availability of arable land for agriculture. Dengue and malaria transmitting vectors are sensitive to rainfall and ambient temperatures. As a result of global warming, malaria may spread further up the foothills of the Himalayas. The lesson of macro-ecology is that all life forms and chemical systems are closely linked in complex ways[1]. It would appear that current and anticipated changes in local and global ecologies would favour some microbes and their insect vectors.

In place of the Cold War there are now multiple local wars and conflicts which along with natural disasters, such as famines and floods, lead to mass movement of people internally within countries and across borders. The living conditions of the refugees provide a fertile ground for infectious diseases.

Changes in human lifestyles and behaviour including sexual behaviour and food habits are another potent factor in the changing paradigms of infectious diseases. Lastly, the continuing inadequacy in the quality and outreach of health services is an important factor compounding the situation.

The challenges presented by the New Paradigms of Infectious Diseases can be conveyed by the following four diseases.

Dengue

Dengue, by the 14th of October 1996, had caused 126 reported deaths and 2,545 cases of presumed dengue fever complex admitted to public hospitals giving a mortality rate of 5 per cent. While this corresponds to the average rate of mortality in other parts of the world, there may be scope for it to be reduced further by improved case management through early recognition of haemorrhagic manifestations and shock, prompt and efficient replacement of lost plasma through fluids and electrolytes, plasma/plasma expanders and platelets as indicated. Blood transfusion will be needed if there is internal bleeding. The modern regimen of treatment of Dengue Haemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS) was evolved by Thai physicians, some years back and now forms the basis of WHO recommendations[2]. This regimen has been shown to reduce the mortality rate to 2.0 per cent or less.

Dengue had been known to be endemic for over two centuries in India and for the most part had been running a benign self-limited course. All the four known serotypes of the dengue virus are now known to be in circulation. The disease has lately changed its course manifesting itself in a proportion of cases in a severe form of the disease, DHF/DSS. This new lethal manifestation of an old benign disease broke out in Manila in the Philippines for the first time in 1953; then attacked Bangkok in Thailand in 1958; Havana, Cuba experienced the worst DHF known to mankind in 1981. DHF had been raging in our immediate neighbour Myanmar since 1970. Cambodia had seen a severe outbreak of DHF last year. In Manila, dengue occurred each year after the rains. Seasonal and cyclical epidemic pattern of dengue with DHF/DSS in a proportion of cases is a recent phenomenon developing in India and Sri Lanka in the same manner as happened in the Philippines, Thailand and Indonesia. In other words, there has been a westward movement of this new paradigm into India, Pakistan, Sri Lanka and Maldives in the 1980s and early 90s in which Dengue 3 (DEN-3) had been the predominant serotype. The history of Indian dengue illustrates the well known transition from a paradigm of small outbreaks to a paradigm of major outbreaks with DHF/DSS[2-4]

The new dengue disease paradigm has now secured a firm foothold in India. The paradigm is manifesting itself extensively in Latin America and the Caribbean since last year. Its emergence as a major health problem has been most dramatic in the American region. This region had an excellent record of eradicating *Aedes aegypti* in the 1950s and 60s as a part of the Yellow Fever Control Programme, but with the discontinuation of this Programme from the 1970s, Aedes returned, and the worst ever outbreaks of dengue are now raging there. This shows that even a year's slackening of vector control measures could bring back outbreaks of

vector-borne diseases. Similarly, relaxation of immunisation can result in re-emergence of disease as in the Russian Federation and the former USSR with respect to diphtheria. Aedes is a very efficient epidemic vector of both dengue and yellow fever[3,4]. The virus type responsible for the current outbreak in Delhi has not yet been identified but work in this area is going on.

The reasons for this dramatic emergence in India and around the globe of dengue/DHF as a major public health problem, as already indicated, are many: Ineffective mosquito control programme, major demographic changes–the most important being uncontrolled urbanisation, excessive population growth and urban decay characterised by substandard housing and inadequate water and waste disposal system. Increased travel by airplanes produces constant exchange of dengue viruses.

There is a crisis approach to public health in our country, public health activities being largely punctuated from crisis to crisis. What is needed are sustained programmes for the prevention of epidemic transmission. Surveillance systems are inadequate as the Technical Advisory Committee's (TAC) Report on Plague demonstrated. Mosquito densities in relation to rainfall and ambient temperatures are not available and early detection and reporting systems are inadequate. No dengue vaccine is available although a quadrivalent attenuated live vaccine representing all four serotypes has been developed in Thailand and is in advanced stages of trial. This vaccine appears to be safe and effective but it will probably be another 5 years before an effective dengue vaccine will be available for public use. Prospects for reversing the recent trends in dengue/DHF do not look bright in Delhi and early breakthroughs may not happen. And yet, there is no cause for despair.

In response to the recommendation of the TAC on Plague, a National Apical Advisory Committee for Disease Control through Surveillance and Response has been established by the Government. Treatment regimens have been developed with respect to dengue[2], control of the disease is possible through efficient management of the disease and through a double-pronged attack on mosquito vectors, namely, insecticide spraying by Government action and elimination of mosquito breeding by family and community action to reduce sources of larval breeding. The latter strategy is particularly important, especially in the long run and one would like to see social mobilisation efforts in much greater evidence.

The most effective means of dengue vector control is environmental management through physical transformation of largely man-made vector habitats, within and around human dwellings. Education and community participation in planning, execution and evaluation of these control measures is the critical factor. Commonsense approach is needed. Aerial spray is often used, where extensive areas must be treated in a short period, but spectacular results may not be expected as the Aedes rests indoors. The approach must be complemented with larvicidal application in and around homes where containers cannot be emptied or covered. The most effective way to control Aedes is larvicidal source reduction by eliminating clean water holding containers that serve as larval habitats[4]. The 1981 Aedes eradication campaign in Cuba depended upon reduction of larval habitats in artificial and natural containers of water, intense public education, and biological and chemical control methods. The Tanzanian campaign consisted of source reduction, education and clean-up campaigns. Dengue control is an area where Science and Society meet. There is considerable expertise on this aspect within the ICMR in India which must be mobilised.

Pro-active, laboratory-based surveillance systems that can provide early warning of an impending dengue epidemic must be developed. An encouraging development is the Thai dengue vaccine which could be available in the next few years. Another possibility is the use of genetically engineered mosquitoes which cannot transmit the disease.

Malaria

After the initial successes India is now unable to get the upper hand in malaria control and over the past decade or so the number of malaria cases has remained unchanged with the addition of increase in falciparum malaria. The issue today is that malaria has now entered new ecological niches[5]. The problem is essentially due to man-made environmental alteration which provides excellent opportunities for the mosquito vector to breed. The new paradigm of malaria consists of new eco-types such as irrigation malaria, urban malaria, development project malaria, migration malaria and border malaria[5]. These eco-types are in addition to the already existing eco-type of rural malaria in rain-fed areas. Environment degradation is conducive to the sustenance of malaria. Epidemics are increasingly common such as that currently in Mewat, Haryana. The revised malaria strategy consists of early diagnosis and prompt treatment and focuses on local transmission dynamics and control based on selective and sustainable technologies. But there is a wide gulf between the fine experimental projects involving source reduction through biotechnological methods and their extension into nation-wide malaria control programmes.

Malaria was one of the first infectious diseases to be treated successfully with the drug, quinine, extracted from the bark of the cinchona tree. The latest and highly effective anti-malarial compound is also derived from plants– *Artemisia annua* L.–for chloroquine resistant malaria. Forty per cent of the world's population is still at risk and is yearning for a vaccine, cheap and effective. There is not much private investment in malarial research throughout the world and most of the work is done under Government sponsored and charity auspices. Drug resistance is a real problem and hence the different strains in different geographic areas may require different drugs. It is disappointing that the antimalaria vaccine developed by Dr. Manuel Patarroyo, has now been shown to be not effective. There were initial successes with this vaccine, but only partially to the extent of 31-34 per cent protection but more recent studies in the Gambia and Thailand confirm the failure of the vaccine. The Patarroyo vaccine is a harbinger of better vaccines to come based on multi-stage antigens.

Cholera

A completely new strain of cholera vibrio had emerged in India in 1992 in Madras. It has been christened *Vibrio cholerae* 0139 Bengal, with pandemic potential. It produces a clinical picture of severe disease, dehydration and hyponatraemia similar to classical cholera but is susceptible to some antibiotics. It has spread throughout India and went to Bangladesh in 1993 and then on to South West China, Thailand and other parts of South-East Asia. What happens to the Vibrio in between the outbreaks is not known and some scientists had postulated that the Vibrio is dormant between outbreaks in the Hilsa fish in the Hooghly. No confirmatory evidence had been forthcoming for this hypothesis but algal blooms have been found to be responsible for the spread of El tor cholera to Latin America in 1991, a continent that had not experienced cholera over the last 100 years. Algal blooms where cholera organisms reside, are, in effect, "giant floating gene pools" in which antibiotic resistance, virulent genes and plasmids move about freely between viruses and bacteria. In the paradigms that are being discussed, marine eco-systems influencing human health should find an important place. It would appear that current and artificial changes in global ecologies favour microbes and their insect vectors.

Tuberculosis

With the increase in the incidence of AIDS, tuberculosis is becoming worse. The efficacy of BCG vaccine in preventing tuberculosis remains variable depending upon geographical and epidemiological factors. A better vaccine than BCG is needed although the disease is still treatable with existing technologies just as HIV/AIDS is preventable. India has not derived maximum benefits from the results of research at the Tuberculosis Research Centre in Chennai with respect to multi-drug and domiciliary therapy, though it is now proposed to use short course multi-drug therapy against tuberculosis throughout the country. Case management with such a therapy, case finding with high quality bacteriological examination, improved programme management and directly-observed treatment can lead to a virtual cessation of transmission of tuberculosis in the general population even under adverse conditions.

DNA Vaccines

Multi-drug resistance is rapidly advancing in India and gradually assuming serious proportions. New drugs and new vaccines are needed to fight tuberculosis effectively. It is noteworthy that mice injected with plasmid DNA encoding a single mycobacterial antigen (65 kDa) developed immunity against subsequent challenge with the tubercle bacillus. It seems likely that a DNA vaccine against tuberculosis might replace the BCG. In animal models of several infectious diseases, DNA vaccines seem to be broadly applicable for conferring protective immunity to a number of infectious pathogens. They seem not to need replicating vectors and adjuvants and seem to be practical and safe. Eventually, a cocktail vaccine, DNA plasmid-based may be needed encoding for a number of mycobacterial antigens to generate complete and lasting protection against tuberculosis.

DNA vaccines may help man to be one step ahead of microbial pathogens which are either staging a comeback or evolving into more virulent organisms. Surely, the biological front is never static. To foresee is to govern.

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