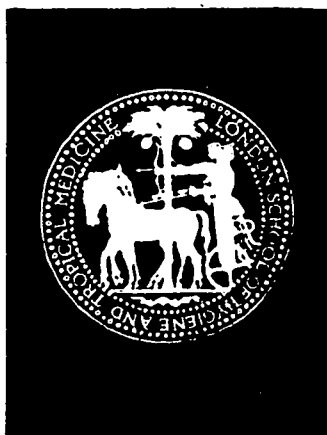


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**THE ROSS  
INSTITUTE**  
**INFORMATION AND  
ADVISORY SERVICE**



BULLETIN No. 9

OCTOBER, 1982

**THE INFLAMMATORY  
DISEASES  
OF THE BOWEL**

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## INFORMATION AND ADVISORY SERVICE

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**T**HE primary object of the Ross Institute is the prevention of disease in the tropics. In the course of working towards this end it has become apparent that the co-operation of industry is essential if rapid progress is to be made. Fortunately, this co-operation has never been lacking, for those responsible for directing tropical industry were quick to appreciate the immense value to them of healthy labour and have therefore been among the strongest supporters of the Ross Institute since its inception.

For this reason the Ross Institute has made it an important matter of policy to keep tropical industry informed of the progress of medical knowledge, and of the practical methods by which the greatest benefit may be obtained from its application. This series of bulletins, which have been specially written for non-medical people, is one of the means by which this information is made available; other publications are issued from time to time and a list of those now current will be found on page 23.

The Ross Institute invites all those whose work is connected with the tropics to refer to it on any matter concerned with health or welfare in tropical countries. The Director and his staff will answer as promptly and as fully as possible all inquiries and requests for advice.

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# THE ROSS INSTITUTE INFORMATION AND ADVISORY SERVICE

Bulletin No. 9

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## The Inflammatory Diseases of the Bowel

By the very nature of the dramatic symptoms which they present, the inflammatory diseases of the bowel have claimed the attention of man since the days of Hippocrates. Though it is now recognised in all countries that these are diseases of filth, flies and food or, in technical language, of an insanitary environment with the associated polluted water sources and contaminated food, the drama remains to the public in the occasional unheralded epidemics of cholera and to the individual in the mundane occurrence of dysentery so well depicted in a few words by a Dutch physician in Java in the 17th century as 'an ulceration of the intestines with a perpetual purging, at first mucous, and afterwards bloody and lastly purulent, with intolerable pain and griping of the belly.'

Supreme effort by national and indeed international health organisations render outbreaks of cholera controllable but not before numbers of people have contracted the disease and many have died. Prevention of the occurrence of the enteric or typhoid group of intestinal fevers is best maintained by the purification of centralised water supplies or in the individual by inoculation with the well-known TAB vaccine. The dysenteries, a miscellaneous group of inflammatory infections of the bowel, remain to a great extent uncontrolled throughout those countries in which climatic conditions at some time of the year favour the transmission of these diseases—uncontrolled, yes! but not uncontrollable, if man so chooses.

### The Intestine

The alimentary tract of the human body is a muscular tube commencing at the throat as the gullet which conveys food and fluid to the sac-like dilation or stomach; then follows some 21 feet of tortuous and freely movable small intestine with a diameter in the adult of 1.5 inch; this opens into the large intestine of considerably greater diameter and 5 feet in length which terminates at the anus. The stomach and small and large intestine together with the liver are the major organs filling the abdominal cavity; this is separated from the cavity of the chest, containing the heart and lungs, by the muscular diaphragm. (Figure 1.)

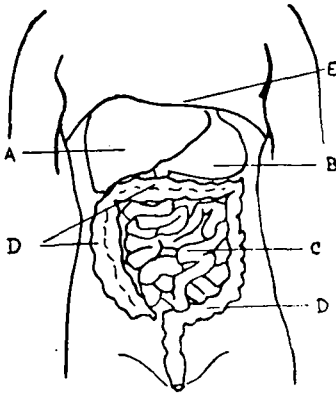


Fig. 1

**THE MAJOR ORGANS  
WITHIN THE  
ABDOMEN**

- A Liver
- B Stomach
- C Small Intestine
- D Large Intestine
- E Diaphragm

The internal lining membrane of the small bowel performs the double function of (a) secreting various ferments to assist in the digestion of the contained food and (b) absorbing the nutrient elements from the digested food ; this absorption occurs mainly in the small intestine. The very fluid residue passes into the large intestine ; here the interior lining is designed to absorb the excess water and so concentrate in bulk the waste material and store it until voided. The contents of the bowel are moved along by rhythmic muscular contractions of the bowel itself which have the effect of a continuous succession of ' travelling squeezes ' ; when these become excessively active, griping pain is felt. In the large intestine mucus is secreted by the lining membrane to lubricate and so facilitate the onward passage of the now more solid content of the intestine.

Whereas the upper part of the intestinal tract is relatively free from bacteria, the contents of the large intestine favour the propagation of an enormous number of a wide variety of organisms. There is now evidence that these bacteria may take an important part in the synthesis of vitamins which are essential to the general processes of the body.

It is obvious that any inflammation or ulceration of the lining membrane particularly if extensive, must have a profound effect on health through (i) interference with the assimilation of nutrients and (ii) absorption of the toxic products of the bacteria causing the inflammation.

**Carriers**

A feature of the inflammatory diseases of the bowel is the presence in the voided fæces of the organisms which are the cause of the illness ; they come from the inflamed or ulcerated lining membrane of the bowel ; they are virulent. In other words, the discharges from the sick person are highly infective to others.

As the patient overcomes his illness and enters convalescence the inflammatory foci in the bowel subside and heal ; the causal organisms gradually decrease in numbers until eliminated. This subsidence process is gradual and may last many weeks after the patient feels relatively fit ; during this time he or she continues to be infective to others and is a *convalescent carrier*. Occasionally after acute illness a patient does not recover and the disease may become chronic but accompanied by periodic exacerbations. In a small proportion of people who have virtually recovered from illness, small foci of ulceration persist almost indefinitely: such persons are *chronic carriers*.

Some people become carriers without ever suffering from symptoms of disease.

Adequate treatment with modern drugs curtails the spread of inflammation of the bowel by killing the infecting organisms ; healing occurs fairly quickly in most instances, the period of convalescence is shortened and a persistence of the 'carrier' state is obviated.

#### PERSISTENCE OF THE CARRIER STATE IN SOME BOWEL DISEASES

	<i>Cholera</i>	<i>Typhoid fever</i>	<i>Bacillary dysentery</i>	<i>Amoebiasis</i>
Tendency to indifferent recovery and chronic disease ... ..	No	No	Occasional	Frequent
Presence of carrier state:				
(i) during illness ...	Yes	Yes	Yes	Yes
(ii) during convalescence	May be weeks	May be weeks, occasionally longer	Up to 10 days, occasionally longer	May be many months, occasionally years
(iii) after apparent recovery ... ..	Sufficient to keep the infection going between outbreaks			

#### ACUTE DIARRHOEAL DISEASE

Diarrhoea (excessive looseness of the bowels) is the cardinal sign of most infectious enteric diseases. It is most frequent in children under five years of age among whom occur most of the deaths attributed to these infections. Where acute enteric infections are well controlled the infant mortality is generally less the 30 *per mille* live births but where these conditions are common the infant mortality usually exceeds 100 *per mille*.

Some enteric infections such as cholera, typhoid and dysentery are separable from the other diseases of the bowel because they are characterized by particular epidemiological and clinical features. The major form of enteric infections, however, is that which manifests itself as 'acute diarrhoeal disease'. This condition is a clinical entity which is believed to have its basis in bacterial infection and *Shigella* is accepted as its most common cause. Other enteropathogenic organisms may also cause it but more often than not no definite infectious agent can be identified. In spite of this, the spread of the condition in communities and families and its diminishing incidence with increasing age all clearly point to infectious origin.

Perhaps the mildest infection is the so-called acclimatization diarrhoea, common in Egypt, India and the East and known by some local name such as Gippi-tummy; minor constitutional reaction is associated with six to ten evacuations in 24 hours over a period of a few days; blood and mucus may be seen in the stools.

The treatment of these cases aims at the eradication of the causal organisms, and the replacement of the fluid and salts lost to the body through the diarrhoea. The management of dehydrated patients involves the intravenous administration of correctly balanced solutions of certain sodium and potassium salts, but to do this effectively and safely special training is required.

## CHOLERA

Cholera is an acute intestinal infection which in former times spread periodically along the trade routes from Asia to Europe and America. Then it was confined for several years to Bengal, Burma and China and its incidence there was diminishing so that hopes of its eradication were being entertained. Recently, however, the El Tor variety of cholera became established in Sulawesi (the Celebes) and from there spread across Asia to Taiwan and Korea in the east and to the Caspian and Euphrates in the west. Overcrowding and insanitary conditions combined with poor sewage and waste disposal practices favour the spread of cholera.

### Transmission

Man is the sole source of the infection and he is responsible for its dissemination. A typical case of cholera passes about 4 gallons of stool and he vomits copiously. As both his vomit and his stool teem with the causal vibrios he is capable of polluting a large area if he is not promptly isolated. Asymptomatic infections occur about five to ten times as frequently as overt cases and, though they secrete considerably fewer germs for shorter periods, their numbers and their freedom of movement make them a serious menace to public health. At one time carriers were considered unimportant but they are now thought to be responsible for keeping the infection going between outbreaks. Water and food become contaminated by soiled hands or utensils, or by flies, and then

act directly as infecting agents. The initial wave of a cholera epidemic regularly starts from polluted water and then spreads by contaminated food or drink, by soiled hands, flies, etc.

### **The Illness**

The severity of cholera varies from infections producing no clinical signs to those in which death occurs soon after onset. The majority of overt cases are severe and start suddenly with profuse purging which is not accompanied by any colic or straining—the 'painless passage of pints of pale fluid.' This immediately empties the bowel of all faecal matter and thereafter the stool consists of slightly opalescent liquid with small white flakes in it—the so-called 'rice-water stool' of cholera. The onset of diarrhoea is soon followed by vomiting in which liquid gushes quite uncontrolled from the mouth. The vomit and the stools are of similar composition and, as they are highly infectious, they are a menace to careless and unwary attendants on the case.

The loss of so much body fluid rapidly produces dehydration with hollow cheeks and sunken eyes; the whole body shrinks and the skin becomes clammy and inelastic; thirst is extreme and the patient, though mentally clear, is anxious and restless; the secretion of urine is markedly diminished or ceases; severe and painful muscular cramps of the extremities and the abdomen soon occur and the patient's condition resembles medical shock. Death may intervene at this stage or the vomiting and purging may gradually disappear, and be followed by complete recovery in a matter of a few days. Unfortunately an attack of cholera provides only a short term protection against another attack.

Previously about 75% of cases in the explosive epidemics died but with modern treatment promptly and properly applied, the mortality is very low.

### **Treatment**

The immediate need is to replace the fluid and the salts lost by the patient before admission and then to maintain his body's fluids and salts in balance by carefully measuring and replacing intravenously all that is lost as the case continues. This requires clinical skill. In addition tetracycline, one gramme for adults, half that dose for children, should be given daily in single or divided doses for five days. (The duration appears to be more important than the dose for preventing the carrier state.)

As soon as an outbreak starts a fully trained team with its special cholera cots and its intravenous equipment should be mobilised.

**Control** of spread of cholera is to be obtained by:—

- (a) early detection and isolation of cases and suspects;
- (b) immediate disinfection of all the patients' discharges and their protection from flies.

- (c) family and other close contacts should be given tetracycline, one gramme daily (*vide supra*) for five days.
- (d) ensuring safe water (superchlorination etc.), uninfected food (strict sanitation of markets, eating houses, dairies etc.);
- (e) control of flies (particularly near the hospital, the latrines, the kitchens and all the eating places);
- (f) mass inoculation (cholera vaccine gives a good degree of protection for about 3 months—and its use greatly restricts an outbreak);
- (g) educate the people in hygienic methods, and enforce them by temporary regulations where necessary;
- (h) isolation of travellers from cholera-infected areas and their treatment with tetracycline would greatly reduce the risk of introducing the disease into a new area;
- (i) the occurrence of cases must be notified immediately to the local health authorities and they must warn the World Health Organization.

### THE ENTERIC FEVERS

The group of 'Enteric Fevers' consists of typhoid fever and the paratyphoid fevers A, B and C.

#### Typhoid Fever

Typhoid fever is a communicable disease associated with continuous fever, involvement of lymphoid tissue, enlargement of the spleen, a rash of rose-coloured spots on the trunk and diarrhoea or constipation either of which is accompanied by abdominal distension and tenderness. The fever is associated with the presence of the causal organism in the blood and with a toxæmia which may be severe. It affects only man and it is his habits which keep it going.

Typhoid is less common in children under 5 years of age.

**Distribution:** Typhoid fever is world wide; paratyphoid A is more common in the East and paratyphoid B in Europe. Paratyphoid C cases are few but may occur in the northern part of South America (e.g. Guyana).

This group of diseases is common where standards of personal and public hygiene are low and rare where they are high. Its incidence thus forms a useful indicator of the prevailing standard of sanitation.

**Causal Organism:** *Salmonella typhi* (the typhoid bacillus): and *Salmonella paratyphi* A, B and C in the case of the paratyphoids. The bacilli are present in the blood during the first two weeks of the illness and in the faeces and urine after the second week. It is a parasite of man only.

During the incubation period the *S.typhi* invade and multiply in the lymphatic tissue of the small bowel and the neighbouring lymph



glands. They then invade the blood stream and localise in the spleen, bone marrow and gall bladder. The bacilli are also present in the faeces and urine generally from the second week, during convalescence and for a variable period thereafter.

Outside the body the bacillus rarely survives in water for 7 days and it often dies within 2 days: in sewage it may live for 12 days and in a septic tank for 14 days; in ice cream for as long as 1 month and on butter for up to 2 months. It multiplies in fresh milk but is quickly destroyed if the milk becomes acid (*vide* WHO Tech. Report Series No. 288, 'Enteric Infections' 1964).

**Source of Infection** is man, i.e. a frank case of typhoid or a carrier.

**Carriers** play an important part in the spread of the disease depending on their standards of personal hygiene and whether they have anything to do with the handling or preparation of food. About 50% of typhoid cases continue to excrete the *S.typhi* in their faeces for about 3 weeks and about 10% for 3 months after recovery (i.e. convalescent carriers). Some 2% to 5% become permanent carriers.

**Routes of Transmission:** Infection results from swallowing food or liquid that has been contaminated by faeces or urine, or through using contaminated water to wash raw vegetables, milk containers etc. Soiled hands are a common source of infection—they may infect food or utensils. Raw fruits and vegetables are important factors; so also are milk, milk products, and shellfish. Flies often spread the disease and the phrase 'faeces, flies, food' applies particularly to typhoid.

**Incubation Period** is usually 10 to 14 days, but may be as short as 7 or as long as 21 days.

**Clinical Description:** The onset is usually insidious and is associated with loss of appetite, general weakness, a frontal headache, and some intestinal upset. The temperature is of the 'staircase' type, rising daily with small remissions till it reaches 103° or 104° by the end of the first week. The pulse is comparatively slow and bleeding from the nose is common. The symptoms gradually get worse and a varying degree of prostration occurs. The abdomen is distended and tender and there may be diarrhoea or constipation but diarrhoea is general by the seventh day.

Between the 5th and 7th days a rash of small rose-coloured spots (2 to 4 mm in diameter) appears on the abdomen and flanks and is confined to that region. Each spot lasts about 4 days and then disappears and a new crop of spots appears over the next 10 to 18 days. The spots fade on pressure, but in severe cases purpura may appear. About the 10th day the Widal reaction becomes positive.

There is a decrease in the number of white blood cells and the spleen becomes enlarged and palpable.

During the second week the temperature remains high with slight morning remissions. The pulse becomes quicker; the blood pressure falls; the toxæmia increases and is often associated with a low muttering delirium.

Commonly some catarrhal bronchitis is present during the first week and is followed by some congestions of the base of the lungs.

The abdominal distension and discomfort become more marked and the loose stools (like pea soup in appearance) become darker as a result of bleeding from the ulceration of Peyer's patches (i.e. the islands of lymphoid tissue in the wall of the bowel).

During the third week the patient's condition may improve; his symptoms diminish and his temperature fall gradually with daily remissions. In other cases the abdomen may become more painful and more distended. It is during this week that the serious complications of hæmorrhage and/or perforation of the ulcers of the bowel may occur. These cause a sudden collapse. The pulse becomes rapid and thready, and signs of peritonitis may appear.

It is also about this time that increasingly severe toxæmia may cause the 'typhoid state', i.e., a low muttering delirium or stupor, with muscular twitching or picking at the bed clothes. The toxæmia may also cause myocardial degeneration and this is a common cause of death.

During the fourth week favourable cases become convalescent. In other cases lobar pneumonia or thrombophlebitis may occur as complications.

In some 10% of cases relapses occur about 10 days after the end of the primary attack. These relapses resemble the initial attack but they are usually milder in degree and shorter in duration. Occasionally there is more than one relapse.

Recovery confers a lasting immunity.

**Treatment:** The specific treatment is the administration of chloramphenicol by mouth (by injection if the patient is vomiting) and the dose is 3 grammes a day in divided doses for 7 days, followed by 2 grammes a day for another 7 days. The temperature generally returns to normal in 48 to 72 hours and recovery follows. If chloramphenicol fails it is well worth trying ampicillin (Penbritin).

**Prevention:** Safe water supplies—chlorination for the community, boiling for the individual or family.

Sanitary disposal of human excreta.

Proper sanitary control of food supplies—e.g. pasteurisation (or boiling) of milk, whether it is used for direct consumption or for making butter, cheese, ice cream etc. Correct storage and marketing of food stuffs. Sanitary control of the preparation of food.

Control of flies. (*Vide* Ross Institute Bulletin No. 5.)

Immunisation (T.A.B.) of people going to areas where typhoid is endemic. T.A.B. (acetone killed) gives 88% protection; phenol-heat-killed gives 65% protection. Those still at risk should have a booster at the end of 2 years. Immunisation against typhoid alone is also possible but the protection afforded by T.A.B. against paratyphoid is probably worth while.

The medical authorities will search for typhoid carriers and deal with them.

Health education on the value of good personal hygiene is usually required.

### **Control of an Outbreak**

Isolate the case in a fly-proof room. Strict current disinfection. Immunise contacts and the local community with T.A.B. Chlorinate the water supplies. Families should boil their drinking water. Careful personal hygiene—e.g. washing hands after going to the latrine and before eating. Check all milk supplies. Avoid uncooked food that cannot be properly washed.

## **BACILLARY DYSENTERY**

If particular bacilli of the *Shigella* group gain access to the intestinal tract through food contaminated with them and survive the acidity of the stomach and the digestive fluids of the intestine to reach eventually the large intestine, they can there propagate with extreme rapidity, in some instances almost replacing the normal bacterial flora. In the process of their existence in the bowel they liberate toxic products which are absorbed into the general system giving rise to the early symptoms of dysentery. Coincidentally the dysentery bacilli invade the bowel wall setting up inflammation and ulceration; with this there is an excessive production of mucus and even bleeding into the contents of the bowel and the typical symptoms ensue, including frequent evacuations, griping pain and straining.

### **The Illness**

The severity of the illness which follows depends largely on the virulence of the strain of the infecting dysentery bacillus. A typical acute attack lasts about three weeks with fever, severe diarrhoea (twenty or more stools a day), acute griping pain and persistent straining to empty the bowel, and stools which contain little other than bloody mucoid discharge. Recovery usually ensues but some cases relapse into chronic intractable dysentery.

Fulminating or severe attacks with vomiting, severe headache and collapse occur in times of epidemics often associated with prisoner of war or concentration camps or ill-nourished communities; with such cases fatalities are common. A natural immunity in individuals or races does not exist.

Chronic bacillary dysentery is a chronic diarrhoea with occasional recurrences of acute attacks involving the passage of blood and mucus; obviously, persistent ill-health is a sequel.

An attack of bacillary dysentery confers a certain degree of immunity, the duration of which is variable but is not likely to be longer than four months. However, fresh re-infections with dysentery bacilli in small doses are apt to occur in tropical countries without causing any obvious symptoms of illness; such fresh doses of organisms may support and maintain any previously acquired immunity.

Although common at all ages amongst indigenous peoples, bacillary dysentery has its most serious effects on children particularly those under five years of age and mortality is highest in this age group.

Children are the principal reservoir of infection in tropical communities; bowel evacuation is indiscriminate in and around dwellings and as the bacilli of dysentery are present in the stools during and for many days after an attack, it is obvious that widespread transmission of the organisms can occur, in particular through transfer to food of minute amounts of stool containing organisms by unclean hands or by house flies.

### **Treatment**

Mild cases require little general treatment. In the more severely ill cases the administration of intravenous fluid is often more important than the administration of antibiotics.

At the earliest sign of illness—commonly unexpected looseness or mild diarrhoea associated with some feverishness (often in children the passage of blood and mucus is the first noticeable sign) a doctor should be called as correct diagnosis is very important. If a doctor is not available the patient should be given 2 tablets (1 gramme) of sulphadimidine every 6 hours for 5 days. This dose is for those 15 or more years of age (for those 1 to 15 years the dose should be half, and for those under 1 year, quarter this amount). Alternatively phthalylsulphathiazole\* 10 to 20 tablets (5 to 10 grammes) in divided doses daily for 5 days may be administered. See that the patient's stools are protected from flies and disinfected as soon as practicable. The antibiotics streptomycin, choramphenicol and the tetracyclines are satisfactory, but should be taken only on medical advice. They are specially indicated where there is reason to suspect that the dysentery bacilli concerned have developed some resistance to the sulphonamides.

Prevention of bacillary dysentery depends on stopping the *Shigella* organisms excreted in the faeces of the patient reaching the mouth of another person. The methods of doing that are discussed in the section on transmission and control. Flies generally play an important part in the dissemination of this disease.

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\*Phthalylsulphathiazole is the official name of the substance, but it is also obtainable under the proprietary names of Thalazole, Sulphathalidine, Thalostatyl, etc., as tablets or as liquid suspensions. Succinylsulphathiazole may be purchased under the proprietary names of Sulfasuxidine, Colistatin, etc., as tablets or suspensions.

## AMŒBIC DYSENTERY or AMŒBIASIS

Amœbic dysentery is caused by the swallowing of food or water which has become contaminated with the living cysts of *Entamoeba histolytica*. Young adult forms of this amœba are liberated from the cysts into the contents of the bowel ; they are in fact, minute parasitic animals which are considerably larger than bacteria ; some form colonies within the tissues of the body ; to achieve this, each can penetrate the lining membrane of the large intestine, propagate there by repeated sub-division and live on the surrounding tissue of the gut ; the colony so created increases in numbers and burrows even deeper. A multiplicity of such colonies means multiple minute ulcerations of the bowel lining giving rise to symptoms not unlike those of bacillary dysentery.

Not content with invasion of the intestine itself, amœbæ may find their way into the veins of the intestine and be conveyed by the blood stream to the liver, where further colonisation occurs causing inflammation of the liver and subsequently an amœbic abscess.

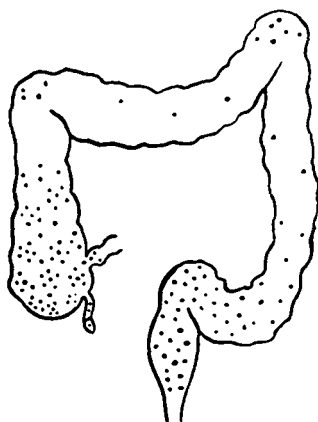


Fig. 2

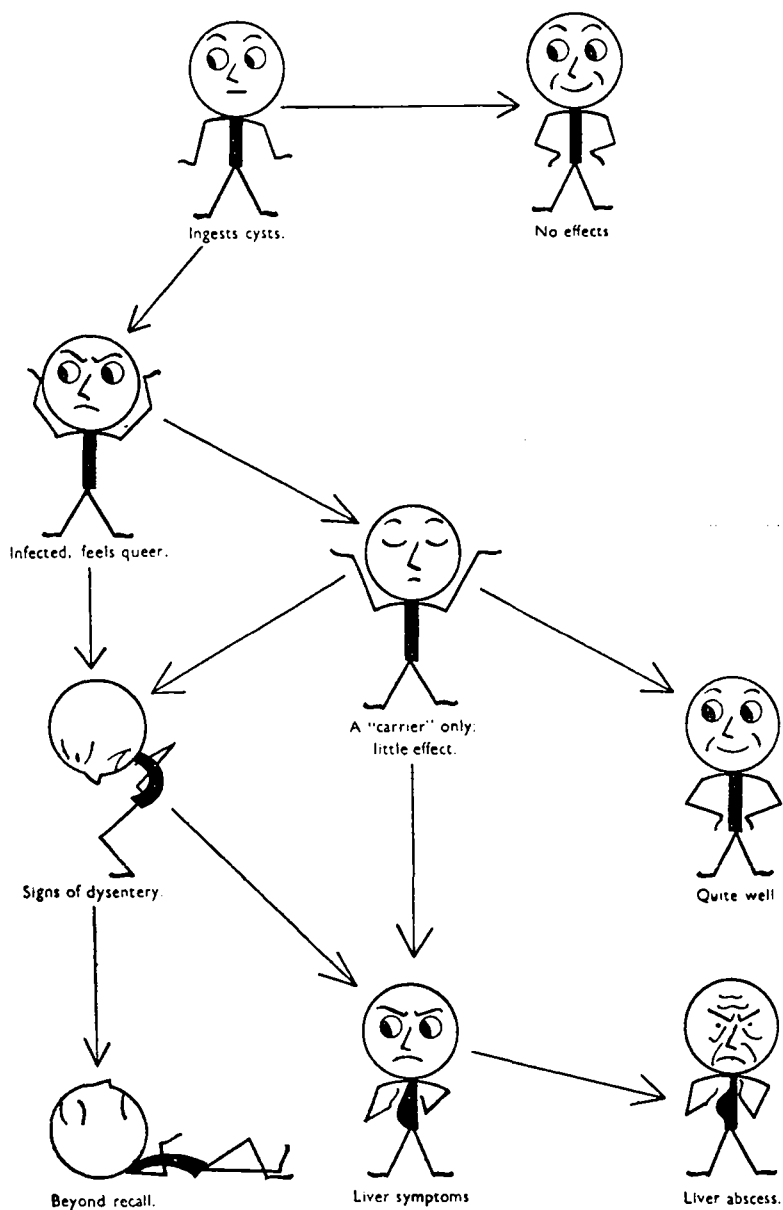
### THE LARGE INTESTINE

The density of the dots is an indication of the parts of the large intestine most commonly affected by amœbic dysentery, *i.e.*, the beginning and the end.

In both bacillary and amœbic dysentery the patient suffers from diarrhoea with the passage of some blood and mucus in the stools, but the former disease is generally acute in its onset, is associated with fever, painful straining to empty the bowel, and the passage of numerous, small, odourless stools sometimes consisting of little else than bright red blood and mucus (resembling red currant jelly). Amœbic dysentery, on the other hand may be insidious in its onset and is not associated with fever. The patient passes few stools daily but they are large, offensive and smelling of decomposing blood.

**Treatment:** Whereas an attack of bacillary dysentery may be cured by the administration of a sulpha drug or an antibiotic an infection with *Entamoeba histolytica* demands skilled medical attention. It is, therefore, important that the correct diagnosis be made as early as possible, so

Fig. 3  
AMOEBIASIS — ITS POTENTIALITIES



anyone suffering from an attack of diarrhoea lasting more than 24 hours should consult a doctor. Modern treatment, properly applied, has proved very successful, so relapses are uncommon and the incidence of liver complications has been very greatly reduced. A large number of people in developing countries, however, never get any treatment at all and among them the disease takes a heavy toll.

Emetine is still the most effective drug in the treatment of acute amœbic dysentery, but toxicity limits its use. An acceptable regimen would be 60 mgs. daily by intramuscular injection for 4 days, followed by diloxanide furoate (Furamide or Entamide) 0.5G thrice daily for 10 days, or metronidazole (Flagyl) 2 tablets (400 mgs.) thrice daily for 10 days. For amœbic liver abscess either a continuation of Emetine and chloroquine or metronidazole (Flagyl) alone is currently advocated.

There is no doubt that the degree of severity of illness after infection varies very considerably in different persons. Outwardly healthy individuals may be cyst passers and if some other illness occurs such as malaria, actual amœbic dysentery may flare up. A single massive dose of cysts in food would almost certainly give rise to acute illness but more often it is repeated doses of small numbers of cysts from, for example, an infected water supply which ultimately break down the natural resistance of a healthy person to produce dysenteric symptoms, mild, acute or severe. An amœbic infection treated and eliminated does not confer any degree of immunity against re-infection later.

At present no prophylactic inoculation is of any avail for protection against infection with *Entamaba histolytica*, nor is it customary to take a drug for preventive purposes. Nevertheless under limited periods of exposure to infection the taking daily of Furamide, di-iodoquin or Embequin, etc., by an individual may protect against amœbiasis; a dosage for the adult would be (di-iodoquin) one tablet (0.3 grammes) after each of three meals per day.

The possible results of the ingestion of amœbic cysts are illustrated in Fig. 3: they are influenced by the size and frequency of dosage of cysts, by the varying susceptibilities of different persons and by the adequacy of treatment.

## RISK OF INFECTION

Bacillary dysentery is present in most countries of the world but the risk of infection is perhaps greatest where and at the time of year at which houseflies are plentiful. Human infection with *Entamaba histolytica* is also widely disseminated in both tropical and temperate climates. In bacillary dysentery, infection originates from the person who is ill or recovering from an attack but in amœbiasis it comes from an individual who has become a more or less permanent cyst passer after contracting the disease; the number of these in any one community will bear a relationship to local standards of personal and food hygiene; to the purity of water supplies; and the prevalence of flies.

The highly susceptible newcomer to communities in warm countries is faced with the problem of taking every possible precaution to ensure that fluids and solid food taken by mouth are uncontaminated by minute quantities of human excrement—in practice not so easy to achieve in the home and the hotel without constant watchfulness. The size of the dose of the infecting germs and the frequency are important in that partially effective precautionary measures may lessen the dose to a degree that the infection fails to develop and illness does not result.

If the newcomer develops an illness suggestive of dysentery he should obtain medical attention ; the doctor may be able to make a diagnosis on clinical examination as to which form of dysentery his patient has developed ; he can only be certain by a laboratory examination of the stools, in itself a skilled procedure. In the absence of a doctor the patient can treat himself on the assumption that it is bacillary dysentery according to the regime on page 12. If the symptoms fail to respond to treatment within a few days it is quite likely that he may have an amœbic infection. Double infections can occur also. Under these circumstances the services of a doctor are imperative if chronic illness is to be avoided.



## TRANSMISSION AND CONTROL OF INFLAMMATORY INTESTINAL DISEASES

All the various organisms which can cause these diseases enter the body by the mouth, reside, propagate and colonise principally in the large intestine and leave the body via its waste products or excrement. Man is the only, or the chief victim of these maladies and it is his own insanitary habits that are responsible for their continuation and spread.

The methods of transmission of any intestinal organisms from person to person are readily seen in Fig. 4.

### Water

In many countries it is common practice for children in particular and adults to empty the bowel on the ground; rain can wash the excrement into water courses which may reach the source of supply of water to the community; although large numbers of bacilli and amœbic cysts die, some may live a long time in water of stream, river or pond. Deep well water is usually uncontaminated if the entrance is protected from seepage and if there are no pit latrines in close proximity.

In the absence of purification processes well water is the safest for communal use. (*See Ross Institute Bulletin No. 10.*)

The simplest domestic method of purification is boiling. Domestic filters are reliable only if they are carefully looked after by intelligent people. In industry, mines and plantations, water which has passed through a purification process should be laid on by pipe line to individual or groups of houses. Filtration followed by chlorination eliminates bacilli and amœbic cysts but chlorination alone without filtration may not kill all cysts.

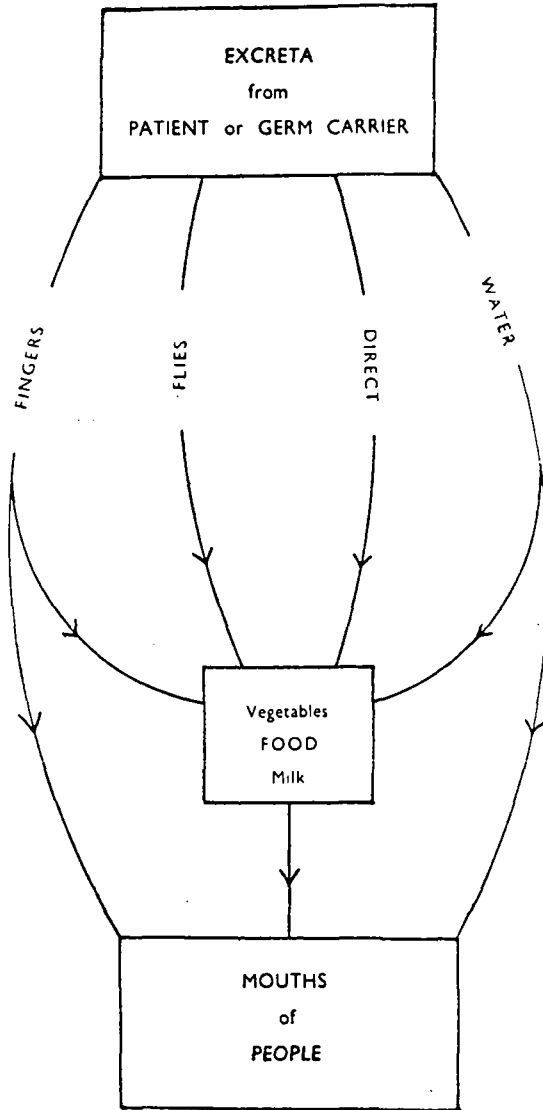
### Food

The contamination of food can occur in several ways:

- (i) Human excrement is commonly used in some countries to fertilise the soil of garden crops, particularly those for marketing in bazaars, etc. The danger lies in the use of fresh excreta; it could be obviated by some method of ripening, e.g., composting of refuse with excreta or by the addition of small quantities of ammonium sulphate which has a sterilising action and is itself a useful fertiliser. The problem is a difficult one but not insoluble on estates or market gardens which could be controlled by enlightened owners or by the local health authority.
- (ii) Otherwise clean vegetables of the salad variety or fresh fruit which are normally eaten in the raw state may be contaminated by washing in polluted water during preparation for the table; a safe water should obviously be used for this purpose.

Fig. 4

HOW THE GERMS OF THE BOWEL DISEASES  
PASS FROM ONE PERSON TO ANOTHER



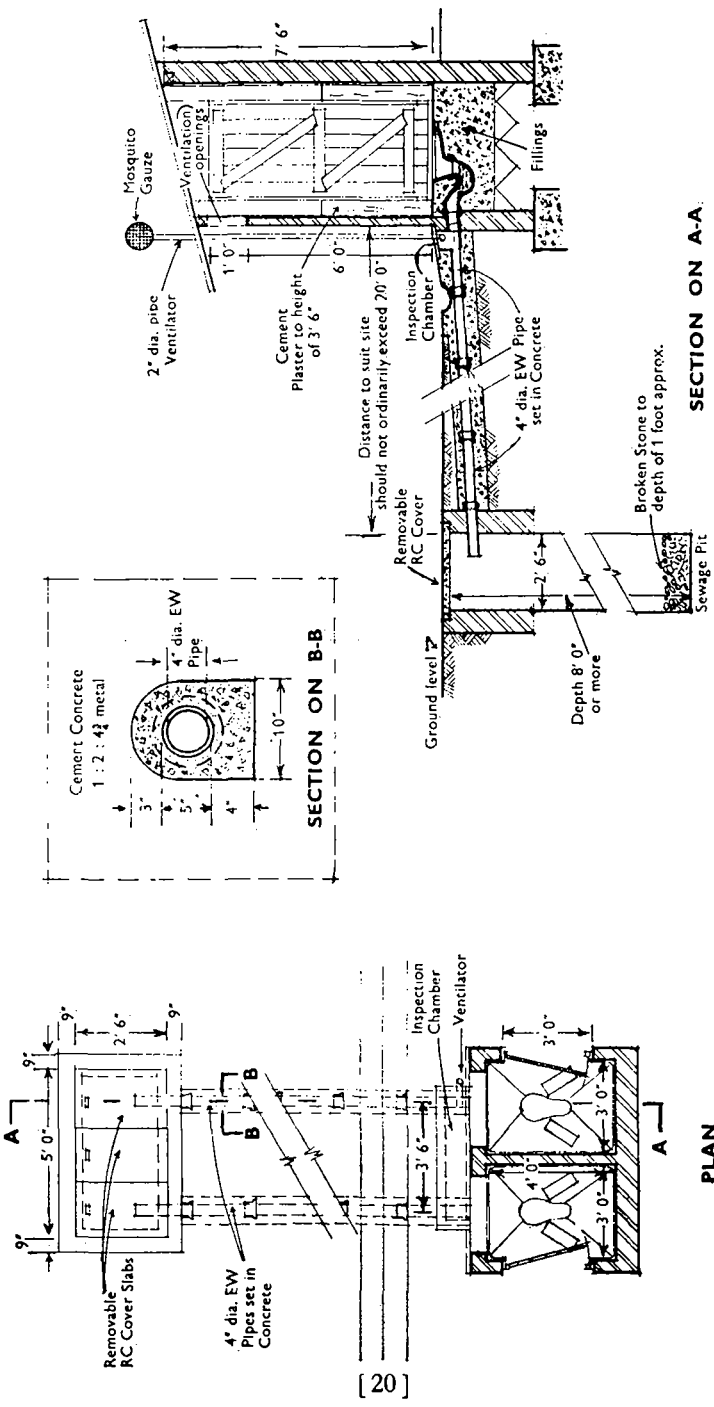
The individual may take the following precautions when he considers his food to be suspect whether it be in a public or private eating place :—

- (a) Select only dishes which have been adequately cooked just before consumption.
- (b) Do not take any uncooked vegetable particularly of the green salad type, or fresh fruit without intact skins unless you know they come from a safe source. Intact fruit should be peeled at the time of consumption.
- (c) Sterilise green salad leaves by immersion for 15 minutes in undiluted vinegar, which should be the equivalent of a 5 per cent. solution of acetic acid. (The lettuce should be eaten soon after, as this treatment impairs its quality.) Other chemicals, e.g. solutions of chlorine or permanganate of potash are of very doubtful value.

(iii) *Food handlers.* A person who has had dysentery recently or amœbiasis at any time is a potential menace if he or she is involved in the preparation or handling of food; included are the housewife, children's nurse, the servant, cook or waiter and the employee in the catering business or public eating place, hotel or restaurant. Skilled laboratory investigation is necessary to determine whether a person is a carrier of pathogenic intestinal organisms ; from a practical point of view, this may be possible in individual instances but is very difficult and almost impossible to apply effectively in public eating places. It is conceivable that in some countries an enlightened public through its government might insist that catering establishments should be open to inspection by health officers to ensure that the ablution facilities for the employees and the methods of preparation of food will diminish to a minimum the risk of contamination of the food.

To be precise, the infective food handler is the person who at the time of evacuation of the bowel, gets the fingers contaminated with small and frequently invisible amounts of excrement. Dangerous bacilli and amœbic cysts will not survive on *dry* fingers for more than ten minutes or so, unless they happen to get into the moist crevices under or about the nails. It is obvious therefore that scrubbing of the hands, preferably with soap in warm water, after the act of evacuation and certainly before handling food, is an elementary and simple practical preventive measure easy of application under all circumstances and depending for its success on the provision of washing facilities at or adjacent to kitchens or food preparation rooms and the insistence on creating the hand-washing habit in all concerned. Careful drying of the hands on personal towels and adequate care of the nails are an essential part of the cleansing process. In some places and under local circumstances of high prevalence of dysenteries it may be advisable to go a step further and sterilise the hands after washing by immersion in an antiseptic solution such as Dettol or an 0.5% alcoholic or aqueous solution of Chlorhexidine (Hibitane); or the application to the hands of a cream containing 1% Chlorhexidine.

**Fig. 5**  
**FAMILY PRIVY**



Two semi-detached family pour-flush latrines discharging into a single pit.

In instances where food-handlers are known to have had dysentery periodic short courses of drug treatment with phthalylsulphathiazole or di-iodoquin can be given ; results should be checked if possible by laboratory examination.

(iv) *Houseflies* have been incriminated as transmission agents of every form of communicable intestinal disease, food poisoning, typhoid fever and bacillary and amœbic dysentery. Indeed so important are they in many countries that the incidence of bowel disease waxes and wanes with the increase and decrease in the fly population. The mechanism of transmission is simply that flies normally and equally feed on human or animal excreta and human food ; they may, therefore, carry organisms of disease on their bodies or ingest them and later deposit them in vomit drops on human food in the food storage room, the kitchen or on the dining table.

Flies must therefore be dealt with by every available means (*vide* Ross Institute Bulletin No. 5), summarised as follows:—

- (a) Control of breeding places by incineration, burying, or composting of animal, human and vegetable filth.
- (b) Prevention of access of flies to latrines, dwelling houses, and in particular, food storage rooms and kitchens.
- (c) Protection of food in the house by suitable covers; this applies particularly to fruit, sugar and milk.
- (d) The regular use of insecticides of both the residual (D.D.T. and benzene hexachloride) and space spray knock down types (pyrethrum) in the household or catering establishment. The routine outdoor use of residual insecticides on breeding grounds, e.g., rubbish dumps, only succeeds in producing sooner or later a race of flies resistant to insecticide.

**Direct Transmission** can readily occur in institutions or households where the level of personal hygiene is low, particularly between children. Clothing, toys and similar objects can be readily contaminated with excrement which reaches the mouth by way of the object itself or the fingers. In tropical places where children evacuate the bowel indiscriminately, amœbic cysts may remain alive on the surface of permanently damp soil to constitute a risk to other children playing on it.

### **Sanitation**

It may be a 'counsel of perfection' to insist on the importance of the proper disposal of human excreta (*vide* Ross Institute Bulletin No. 8) by whatever means may suit the local circumstances and so help to ensure that water supplies are not contaminated and that flies do not readily pick up pathogenic organisms. The higher the degree of insanitation the higher the incidence of intestinal diseases! The solution of the problem of the sanitary disposal of human waste matter is fundamental in the control of these diseases.

Basically it is a household or family problem: an easily accessible latrine should be the only place of excreta disposal by men, women and children. Such a system universally applied in rural areas and villages of a country can only be achieved when the people collectively realise the reason for the radical change in age-old traditional habits, namely the control of intestinal disease and worm infestations. A change of this nature could occur over a period of years and would depend largely on the enlightenment of women and the training of children; the demand for this household convenience must come from the people themselves.

In urban areas and industrial villages the sanitarian can achieve results more quickly through the application of bye-laws insisting on the provision and maintenance of latrines for individual premises. Experience has shown that communal latrines are actively disliked by most peoples and their construction should be restricted to such public places as factories, schools, meeting places, etc.

Latrine systems are described and diagrammatically illustrated in the Ross Institute Bulletin No. 8, 'Rural Sanitation in the Tropics'.

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