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Indian Medical Journal

Sep 1939

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The Indian

Medical Journal



FOUNDED 1906

Organ of the All-India Medical Licentiates' Association

(With which THE AGRA MEDICAL CLUB JOURNAL is incorporated)

DYSENTERIES OF INDIA

Vol. 33. No. 9

CONTENTS

September, 1939

274

## EDITORIAL

Page

Relative Incidence of Dysenteries—Dysenteries and the Seasons—The Dysenteric Carriers—Diagnosis of Dysenteries—Prognosis in Dysenteries—Advances in the Treatment of Dysenteries—This Special Number ... 286

## SPECIAL ARTICLES

Story of E. Histolytica S. BALASUNDARAM ... 290  
 Organisms of Bacillary Dysentery A. VISWANATHAN ... 293  
 Pathology of Dysenteries K. RAMA RAO ... 294  
 Clinical Picture of Amoebic Dysentery S. G. DUTTA ... 296  
 Clinical Picture of Bacillary Dysentery D. D. SARMA BARUAH ... 296  
 Treatment of Acute and Chronic Amoebic Dysenteries K. NARAYANA MURTHI ... 298  
 Choice of Methods and Drugs in the Treatment of Dysenteries A. VISWANATHAN ... 301  
 Hepatic Amoebiasis K. NARAYANA MURTHY ... 307  
 Bacteriophage Therapy in Bacillary Dysentery S. P. DE ... 310  
 Non-Specific Dysenteries N. R. N. REDDI ... 312

## MEDICAL NEWS

... 318

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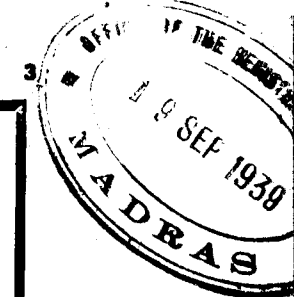
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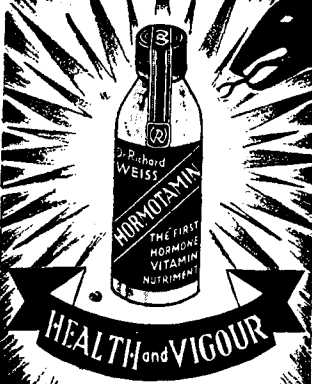
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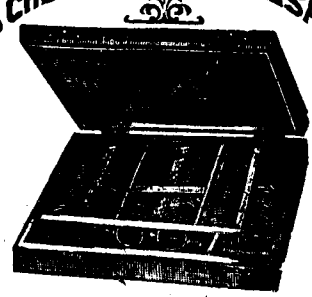
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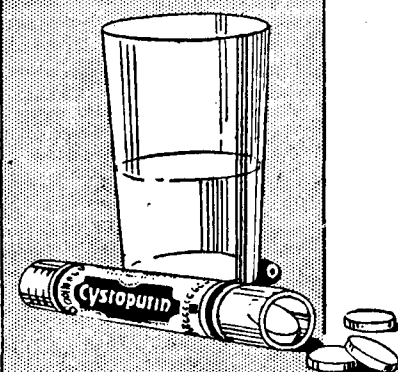
Ztschr. f. Gynakol,  
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(Ztschr. f. Urologie, Vol. 31, No. 9)



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### Important Announcement

To commemorate the memory of the late Sir Bahadur Dr. Saraju Pershad Tiwari, Honorary General of Civil Hospitals, Indore, one of the Founder Members of the All India Medical Licentiate Association and one who has given his best in mind and money for its growth and development, the Standing Committee of the Association has resolved to waive the admission fee of Rs. 2 for membership in the case of those medical licentiates who enlist themselves as members during this month of September as a mark of anniversary remembrance and as a means to foregather the medical licentiates of India in a common bond of fellowship and endeavour for whom he had toiled so much during his life time.

The General Secretary fervently appeals to all the medical licentiates of India to respond in a fitting manner.

Every member is requested to show this appeal to the non-members in his locality.

Application for and remittance of membership fees of Rs. 4 may be made to Dr. Khan Chaud Wohra, Hon. Treasurer, All India Medical Licentiate Association, Civil Hospital, Karnal; or Dr. Manohar Lal Kapur, General Secretary, King Edward Memorial Hospital, Karnal; or Managing Editor "Indian Medical Journal", 131, Coral Merchant Street, Madras.

\*The text of the resolution will be found under "General Association Notes."

Vol. 33 | MADRAS: Sep. 1939 | No. 9

### Relative Incidence of Dysenteries in India

Dysenteries stand third in the list of diseases which claim a heavy toll of mortality in our country—the first two being malaria and cholera.

Considering the slow-paced evolution of Public Health, and the unfortunate want of wherewithal to quicken it, no wonder that a disease which depends for its spread on water and food infection, rages still mercilessly in our midst. The annual reports of the Public Health Commissioner since 1930 record the lamentable story of its rising trend.

It is however heartening to read in the same reports that rural areas are less affected than the urban, and this is most probably due to sun-baked soil and water where the infective organisms find a premature grave. From this, we can safely draw the conclusion that dysenteries are mainly a problem of the cities, closed communities and congested areas. If these regions are tackled first, by the provision of suitable latrines, arrangements for proper

conservancy, supply of pure drinking water, and control of food-stuffs much might be achieved in the reduction of dysenteric incidence. The Government have been ever willing and anxious to assume public health control, but their handicap has been the lack of co-operation by the public. That is the greatest tragedy in our country. The Public Health Act of Madras, a beneficent result of the farseeing statesmanship of the Hon'ble Dr. Rajan, has come not a day sooner than necessary. This Act statutorily compels the local authorities to spend not less than 30% of their income on public health amenities, such as provision of latrines and conservancy, pure water supply and general sanitation. Similar Act is needed in other provinces also, and we hope that at no distant date it will be an accomplished fact.

Owing to the difficulty of making a differential diagnosis between the chief types of specific dysenteries on one side, and between dysenteries and diarrhoeas on the other, those two groups are ordinarily classified together in all the provincial and central public health reports in our country. Therefore, the published statistics are no guides to the question of determining their relative incidence. For this we have to fall back on the reports of Indian army, central jail and general hospitals. It is to the credit of the Calcutta School of Tropical Medicine that its workers have, by frequent epidemiological survey and stool analysis, proved that bacillary dysenteries form more than 80% of cases, and only the rest are due to amoebic and other rarer infections. Of the bacillary dysentery, it has also been proved beyond doubt that infection with B. Flexner is commoner than Shiga's, that Shiga's is more virulent than Flexner's (although not absolutely), and that bacillary dysenteries are essentially epidemic in nature while the amoebic dysentery is mostly endemic in its spread and incidence.

This outlook is necessary to aid the diagnosis, choice of methods and drugs in the treatment and the appreciation of the general conception of dysenteries as a whole.

### Dysenteries and the Seasons

It would be incorrect to assume that the climate and the altitude influence the incidence of dysenteries to any appreciable degree or extent. But as Sir Leonard Rogers used frequently to point out, there is a close association between the rainfall and the incidence of dysentery, particularly the bacillary type. Except in the North-West Frontier, the monsoon begins in June and ends in

October, and this is the season of greatest incidence. The reasons are obvious: the rains connect the cesspools with the sources of drinking water and the infecting organisms are thus spread far and near; they also exacerbate the prevailing insanitation of the houses, the streets and the fields; the flies enter the area like the marauding tribes of old and act as mechanical carriers and distributors of infection, and accomplish this work literally and figuratively with winged speed. Rainy season thus offers a suitable environment for the epidemic spread of dysenteries. Although amœbic dysentery has not been known so far to assume epidemic proportions, it is yet true that its incidence is greater in the rainy season than otherwise. On the whole, the observation of Acton and Knowles that there is more or less constant level of new infections with entamœba histolytica all the year round may be accepted as generally true, but we do not believe with them that it is more prevalent during the hot weather because of the possibility, they say, of the infective cysts being blown about and disseminated by dust. There is no doubt that hot weather is favourable for such a dissemination of cystic amœba through currents of dusty air, but it lacks effective modes of communication to man by means of infected water or the fly, none of which figure prominently in this season. Rainy season can therefore be considered as the season of greatest incidence of all dysenteries, and like all general statements this one has wide variations and exceptions, especially in a vast country of ours subject to the impact of two chief monsoons at two different periods of the year, and to the varying state of natural subsoil drainage in the different areas of our country.

#### The Dysenteric Carriers

The carrier problem of dysenteries is a difficult one to deal with, particularly among the general population. The utmost we can do, and which is being done, is to weed out the carriers in such institutions as jails and hospitals, students' lodges and public restaurants and submit them to proper treatment. The importance of tackling the carriers lies in the fact that they are the primary sources of epidemic and endemic infection. If the carriers are numerous and the environment for the spread is favourable, epidemic outburst is certain. If the carriers are few, the chances are more in favour of sporadic occurrence and for a state of endemicity. In any case, the dysenteric carrier is a danger to the society in which he lives, but to find him out as such and to deal with him is a problem

for which we fail to find any effective solution. But the medical men can contribute a part to this solution by making it a definite point not to discharge their dysenteric patients till the stool is rendered negative.

The spread of infection is no doubt through the infected water and the infected food, but we are inclined to lay greater emphasis on the house-fly as a convenient means of disseminating the infection. It has been our experience that without the fly as an intermediary the infection rarely becomes widespread. If only our readers can picture the presence of a case of dysentery in an ordinary Indian home, the way in which the evacuations are removed and thrown out in the street, the number of flies hovering in the very citadel of the disease, the incessant besiege of men and foodstuff by promiscuous battalions of flies, the intercommunication of people and flies between home and home, and homes and market place where putrid fish, flesh and vegetables form a favourable nidus of infection, they will not but feel inclined to realise the importance of insanitation and its product, the fly, in the right understanding of the problem of dysentery transmission, as indeed it is so in infections of like nature. Thus the role which the flies play in the rapid spread and dissemination of dysentery in our country is significant and important.

#### Diagnosis of Dysenteries

It is not our intention to write on this aspect of the subject in any detail. We are aware that conditions of medical practice in our country are such that it is not possible to obtain laboratory diagnosis in a large majority of dysentery cases, and hence we are impelled to stress on the importance of naked-eye examination of stools, which in our experience, as certainly in others, has revealed sufficient information as to arrive at an approximately correct diagnosis. The litmus test is another handy and useful aid—it is acid in amœbic and alkaline in bacillary dysentery. These are really parts of the method of clinical diagnosis, and most certainly important parts which in cases of dysentery should be made a regular routine. Laboratory help is indeed invaluable not only in diagnosis but also in studying the progress of the case under treatment. It should not be avoided when it is available, but the actual position is it is rarely available. In most of the Western cities, the municipalities or the Government maintain Public Health Diagnostic Laboratories for the benefit of the practitioners at public expense, and it is right that it should be so because correct

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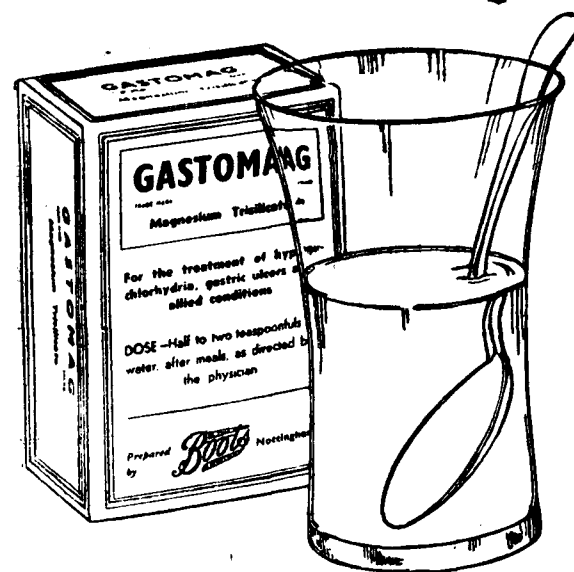
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diagnosis of epidemic or infectious diseases is an indispensable part of a public health campaign. Such facilities are not available even in the big and important cities in our country, and we think that, if the public health programme is not to be merely a paper programme and if the authorities are anxious to make a right and effective beginning, the time has arrived for starting Public Health Diagnostic Laboratories in at least the important and overcrowded cities, either by the municipalities concerned or the local governments. Otherwise the vicious circle of infection cannot be broken, and it is not strange that the last ten years' reports of public health in our country reveal an upward trend, not only of dysentery but of other infectious diseases also. The practical value of public diagnostic laboratories lies in the almost certain detection of early infective cases and the corresponding help it gives in nipping the infection at its source.

### Prognosis in Dysenteries

Both in the acute amoebic and bacillary dysenteries, prognosis is very good provided proper methods and remedies are applied early, and provided also the patients submit to a full course. In actual practice, however, the prognosis is uncertain, because the majority of patients give up treatment after a clinical cure, easily obtained by a few preliminary doses of emetine or serum as the case may be, and the whole thing depends on the persuasive power of the doctor to retain his patient and the purse of the latter. The result is that such patients who ought to have recovered completely slowly lapse into various states of invalidism corresponding to the degree of chronicity which the disease is bound to establish under such circumstances. And a chronic dysentery, amoebic or bacillary, cannot always command a favourable prognosis in spite of the best available treatment. The reasons are obvious. In acute dysenteries our primary aim is quick eradication of the offending organisms before they destroy the intestinal mucosa beyond repair, and fortunately there are effective remedies which can give very good results in this respect. But in chronic dysenteries our primary aim is to restore, if possible, the physical and physiological integrity of the intestinal mucosa, and this is not possible if the tissue destruction is far advanced even though we may be able to rid the intestines of amoebic cysts or any other bacterial pathogens. As d'Herelle has said in connection with another subject: "The value of a specific remedy is absolute

when it is applied at the appearance of symptoms; if its application is delayed or incomplete (italics ours) its efficacy diminishes and becomes nil when fatal organic lesions are established." This is the criterion for prognosis in dysenteries, as indeed it is for any other disease for which a specific remedy is available.

### Advances in the treatment of Dysenteries

During the last two decades specific remedies for the effective treatment of dysenteries have been evolved and put into use, and during the same period many fallacies in the conception and methods of treatment of dysenteries have been discovered and discarded. This progress has resulted in the crystallization of knowledge in most of its important aspects. We have now learnt that emetine is effective in acute intestinal and hepatic amoebiasis and that outside these disorders it is practically of no use. We have now almost given up the surgical treatment of hepatic abscess due to amoeba, unless the abscess is too huge or complicated by mixed infection to render evacuation and drainage necessary. The problem of effective eradication of amoebic cysts still confronts us, but preparations of emetine, of oxyquinoline and arsenical derivatives are now available for use with a fair amount of success. Antidysenteric serum is a definite advance in the successful treatment of acute bacillary dysenteries. Auto-genous vaccines for chronic bacillary dysentery have been found fairly useful and sufficient. The perspective in the treatment of chronic dysenteries has changed from the feverish rush to amoebicides and bactericides to one of securing the healing of chronic intestinal ulcers and of restoring the functional integrity of the intestines by means of rectal infiltration of mild antiseptics, coupled with body rest and balanced diet, reinforced if necessary by a mixed vitamin and iron therapy.

It must however be mentioned that while the medical advances in dysenteries have been well understood and followed, public health advances, though familiar, are yet to take a practical shape and yield tangible results. The responsibility for the latter rests with the Provincial Governments and unless this is shouldered early, the rising trend in the incidence of dysenteries cannot be resisted.

### This Special Number

We feel that no apology is needed for selecting such a trite subject as dysenteries for discussion in this Special Number, because, as indicated earlier in the course of this leading

(Continued at foot of next page)

## Story of Entamoeba Histolytica

• Dr. S. Balasundaram, Director,  
The Ehrlich Laboratory, Madras

ANTONI VAN LEEUWENHOEK, the lens polisher of the 17th century in Europe, never had an idea of the possibilities and implications of the discovery he had made. Grinding the lenses was his duty, and fitting the lenses together and looking through them was his great hobby. This indefatigable and remarkable man laid the foundation not only of bacteriology but also of protozoology. The microscope used by him was a simple biconvex lens of short focal length clamped between two metal plates. History tells us that this man grinding the lenses himself made more than 400 microscopes including one with a highest magnification of 270 times. Some of the first observations on protozoa were made by him.

The first person who described and observed motile amoebae was LOSCH who in 1875 found these organisms in the stool of a patient suffering from a relapsing type of dysentery in St. Petersburg, Russia. From his description of the organisms one could gather that he was dealing with ENTAMOEBA HISTOLYTICA though he called them AMOEBA COLI. An autopsy was also done on that patient which revealed marked ulceration of the large intestine and

(Continued from previous pages)

article, it stands third in the list of diseases with heavy mortality, and also because it is a disease commonly met with and treated by general practitioners for whom this *Journal* is intended. In this task, we have received a hearty collaboration from specialists and practitioners who have readily responded to our invitation for contributions. We only hope that the way in which the subject is tackled, though all the details could not exhaustively be presented within the compass of a single issue, would yet be found useful to and appreciated by our readers. Some of our previous *special numbers* on LEPROSY, ANTENATAL DISEASES, NUTRITION, HISTORY OF MEDICINE and other distinctive subjects of topical interest have been appreciated by the press and were even in full demand from medical men and institutions even outside our country. We hope that this *special number* will also meet the good fate of its fore-runners.

We may inform our readers that the next issue of the *Journal* will deal with Cholera.

numerous motile amoebae were found in the material from ulcers. Some experimental work was also done by LOSCH. He tried to produce dysentery in dogs by feeding, and rectal injections of stools containing motile amoebae. One or two of his dogs developed the typical dysentery. The AMOEBA COLI, as he named these motile organisms, were not held by him to be responsible for the dysentery but were suggested as the organisms responsible for the prevention of healing of ulcerations of the bowel.

The name AMOEBA COLI continued till 1903, until SCHAUDINN published his observations. Thereafter in every part of the globe endeavour was made to spot out these motile organisms in the stools of patients suffering from dysentery. In many of the countries they were successfully recognised.

KOCH observed in 1883 five cases in Egypt with liver complications. KARTULIS described 150 cases of dysentery in the same country. About 60 cases were recorded in Prague. OSLER was the first man to identify them in United States of America in 1890. HUBER was the first man to give the first accurate description of the cysts of the parasites. During the period 1879 to 1901 the idea that ENTAMOEBA HISTOLYTICA was probably the cause of one form of dysentery was fairly well established.

SCHAUDINN was the man to differentiate the two varieties of amoeba: one the pathogenic and the other non-pathogenic. He was the man who gave the name ENTAMOEBA HISTOLYTICA to differentiate it from the non-pathogenic species—the Entamoeba Coli.

The beginning of the 20th century marked the successful cultivation of the amoeba in vitro. Thus at present our knowledge of the organism is fairly complete.

The fact that wherever infection of Entamoeba histolytica has been looked for it has been found, shows that the geographical distribution of the parasite is fairly world-wide, though infections are more numerous in the tropics and sub-tropics. Russia, Poland, Spain, Malta, Sardinia, Turkey, Greece, Italy are all places of sporadic incidence of amoebic dysentery. India, Mesopotamia, Palestine, Syria, China, East Indies, Malay States and Japan, are the places affected in Asia. The Philippine Islands were once full of this disease. It was noticed to be deadly in the American troop when they were operating in these islands during the Philippine insurrection. Tropical Africa, and Egypt were the hotbeds of this disease, and many

of the experiments and discoveries were made in Egypt. America also is not quite free from this disease. The Argentine Republic, Brazil, Chile, Peru, and Mexico have all reported cases of amoebic dysentery. The U. S. A., the Islands of the West Indies, especially Cuba, San Domingo and Haiti have reported sporadic cases, especially in parts where sanitary conditions are poor. Louisiana, Texas, Florida, California and Alabama are the places most affected in U. S. A. Thus amoebiasis is fairly widespread throughout the world.

ENTAMOEBA HISTOLYTICA shows three stages in its life cycle:

1. The Vegetative stage
2. The Pre-cystic stage
3. The Cystic stage.

### THE VEGETATIVE STAGE

Every stage is recognised by a particular morphology. The vegetative stage is the stage of great motility and the organism is recognised easily in freshly passed faeces especially in the mucous particles of the liquid or semi liquid portion of the faeces. Microscopically the cytoplasm of the motile amoeba is found to be differentiated into the outer ectoplasm and the inner endoplasm. The ectoplasm is easily recognised by the pseudopodia formed by it. The endoplasm forms the major portion of the organism, and is coarsely granular containing within it the nucleus and the ingested bodies, especially R. B. C. The nucleus is generally invisible in amoebae found in freshly passed faeces but becomes visible when the organism undergoes degeneration. In a stained specimen, I mean the one stained with one of the hematoxylin methods of staining immediately after the passage of the faeces, the cystoplasm is found to be greyish or bluish in colour while the nucleus is stained jet black. In hematoxylin preparations, the R. B. C. ingested by the amoeba are not found to be stained but appear as yellowish green bodies within the cystoplasm. This form of the amoeba, namely the motile form, is the "tissue invading" form of the parasite and may be regarded as the most active phase of the development. The remarkable activities of these forms is due to the formation of pseudopodia.

### THE PRE-CYSTIC STAGE

Under certain conditions the Entamoeba histolytica becomes encysted. As the cysts are smaller than the tissue invading amoebae, it can be seen that it is quite essential before encystment that smaller sizes of amoebae must

be produced. This is brought about by the multiplication of amoebae by binary fission. Thus in the precystic stage the daughter amoebae, instead of increasing in size, as they do in tissues, divide again producing smaller and smaller amoebae. Before encystment these small motile amoebae lose their motility, become somewhat reduced in size, rid themselves of ingested material such as R.B.C. and become spherical or oval in shape. These precystic amoebae appear as colourless, hyaline, round or slightly oval bodies under microscope. The nucleus is generally visible and it is extremely difficult in many instances to recognise them as the precystic forms of Entamoebae coli.

### THE CYSTIC STAGE

The cystic stage appears in the living unstained condition as colourless, hyaline body, the shape varying from round to oval. The nuclei within the cysts are very poorly differentiated and appear as refractory round masses. The cysts do not contain R. B. C. or other ingested material. Amoebic cysts form the link in the infective chain. They are transmitted from man to man through food and drink polluted with faeces. Though the faeces may contain organisms in the vegetative and the precystic stages of development, yet they are likely to be killed by normal gastric secretion of the person who ingests them, and hence the cysts alone are able to pass through the stomach to infect the intestinal tract. It naturally also follows that patients suffering from acute diarrhoea or dysentery are not those concerned in the transmission and production of this disease in others as the cysts do not occur in the acute dysenteric stools. But the persons who are apparently healthy and who are convalescent from acute attack of amoebic dysentery and whose stools are apparently normal are the most dangerous as carriers of infection as they are the persons likely to pass cysts in their motions. The recognition of this vital and important fact was brought home by Walker and Sellards (1913). The cysts passed by these carriers are usually ingested by persons taking food and drink contaminated with faecal matter, or even through infected water supply. The droppings of flies and the handling of food by infected individuals play their own parts in the transmission of the disease. The former may form the most important cause of epidemic dysentery. Amoebic cysts pass unchanged through the intestines of the fly and thus be capable of producing infection by contaminating food and drink. Hotels and

restaurants, where both the methods of transmission are easy, are the important centres from which the disease is spread. Water contaminated by cysts passed in the faeces may keep the cysts for a number of days viable and temperatures to the extent of 60°C. and more are required to kill them. The cysts are quickly killed by drying but are most resistant to chemicals. Though such an infection by a contaminated food is possible in several persons, the presence of severe symptoms in some and comparative absence of mild symptoms in other individuals is explained by the theory that there may exist strains of the parasites differing in virulence.

When a human being ingests amoebic cysts it is not necessary that symptoms of dysentery must be produced immediately. We do not know the exact method of escape of the encysted amoebae, nor the exact time necessary for such a thing. Good lot of investigations have been made in this direction to find out the exact incubation period. This period is found to vary from a few weeks to nearly a year. But whatever may be the time taken or the method adopted by the encysted amoebae to liberate themselves from the cysts, that they do liberate themselves and enter the intestinal tract has been observed. In the very early stages, the amoebae tries to invade the intestinal wall, especially the places where the glands are situated in the large intestine. Investigations have shown that *Entamoeba histolytica* secretes a substance (Dr. Craig was able to extract such a substance from the cultures of parasites) which dissolves the intestinal epithelial cells and R. B. C. So it is evident that by an alternate process of cytolysis produced in the intestinal wall by this substance and the mechanical penetration of the amoebae themselves they are able to make their way into the glands of large intestine and crawl to its bottom. Multiplication takes place and as a result of the pressure of the increasing number and probably also due to the toxins produced, the gland cells degenerate and disintegrate. The tubules of the glands are blocked by the debris, and adjacent glands over a small area of surface are affected. In the same way the whole thing results in the formation of a slightly raised, yellowish, nodule. The amoebae, by this time, make their way into the interglandular connective tissue. The yellow nodule is, in fact, a small abscess formed at the mucous membrane, which eventually bursts discharging mucus blood and plenty of amoebae, leaving a typical undermined ulcer over the mucous membrane.

The amoebae which escape, invade other glands and produce similar ulcers and thus spread the disease. The frequent discharge of mucus and blood by the patient is only from such ulcers formed by the bursting of small abscesses described above. The formation of such ulcers may be limited to a small portion of the intestine or extend to a wider area. The lesions of amoebic dysentery are most commonly observed in the rectum and just below the ileocaecal valve. The ulcers formed may become gradually larger on account of the multiplication of the amoebae and their infection in the neighbourhood. They even break through the muscularis mucosae and extend to the submucous tissue. After the amoebae have multiplied and caused ulceration for varying periods a healing process sets in. The amoebae gradually disappear and the site of the ulcer is found to be in the form of a puckered scar with the peritoneal lining opposite to it being thickened. Though such a healing process takes place, the ulcers do not heal at the same time and hence persistence of infection with *Entamoeba histolytica* is quite common for years, unless definite treatment is given. In the process of extension through the tissues, the amoebae, not infrequently, make their way into the blood vessels and are carried as emboli to the liver, brain and other organs giving rise to abscesses. The extension of ulceration in the intestine itself may be very rapid and such cases are acute ones. In other cases, though considerable area of the intestine is involved, extension is not very rapid and hence a carrier condition results. The precystic and cystic stages are usually found in the faeces of these cases. A large proportion of these individuals either present no symptoms of infection or present symptoms of gastro-intestinal irritation or mild diarrhoeal attacks.

Thus a person who gets an acute attack of amoebic dysentery gradually recovers and only then provides a suitable nidus within himself for the production of the precystic and cystic stages of amoebic dysentery and becomes a dangerous carrier of infection to others. The story of *Entamoeba histolytica* begins once again in another person.

The house-fly is able to propagate dysenteric infection in two ways—firstly by its habit of regurgitation preparatory to feeding; and secondly by means of its faeces. The latter is probably the more common method.—Manson.

## Organisms Of Bacillary Dysentery

● Dr. A. Viswanathan, L.M.P., Madras

*Bacillus of Flexner* and of *Shiga* are the two chief and common organisms that give rise to bacillary dysentery, although there are a host of others found in association with the specific bacilli of dysentery, or causing by themselves apparently dysenteric symptoms.

It is probable that the latter are merely secondary invaders of the already ulcerated intestinal surface. As such it is rather difficult to assign them any primary role in the causation of bacillary dysentery, at any rate till more about them is known.

The morphology and the cultural properties of *B. Flexner* and *Shiga*, the technique of isolation, plating and staining are not discussed in this article as it only aims to give a general view of the organisms of bacillary dysentery from the angle of vision of the general practitioner.

The first thing to do is to examine the dysenteric stool with the naked eye, and to find out the apparent nature of the exudates. They would be alkaline in bacillary and acid in amoebic dysentery. This will give the first clue. If the stool is almost composed of bright red blood and mucus with little or no faecal matter it will be due to bacillary dysentery, whereas in the amoebic stool, which usually shows wide variations, faecal matter is more prominent and more or less intimately streaked with blood and mucus.

The next step is to send the dysenteric stool to the laboratory for close and detailed examination. The stool must be fresh and unmixed with urine. Such portion of the stool should be selected which is mostly made up of mucus.

The exudates of bacillary dysentery are very characteristic, and once seen under the microscope they will leave an indelible impression in the mind. They are made up of polymorphs (80%), intestinal epithelial cells, endothelial cells and macrophages. The exudates of amoebic dysentery are characterised by scanty polymorphs and plenty of Charcot-Leyden crystals.

The exudates of the bacillary dysentery, however, vary according to the day of the disease. In the very early stage there are very few visible micro-organisms and preponderance of fresh R. B. C., polymorphs, and other cells. Later on, these degenerate and are

more or less replaced by disintegrating pus cells, free haematoidin crystals and various bacteria.

The specific organisms can only be traced by means of a suitable culture. Their isolation is somewhat difficult and depend for its success on the period of the disease. The longer the duration of the disease the greater the difficulty to isolate them. This is further complicated by the contaminating organisms.

The animals which are very susceptible to inoculation with *B. Dysenteriae* are guinea pigs, rabbits and dogs.

*B. Flexner* is the commonest organism in our country and the cause of epidemic dysentery. It belongs to the mannite-fermenting group and relatively less toxic. Its endotoxin is neither filtrable nor diffusible. It forms no pressor bases, but it ferments many of the sugars and produces irritating acids. The reactions of *B. Flexner* would therefore suggest elimination of carbohydrates from the bowel by a preliminary purge and a protein diet. It is well known that dysentery due to *B. Flexner* shows a marked intolerance to carbohydrates.

*B. Shiga* produces very powerful toxins, and is the cause of fulminant dysentery. But it must not be forgotten, as is occasionally reported in the medical literature, that *B. Flexner* too may cause such severe types. The exotoxin of *B. Shiga* is protein in nature, dialysable and filtrable from its body. But its endotoxin is neither filtrable nor dialysable. It also produces pressor bases which are the real cause of all the serious symptoms in acute bacillary dysentery. These pressor bases, at present not identified with histamine, increase peristalsis, congestion of the portal circulation and haemorrhages into the submucosa of the large intestine. They are formed from the amino-acids of the protein molecules in the gut. Hence it is evident that protein diet should be tabooed in *Shiga's* dysentery.

Specific anti-serum can be prepared and successfully administered to cases of bacillary dysentery due either to *B. Shiga* or *Flexner* or both. Although in actual practice we use a mixed, polyvalent anti-serum, it is said that anti-serum for *Flexner's* infection is not as effective as the anti-serum for *Shiga's*. The writer thinks that no capital need be made out of this.

Specific vaccines can also be prepared and used successfully against both types of infection. But they are absolutely of no value in acute cases and they are to be used only in chronic cases. These cases do always harbour

(Continued at foot of page 295)

# Pathology of Dysenteries

• Dr. K. Rama Rao, L. M. P., Medical Officer, Madras Corporation, Madras

The general intestinal pathology of dysenteric lesions of any kind may be summed up in four words: inflammation, necrosis, ulceration, resolution or complications leading to death.

But the mode of attack and the nature of the lesions and their sequelæ vary according to the type of dysentery and to whether it is acute or chronic.

Serious and extensive lesions are caused chiefly by entamoeba and B. dysenteriae (Flexner and Shiga), but it is quite possible that other less important organisms of dysentery may also cause intestinal lesions. They follow the general rule of infection and inflammation. Mucus and ulcerative colitis are now recognised as forms of allergic manifestation, although various bacteria have been



Types of Bacillary Inflammation and Necrosis of the Intestines.

LEFT FIGURE SHOWS GANGRENOUS PROCESS; MIDDLE SHOWS DIFFUSE NECROSIS; RIGHT SHOWS CORRUGATION OF THE INTESTINES.

now and again isolated from such intestinal lesions or bowel evacuations.

Those who have seen the amoebic or the bacillary lesions in the autopsy room cannot forget the indelible impression which they make in one's mind and memory. The difference in the lesions between the amoebic and the bacillary is so pronounced and definite that very rarely mistakes arise in their non-recognition as such. But there is likely to be mixed infection in some cases, and this should not give rise to any confusion as such instances are not uncommon.

## PATHOLOGY OF BACILLARY DYSENTERY

As a rule, B. Shiga causes the acute lesions whereas B. Flexner causes epidemic, subacute and chronic lesions. The lesions originate in the solitary lymphatic glands of the large intestines followed by inflammation, necrosis

294

and ulceration. These look something like pin-heads, and may be spread over the whole of the large intestines, and in severe cases even the lower third of the ileum. In very acute cases, these pin-point ulcers are almost obscured or replaced by severe general inflammation of the whole mucosa with scattered spots of hæmorrhage. In fatal cases, the whole of mucosa is gangrenous. In chronic cases, there is thickening of the intestinal wall and the mucous membrane resembles "a piece of chamois leather with interlaced fibrotic strands on the surface". Small open cavities of pus are quite frequent.

In fact, it is difficult to give a truly fixed type description of the intestinal lesions in bacillary dysentery, as the state of the lesions depend on the virulence of the organism and the stage and extent of tissue destruction. The accompanying illustrations are some of the types commonly met with in the autopsy room.

## PATHOLOGY OF AMOEBIC DYSENTERY

Unlike B. dysenteriae, the amoeba attacks the large intestine in a totally different

vein and cause a typical, what is commonly called an amoebic tropical abscess.

The degree and extent of intestinal ulceration produced by E. histolytica has been found to vary greatly. Perforations, stricture and localised gangrene are often met with. The intestines may be thinned owing to extensive ulcerations or may be much thickened owing to hypertrophy and fibrosis.

As in the bacillary, so in the amoebic dysentery a type description is impossible, as the nature of pathological changes depend on the stage of the disease and other related factors. A few typical autopsy specimens are illustrated below.

## DIFFERENTIAL PATHOLOGY

From the above description, the readers may have noted the general differences between the lesions of bacillary dysentery and



Types of Amoebic Inflammation and Necrosis of the Intestine

Left to right:—(1) HEALING AMOEBIC ULCERS (2) THICKENING OF INTESTINAL WALL DUE TO AMOEBIC ULCERS (3) THINNING OF THE INTESTINAL WALL DUE TO AMOEBIC ULCERS.

those of the amoebic, but they are assembled here again for the sake of clarity.

B. dysenteriae initiates a catarrhal inflammation of the intestinal mucous membrane and spreads down to the submucosa whereas E. histolytica attacks it from below upwards, that is from the submucous layer which is its natural habitat.

B. Dysenteriae gives rise to "snail-like" ulcerations, acute inflammation and even gangrene of the intestines, diphtheroid membrane, hæmorrhagic spots, and, in chronic cases, a granular and infiltrated condition of the mucosa, whereas E. histolytica gives rise to typical exuberant ulcers, raised and round with undermined edges and black slough in their centre. These ulcers may be in various stages of healing attached by fibrous bands or coalescing to form serpentine ulcers. Some of them may reach the peritoneal coat and may even produce very minute and invisible perforations, temporarily closed by inflammatory adhesions but giving rise to chronic peritonitis of very insidious onset.

Rectum and pelvic colon are more commonly affected in bacillary dysentery whereas the caecum and ascending colon are the common sites for amoebic lesions. The readers should however recognise that exceptions are common, and that any part or whole of the large intestines may be affected with lesser or greater intensity.

B. dysenteriae is noted for its toxæmia whereas E. histolytica is less toxic, if at all, but it may attack other organs, particularly the liver, the lungs and the brain by embolism.

Both B. dysenteriae and E. histolytica can be obtained from the scrapings of ulcers, but the former can only be detected and recognised through cultural methods while the latter can be seen under the microscope by the usual staining methods or even without staining.

As, sometimes, lesions of mucous colitis due to allergy closely resemble those of bacillary

(Continued from page 293)

mixed organisms, and hence a mixed autogenous vaccine will be the treatment of choice.

The serological diagnosis of bacillary dysentery is not often used for obvious reasons. The test is based on the agglutinating powers of the patient's serum against known strains of B. dysenteriae. But the agglutinins in a case of bacillary dysentery appear only after the first week of the disease, reach their maximum in its fourth week after which they decline rapidly. Hence it is that the agglutination test is not of any value in the early case, and probably there is no need for it in the later stages.

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### Clinical Picture of Amoebic Dysentery

● Dr. S. C. Dutta, L.M.P., A.M.O., Itakhooli T. E., Kheto, (Assam)

Amoebic dysentery is a condition of colitis, chiefly ulcerative, caused by *Entamoeba histolytica* and clinically characterised by frequent painful passage of stool with blood and mucus.

According to Rogers the term "amoebic dysentery" is itself misleading, because there may not be any clinical symptoms at all in some cases of intestinal amoebiasis. Looseness of the bowels may even be absent in cases with a few ulcerations limited to the caecum. In others, the clinical picture may vary from mild occasional attacks of diarrhoea to severe acute dysenteric symptoms.

These variations may be due to the following probable causes: (1) Presence of different strains of *E. histolytica*. (2) Variation in the resistance of different individuals. (3) Variation in the tissue-attacking power of the parasites depending on different environments.

The following clinical types are found:—(a) *Latent type*.—In this there may not be any clinical manifestation except occasional attacks of diarrhoea. These cases are often wrongly diagnosed as chronic diarrhoea or tubercular diarrhoea.

(b) *Moderately acute type*.—This is the type commonly met with. Onset is slow and insidious. Often the first symptom is diarrhoea. The stool gradually changes its character, becoming mixed with blood and mucus and is passed very frequently. The general condition of the patient is not bad. Signs of toxæmia are only rarely present. Pain of colicky nature may be complained of. Tenderness over the right iliac region may be present and thickening of the intestinal wall may be detected by palpating the same area. The stool is foetid, mixed with blood and mucus and rarely loses its faecal character.

(c) *Fulminant or gangrenous type*.—There is sudden onset with rise of temperature and moderate toxæmia. The stool contains mucus, fresh blood or black slough. This variety is often confused with bacillary dysentery. Perforation of the bowel may occur suddenly as a complication.

(d) *Chronic type*.—There is a history of alternate attacks of diarrhoea and constipation. The stool contains mucus. Blood is very often absent. Thickening of the wall of

### Clinical Picture of Bacillary Dysentery

● Dr. D. D. Sarma Baruah, L.M.P., A.M.O., Jutlibari T. E., Hoogrijan, (Assam)

Bacillary dysentery is usually epidemic in occurrence, occasionally sporadic, and characterised by griping passage of stools with mucus and blood. Mucus and blood are more copious than in the amoebic dysentery.

A very large number of bacteria have been incriminated as causal agents of this disease of which the commonest organisms found in Assam are the bacilli of Flexner and Shiga and the metadysentery group of bacilli. The infection is spread by flies, contaminated water, food, vegetables and milk, direct contact with patients and by carriers. Sufferers from chronic malaria, kala-azar and hookworm are extremely susceptible to this infection.

Incubation period is from 1 to 7 days. Sudden onset with fever, toxæmia, prostration, passage of blood and mucus with the stools, vomiting, thirst and collapse are the symptoms of acute cases of fulminating type. The stools are usually alkaline to litmus and passed frequently. The stool contains very little faecal matter and in some cases blood and mucus only are passed.

But the symptoms vary a great deal in different patients. (1) A simple diarrhoea may be the only symptom of the disease. (2) Some cases become chronic. (3) In some cases the prominent symptom is toxæmia and the patient may die before dysenteric stools appear. (4) Intestinal haemorrhage may be the only symptom.

Arthritis, myocarditis, neuralgia and paraplegia are occasionally seen as complications. In chronic cases, neurasthenia may be complained of. Conjunctivitis and iridocyclitis

the caecum, flexures of colon and sigmoid may be detected. Occasional attacks of colicky pain and hyperchlorhydria may be present (thus simulating a case of peptic ulcer). In long standing cases signs of focal infection from intestine may be present, e. g., neuritis, neuralgia, arthritis, anaemia and leucoderma. Vagotonic symptoms due to absorption of toxins from the intestine may also be present e. g., urticaria, asthma, eosinophilia.

The usual complications are hepatitis, hepatic abscess, cirrhosis of liver; periappendicular abscess, abscess in lungs, spleen, testes and brain; cystitis and possibly sprue.

rarely occur. Anæmic dropsy and disordered endocrine function are sometimes met with.

It is very important to examine the stools of the patient with the naked eye. The history of the illiterate class of people are often misleading and unreliable. The stool contains little or no faecal matter and is not offensive, especially in acute cases. It is usually alkaline to litmus. The quantity is small. Blood is present and may be bright red. Sigmoidoscopic examination may be helpful, but has the drawback that the operation is very painful and cannot be done without anaesthesia. The laboratory examination of the stools is important for diagnosis which is based on the character of the cellular exudate. Agglutination reactions of the widal type have been found useful in chronic cases.

Prognosis depends upon the duration of the illness. If the patient undergoes treatment early, the prognosis is favourable. But it depends also upon the nature of the infection, the degree of toxæmia, the vitality of the patient and careful nursing and diet.

The stool should be disinfected and burnt. The nurses and attendants should wash their hands thoroughly. Flies should not come in contact with stools. The carrier should pass his motion in a deep pit or in a bed-pan well covered and then the stool burnt. This forms a minimum prophylactic procedure.

In very acute dysentery, absolute rest in bed, use of bed pan and warmth to the abdomen are essential. The patient should have no purgative at all and be given acid and cresol treatment according to the doses and technique advocated by Col. F. J. Palmer\*. This treatment has been found in tea garden hospitals of this circle to be an effective remedy; it is also cheap. Drawbacks are the quantity to be administered is large and its tarry smell is disagreeable to the patient. In practice also it is found that to give the mixture every quarter of an hour is too much for the stomach to retain it. If it is given at longer intervals the patient can retain it and good results follow within 24 to 36 hours. This treatment acts like magic in those cases where the patient is passing "meat-washing" stools with fulminating symptoms. It will be wrong if transfusion is not given, in addition when symptoms of collapse and toxæmia are marked. Morphine or laudanum may be given if the patient is vomiting and is restless. In the interval small sips of water may be allowed or ice given to suck. A careful watch

\* Indian Medical Gazette, March, Vol. LXIII, p. 259.

on patients of the illiterate class is of great importance. They think that during such an illness deprivation of solid food makes the condition worse. When the vomiting ceases and the number of stools become less the patient can be given sago diet, whey, albumen water, barley water or milk.

Bacteriophage treatment may also be of some value in certain cases. Antidysenteric serum 40 to 100 c.c. must be given within 48 hours. It is of no value in late cases.

The classical treatment of the disease is as follows: If there is no prostration, exhaustion and toxæmia, the patient should be given a dose of castor oil followed by two drachms each of soda and mag. sulph. two hourly and then half of this dose four hourly, till he is freely passing faeculant motions. After this, bismuth carb in half drachm to one drachm doses should be given at two hourly intervals. If there is much pain in the bowel morphia may be given hypodermically. When there is no tenesmus, starch and laudanum enemata may be given.

During convalescence the diet is increased very gradually. In chronic dysentery the patient should be kept in bed on a simple diet such as milk (boiled, peptonised, or fermented), whey, eggs, and beef juice. It is advisable to give castor oil twice a week at least on an empty stomach. Drugs by mouth are of little value in this condition but bismuth if given in one drachm doses every three hours may be of value. Salol, beta-naphthol and osmokaolin may be combined. Local treatment with irrigation is helpful in the chronic stage but is almost impossible in tea garden practice. If the rectum is irritable, a morphine or cocaine suppository should be given previously. Yatren, protargol, silver nitrate, boric acid, alum, or tannic acid solutions are used for colonic lavage. Before irrigation the bowel is to be washed with a weak soda solution.

*The number and character of the evacuations in bacillary dysentery may vary enormously in different types. Their number may be uncountable, and the unfortunate victim is glued to the commode ..... The nearer to the rectum the lesions, the more urgent the tenesmus; the nearer to the caecum the more urgent the griping. Vomiting may occur from the outset or be absent altogether—*  
**Manson.**

### Treatment of Acute and Chronic Amoebic Dysenteries

• Dr. K. Narayana Murthi, M.D., Research Worker and Honorary Physician, General Hospital, Madras

The treatment of acute amoebic dysentery requires much skill and supervision. Since the number of motions are few in number and the patient is less toxic as compared with bacillary dysentery there is no harm in giving castor oil emulsion on the first day of the occurrence of the disease. This line of treatment not only gets rid of undigested food which might have been responsible for an acute attack superimposed in a chronic case, but also the free living amoebae which is a frequent occurrence in an acute case are washed out and thereby griping pains and frequency or urgency for passing stools are at a temporary abeyance. The diet should be carefully regulated and nursing is important. On the following day emetine in one grain dose can be given intramuscularly. It is always a bad habit to use emetine indiscriminately as a diagnostic therapeutic measure as it often, particularly in children and old people, causes very toxic symptoms. We have entirely dropped pulvis ipecacuanha in the treatment of either acute or chronic amoebic dysentery. But it has still a place in the after-treatment of liver abscess and amoebiasis of the lung. Emetine which is one of the four alkaloids of ipecacuanha has come to establish a permanent place in the treatment of acute cases. It is given in one grain doses intramuscularly up to a maximum limit of 9 to 12 grs., distributed over a period of 10 days. By the time the patient is given this treatment all the acute symptoms disappear.

The patient feels better, the stools are well formed without mucus and blood, but there may be occasional attacks of colicky pain over the sigmoid region. This pain is an indication that the infection from the bowel is not completely eradicated. The amoebae that were lying hitherto free in the lower bowel get encysted and the presence of these cysts in the stool is an indication that the acute condition is passed over resulting in an early chronic condition. Emetine is very helpful in the treatment of visceral complications like hepatitis and amoebic abscess, as in acute dysentery, but rarely helpful in the chronic stage of the disease.

There are certain toxic symptoms which emetine causes and which every one of us should know: cardiac irregularity, asthenia, emaciation, mental depression, myositis and neuritis affecting a certain group of muscles producing paralysis resembling chronic poliomyelitis. We have also to bear in mind that excessive dosage of emetine gives rise to diarrhoea, which might be mistaken for dysentery itself. Fine desquamation of the skin and an atrophic condition of the nails are at times seen. Death may occur from cardiac failure with paroxysmal tachycardia or auricular fibrillation. On account of these serious toxic manifestations of emetine it should be used with judicious care. In mixed infections one is justified to use emetine. It should not be given by subcutaneous route as it causes stiffness and intolerable irritation at the site of infection, particularly in debilitated persons. Widespread cellulitis, localised abscess and irritative eczema are some of the other manifestations seen after subcutaneous injection of emetine. Children under four years rarely suffer from amoebic dysentery and therefore the use of emetine is very much restricted. With increase of age the incidence of the disease becomes greater and one can give  $\frac{1}{4}$  gr. once a day in children between 6 to 10 years of age. Under no circumstances emetine should be given to patients who are about to go to active work as the physical exertion entails cardiac damage. In cases where the pulse rate and blood pressure are low one can give strychnine and emetine together.

When the patient is free from acute symptoms (probably one takes not more than 5 to 10 days) one can supplement the treatment of emetine with E. B. I. This preparation contains 26% of the alkaloid emetine and is particularly useful in chronic cases and in persistent passers of E. histolytica cysts. This is an insoluble powder dispensed in capsules, in 1 gr. doses. The maximum adult individual dose is 3 grs. per day. This drug is given by mouth for 10-12 consecutive days. It is not necessary to use large doses of E. B. I. as this is found to give equally satisfactory results in smaller doses, particularly when the combined treatment with emetine is carried out. It is important to observe certain precautions while giving this drug. It is given at bed time 4 hours after the last meal of the day is taken, and even a glass of milk is not allowed to be taken thereafter. Patients should remain absolutely at rest and any saliva flowing out of the mouth should not be allowed to be swallowed. Vomiting and

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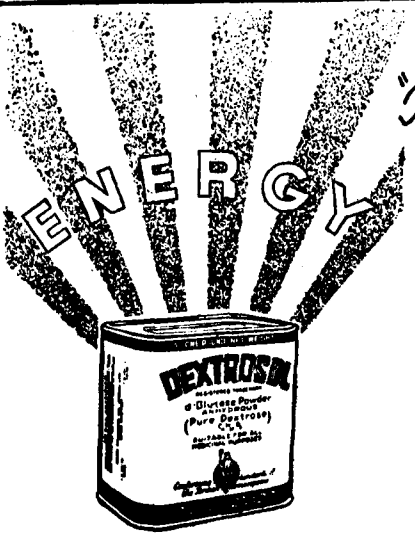
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diarrhoea are sometimes noticed in the earlier part of the course. But these subside in the latter part, and so cannot be regarded as a contra-indication for its administration. It is possible if those symptoms are not present that the drug is not dissolved and absorbed. It is important that the patient should remain in bed and take only liquids like milk, butter-milk with one egg and toast. Excessive nausea and vomiting are controlled by small doses of luminal, or nembutal or Tr. Opi. 15 minims, if given  $\frac{1}{2}$  hour before the administration of the drug. During the course of the treatment one can expect the patient to lose weight at the rate of  $\frac{1}{2}$  lb. daily and usually there is also a definite fall in B.P. and slowing of the pulse. Alcohol in any form should not be given during the treatment. This line of combined treatment rarely gives rise to relapses. But the patient after a complete course of this treatment can take to active work with a careful dieting continued for some months. In my experience it is often found to be unnecessary to give second and third course of the combined treatment outlined above. It is enough if the course of treatment is controlled by microscopical examination of the stool once in three weeks. Though periodic sigmoidoscopic examinations are advised they cause great discomfort to the patient and hence it is not my practice to do such an examination as a routine.

Recently emetine-periodide has been introduced as a therapeutic measure. It is found to be less toxic than E. B. I. and therefore can be used in such patients where the latter is not tolerated.

Auremetine is another drug administered in 1 gr. doses, but this causes much more nausea and vomiting than the E. B. I. over which it has no special advantage.

Yatren Bayer's (iodine-oxyquinoline-sulphonic acid), Chiniofon (U. S. Pharmacopoeia), Quinoxyl (Burroughs-Wellcome) and Entero Vioform are some of the oxyquinoline derivatives used in the treatment of chronic intestinal amoebiasis. There are other substances with similar chemical composition in the market known as Anayodin (U.S.A.) and Dysentulin (Germany). I have no experience with these drugs except with Yatren. Yatren can be given by mouth or as retention enema. Yatren pills (4 grs. each) are given, after the acute symptoms are controlled by emetine. If the motions are slightly controlled, one can give Yatren by mouth, 4 pills a day, and emetine by injection for a period of 10 days. The pills may be repeated, if necessary, after a

week's interval. Some physicians give Yatren by mouth and also as a retention enema on the same day. Really there is no need to carry such a treatment except probably in intractable cases. It is necessary to give a bowel wash with 1 pint of 2 per cent sodium bicarbonate to remove mucus and an hour later 6-8 oz. of a 2.5% solution of Yatren in warm water is run into the rectum and retained for as long as 8 hours, if possible. This course is continued for a period of 10 days. Prolonged course of treatment with yatren gives rise to pain and discomfort.

Entero-Vioform is an oxy-quinoline derivative like Yatren. It does not produce vomiting, and has no effect on the vaso-motor centre or the heart as is the case with the alkaloids of ipecacuanha. It excels the organic arsenical preparations in that it has no toxicity. It does not cause diarrhoea as is sometimes seen in other oxyquinoline compounds. In my series of 9 cases, it proved efficacious in destroying the vegetative forms of the amoebae from the stools in 2-5 days. It is given orally, 1-2 tablets three times a day till the tenesmus disappears. Then it may be given as a retention enema preferably at night, 4-8 tablets disintegrated in warm water. In chronic amoebiasis a combined oral and enema treatment can be given. In children a correspondingly smaller dose of Entero-Vioform may be administered.

Rivanol, an acridine derivative, is also given as a retention enema. It has a similar action like Yatren. But I prefer to give this drug as a retention enema as it relieves pain and tenesmus, on account of its antispasmodic and antiseptic action. I do not advocate its use by the mouth.

With the drugs mentioned above, one can be sure of tackling a case of chronic amoebic dysentery at whatever stage one may meet with. But there are also other drugs which might be helpful.

Stovarsol is an arsenical preparation (3 acetylamino-4 hydroxyphenyl-arsonic acid). I am accustomed to use it after the combined treatment is finished. Really it has a feeble amoebicidal action, but it possesses stimulating properties, serving as an antidote to the depressing qualities of emetine. One tablet (4 grs.) is given twice daily for 10 days. One has to watch for symptoms of arsenical poisoning as dermatitis; toxic erythema with pyrexia may also be noticed. There are similar preparations in the market known as Spirocid, Orarson (Boots), Acetar-sol etc. I have not much experience with these drugs.

A more recent preparation is Carbarsone. It is a pentavalent arsenical preparation. It is found to act both on the vegetative forms and cysts and is definitely less toxic than stovarsol. It does not, however, cure hepatitis or amoebic abscess. It is administered in pulvules containing 25 grains (or 4 grs), one pulvule being given twice daily after food for 10 days. It can be used in ambulatory cases on a normal diet, and from these points of view it possesses distinct advantages over the more toxic emetine preparations.

Kurchi and its derivatives are also used. The total alkaloids of the bark, according to Chopra is given in 10 gr. doses twice daily for 10 days, without producing deleterious effects.

Kurchicine hydrochloride, a crystalline alkaloid from kurchi bark has been used both for oral and hypodermic injections in doses of  $\frac{1}{2}$  to 1 gr. on three successive days. This is succeeded by kurchi bismuth-iodide (Anabin-B. C. P. W.) which contains the total alkaloids of the kurchi bark.

There are extracts of kurchi, as kurchibin, extractum kurchi liquidum etc., in the market. Although the first reports of the treatment with kurchi and its derivatives were encouraging subsequent experiences have been very disappointing.

Bismuth carbonate and other bismuth preparations are used in heroic doses in the treatment of acute and chronic conditions. I prefer to use bismuth carbonate, 60 grs. three times a day, suspended in milk or mucilage for 10 days. This can be given with small doses of Tr. opium or Liq. morphine hydrochloride 10 m. as in our General Hospital Pharmacopœa Mixt. Bismuth Sedativa. This drug has no action on the amoebæ or cysts and therefore has a limited scope in the treatment.

The most favoured treatment in cyst carriers, in my opinion, is to give retention enema in the morning and one E. B. I. at night or retention enema of rivinol followed by carbarsone. Emetine has no place in the treatment of cyst carriers.

During convalescence a non-irritating low residue diet reinforced with vitamins is advisable. Meat is very much restricted.

The results of modern treatment as a rule is satisfactory, though relapses are common in those half-treated or improperly treated cases, necessitating further course of treatment, no matter what drug is used.

As for treatment of complications, one has to remember the possible supervention of hepatitis with pyrexia and rigors. Hepatitis may occur some months or years after the first attack of dysentery. If pain seems to suggest abscess formation, repeated doses of emetine is given intramuscularly. Saline aperients, rest, low diet, fomentations, dry cupping and similar measures might avert graver complications. Emetine acts in these cases most marvellously and one need not be put to the necessity of giving more than a total quantity of 6 grains to relieve the symptoms of hepatitis.

In fulminating cases perforation of an amoebic ulcer may occur. But this is very rare and I have not seen a single case of perforation, and even if it occurs it is invariably fatal, unless immediately operated.

Other symptoms as abdominal pain is counteracted by means of hot applications, colic by means of a belladonna and bromide mixture, and tenesmus by morphia suppositories or by starch-and-opium enemata.

*Diet* is all important during convalescence. Co-operation of the patient is absolutely necessary in view of the tendency of this disease to relapse when the patient indulges in rich nitrogenous foods. Eggs, butter-milk, eledon, sago, rice congee, boiled apples can be taken. Cheese, potatoes, fats, puddings, cakes, spices, pickles and pastry of all kinds are forbidden.

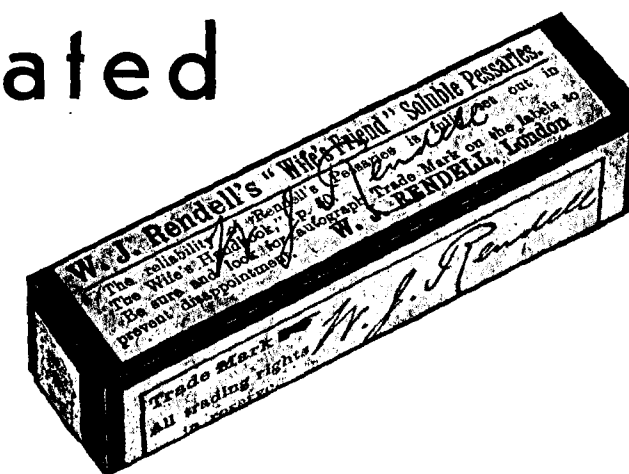
While one should be strict in this dietary, it is not necessary to starve the patient.

Surgical measures must be reserved for the complications of amoebiasis. Such heroic procedures as caecostomy have no place in the treatment of intestinal amoebiasis at the present day.

I wish to draw the readers' attention to the fact that there is considerable amount of evidence of the close association between sprue and amoebic dysentery. The two diseases often co-exist while on occasions sprue appears to be grafted on to intestinal amoebiasis. Possibly too, in the apparent absence of this disease from Central Africa may lie the clue to its true etiology. At any rate, sprue is a frequent sequel of amoebiasis.

What exactly determines the onset of sprue in a case of amoebic dysentery is not known and the treatment of sprue is not within the scope of this article.

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prepare Horlicks with milk or milk and water. Would a jelly be an attractive variation? Then make it with the addition of Horlicks.

## Choice of Methods and Drugs in the Treatment of Dysenteries

• Dr. A. Viswanathan, L. M. P., Madras

Methods of approach in the treatment of dysenteries are as important as the choice of drugs, and the writer may therefore be excused in laying stress on points which are of common knowledge among the practitioners but which are often unfortunately over-ruled by them due either to the importunate requests of the patient or the exigencies of busy practice.

The first irreducible minimum duty of a practitioner who is faced with a case of dysentery is to insist on at least a personal macroscopic inspection of the stools before they are passed on to the laboratory for examination.<sup>1</sup>

Particularly important and invaluable is this method in view of the fact that very few practising physicians of India can afford to have a microscope or have laboratories within easy access to them. Macroscopic examination would reveal, in more cases than we imagine, the true nature of dysentery with reasonable certainty before the laboratory report is received, and this would enable them to initiate specific treatment without any avoidable loss of time.

The second duty is not to yield at any cost to the natural request of a dysenteric patient for powerful astringents to stop his motions. This will be going against nature's method of combating the disease, as dysenteric evacuations are the best and safest means of elimination of bacteria and toxins. This process of nature must be helped rather than retarded. It is therefore incumbent on the physician to give a purgative forthwith, except in acute fulminating cases of bacillary dysentery, and in this no waiting for a convenient or conventional hour is permissible<sup>2</sup>. How this and what purgative should be administered will be discussed later.

It is suffice here to mention that, whatever may be the specific nature of the dysentery as may be subsequently revealed by laboratory and sigmoidoscopic examinations, the omission of preliminary purgative will prolong the duration and severity of the disease. In one word, purgative is our first inevitable and indispensable line of attack and in no case, unless it is distinctly contraindicated, should it be omitted.

The third duty is to realise the futility and the danger of rushing to use emetine before

the physician is convinced that the case before him is one of amoebic dysentery. More commonly than is supposed, bacillary dysentery is greater in incidence than the amoebic; therefore there is no need for haste in the administration of emetine. The writer is aware that it is used by some as a method of therapeutic diagnosis, but this method is inexcusable in a disease which is capable of quick diagnosis even by ordinary clinical methods. Even in cases where diagnosis is difficult, it is more prudent to withhold emetine than to use it in doubt.

While these are the essential attitudes, proved necessary by experience, there are still other methods of approach equally important in the successful treatment of dysenteries. This is the same thing as saying that one should have a proper perspective of the pathology and symptom complex of dysenteries. Without it, the treatment is bound to be shifting, aimless and inconclusive. With it, it will be precise, quick and fruitful of results, and, above all, a proper knowledge of the subject in all its aspects will suggest to one's mind proper methods of treatment in proper time. Thus for example the so-called hill-diarrhoea which was frequently reported in the Indian medical press a decade and two ago has been subsequently proved to have no connection with climate or altitude but has been traced to a variety of bacterial flora among which Flexner's bacillus presented a large incidence<sup>3</sup> & <sup>4</sup>. This is also true of dysenteries among the people in the plains: the bacillus of Flexner is important on account of its universal prevalence; the bacillus of Shiga on account of its virulence; entamoeba histolytica on account of its tendency to chronicity; and balantidium coli on account of its rarity<sup>5</sup>. While abdominal pain, tenesmus, gripping and frequent muco-sanguinous evacuations are typical symptoms of dysentery in a large majority of cases, particularly the acute cases, it must never be forgotten that this picture may be completely absent and replaced by the symptom of chronic diarrhoea of varying severity both in the amoebic and bacillary dysenteries, more commonly in the chronic and carrier types. Therefore, cases of diarrhoea should also be investigated from the point of view of its probable dysenteric origin, and the writer can recall to his mind many cases of diarrhoea, at one time thought by him to be due to indigestion, dyspepsia, tuberculosis and over-eating, proving ultimately to be due to amoeba or B. Flexner. Then, in the case of mixed infection, confusion is likely to arise as to which is the more important infection to be

dealt with. Commonly, acute bacillary dysentery supervenes on more or less chronic amoebic infection,<sup>1</sup> in which case the former would require prior treatment, but the case would still linger on if the latter infection also is not treated subsequently. Often the difficulty would arise as to when to use the anti-dysenteric serum or in which case rectal instillations of medicaments are needed. It must be remembered in this connection that serum is indicated in all cases of bacillary dysentery where the symptoms are acute and toxæmia high, and a polyvalent serum should be selected, as B. Flexner is also known to become as virulent as, if not more than, B. Shiga. As in the case of every serum, so in the case of anti-dysenteric serum it should be administered very early and in massive dose till toxæmia and fever are reduced. Mild cases of bacillary dysentery require neither serum nor vaccine, but the writer would warmly advocate autogenous vaccine in such cases to avoid, if nothing else, the passing of the case into chronic one which is always the despair of the physician. Rectal instillation of medicaments which vary according to the infection being amoebic or bacillary should be resorted to only in chronic cases, as the main purpose of instillation is the healing of the chronic and indolent ulcers in the lower part of the large intestines.

It is only after choosing a proper method of approach should the physician exercise his choice of drugs in the treatment of dysenteries. It may be stated at once that there is nothing else to choose except emetine or kurchi for acute and subacute amoebic dysentery, and sera and vaccine or specific bacteriophage for acute and subacute bacillary dysentery. But there is a wide range of choice of drugs in the treatment of chronic and relapsing dysenteries, most of which have to be chosen by the only method of trial and error. Oxyquinoline derivatives like "Yatren 105" do some good in, and oftentimes wholly eradicate, chronic amoebic dysentery, while yet in others arsenical derivatives like carbarsone, amibiarsol or stovarsol have a strong reputation. Indication for demulcents is common and insistent in all types of dysenteries, and they may be given with safety and comfort to the patient. The pharmacology and therapeutics of some of the more commonly used and available drugs will be described below.

#### PURGATIVES

Since dysenteric organisms mostly inhabit the intestinal canal either in the submucous

coat or lie loosely in the lumen of the large gut, the only safest and natural way of expelling them and their toxins is by means of mild purgatives, and, as has been said earlier, this is our first and indispensable line of attack. One ounce of castor oil, immediately when the case is seen, must be given—the dose being adjusted according to age—and this may be repeated everyday till the bowel is empty of faecal matter. It is then best to begin the saline treatment.<sup>2</sup> If the case turns out to be bacillary dysentery the treatment will simply be continued, and if it is amoebic the patient will benefit equally, and as soon as the diagnosis is established the dose of saline can suitably be adjusted.<sup>3</sup> As a rule amoebic cases require only a few days of saline treatment while the bacillary cases should have the benefit of a more prolonged period of daily evacuations by means of regulated doses of saline. Castor oil emulsion is more suitable for children, since they retain this much better than the saline. If the pain, griping and tenesmus are too severe, there is no objection for the addition of small doses of astringents such as Tr. catechu, Tr. opii or chlorodyne, and a little of Tr. belladonna or hysocyamus. It may be mentioned here that administration of alkalies in bacillary dysentery is of no real use or necessity as the pH of the intestinal contents in this infection is already alkaline ranging between 5.4 to 9.1. Contrary is the case in amoebic dysentery where the intestinal contents are acid and therefore it requires alkaline drugs like bismuth carb and sodi bicarb.<sup>11</sup> The following prescription is recommended by Manson-Bahr for cases of bacillary dysentery:

|                     |     |        |
|---------------------|-----|--------|
| R/ Sodium sulph     | ... | gr. 60 |
| Acid sulphuric dil. | ... | m. 15  |
| Tr. Zingiberis      | ... | m. 5   |
| Aqua mentha pip     | ... | oz. ½  |

½ oz. every 4 hours till the stools become faeculent, later every 6 hours and still later three times a day.

In amoebic dysentery, acid sulphuric dil. should be omitted for reasons already explained, but heavy doses of bismuth carb should be given when the effects of saline are over. The details are as follows: Saline mixture till the bowels act freely; now stop saline and administer 3 drs. of bismuth carbonas every 3 hours till the bowels are confined; again give saline, to be followed once more by bismuth.<sup>8</sup>

#### INTERNAL INTESTINAL ANTISEPTICS

While there are a legion of vaunted intestinal antiseptics, none of them have yet been found to produce even a fair degree of intes-

tinal disinfection and their use, therefore, in dysenteries has never been hailed with any enthusiasm and satisfaction. It is in the caecum and the colon the dysenteric lesions lie, and it is exactly the places in which it is difficult to get an antiseptic to act with its full power.<sup>5</sup> Besides, healthy intestinal mucosa which normally possess germicidal action like other mucous membrane, becomes devoid of this property by the use of antiseptics.<sup>8</sup> Salol and dimol are the drugs which are usually used for this purpose, but for the reasons mentioned above they are practically ineffete, though they may be tried if the patients tolerate them. Perhaps, the time-honoured calomel is more useful for this purpose, but it must be recognised that it acts not by any bactericidal action but by expelling the putrefying contents of the gut.<sup>8</sup>

#### DEMULCENTS

What we fail to achieve by intestinal antiseptics may be obtained by a judicious use of demulcents which possess the additional advantage of allaying or arresting the inflammation and necrosis of the dysenteric ulcers. They form a protective coating over the whole of intestinal mucosa and absorb bacteria and toxins.<sup>8</sup> Their action is purely mechanical, and they are devoid of all toxicity even when given in large quantities. The most useful demulcents in dysentery are acacia combined with bismuth carbonas, kaolin or osmokaolin, and ispagul. Bismuth, of course, is the drug of choice, and if proper effect is desired it should be given in heavy doses, say 2 drs. well emulsified with pulvis acacia or tragacanth or mucilage. This effect is enhanced if it is given when the saline has acted fully. Bismuth poisoning is rare since it is insoluble. The carbonate is better tolerated than the subnitrate and its effects are purely physical: in the stomach, it protects it from the irritation of the food and secretions, and removes the mucoid slime; lower down in small intestines, it acts as a protective covering to the mucous membrane, diminish the stimulation of the bowel by the food and secretions contained therein and consequently lessen peristalsis; it acts like the lead lotion when applied to the inflamed part of the skin.<sup>7</sup> Besides these salutary effects, it also acts as a mild astringent. Kaolin is aluminium silicate and is noted for its demulcent qualities. One hundred grammes are mixed with 250 c. c. of water, and half a pint of this is given every ½ hour for the first 12 hours, continued if necessary. Colloidal

kaolin or osmo-kaolin is usually preferred being more certain and definite in its effects. Mucilage of ispagul is a demulcent of Indian antiquity and reputation, a favourite drug for dysenteries with the ayurvedic and unani practitioners. It is physiologically inactive and the tannins contained in the shell have little action on the entamoeba or bacteria. Its chief glucoside is aucubin and its action is purely mechanical due to the large amount of the mucilage contained in the superficial layers of the seeds. It acts in the same way as liquid paraffin but is much cheaper than and free from injurious effects of the latter.<sup>3</sup> The ayurvedic practitioners separate the shell from the seeds by crushing and the husk by winnowing, and administer the mucilage-containing shell soaked in a cup of water, flavoured with sugar or syrup of lime. The other but less commonly used methods of administering ispagul are: (1) soak a teaspoonful of cleaned and dried seeds in a cupful of water, stir and take, (2) seeds are allowed to stand till the mucilage comes out, and this mucilage is administered separately. It may be mentioned in passing that Alembic's liquid paraffin contains ispagul.

Dysulin is a non-toxic herbal preparation, but from what herb it is prepared the writer does not know. In a personal communication from the manufacturers, The Dysulin Pharmaceutical Co., it is stated that it contains no alkaloids, and that ispagul is one of its constituents. Given in good doses every hour or two followed by copious draughts of water it forms a mucilaginous matrix in and around the ulcerated bowels and thus act as an efficient emollient and adsorbent. The writer has used Dysulin for nearly two years between 1930 and 1933 and is satisfied with the results. It does not certainly act as a specific, but the relief of dysenteric symptoms which it affords is definite, pronounced and quick. It is handy and pleasant to take, and is available in powder form. It is not toxic, and hence could be given in big doses according to indications. It is obviously an empirical remedy but none the less very useful in the alleviation of the acute and chronic symptoms of both amoebic and bacillary dysentery. Its use is analogous to Felsol in asthma.

#### ASTRINGENTS

The writer's experience is that astringents are better avoided in the treatment of dysenteries as they tend to make the disease chronic by masking the symptoms, lull the patient to a false sense of security and expose him to the temptation of giving up medical

treatment before it is duly completed—a too common failure among our people. For these reasons alone, if not for any other, astringents should be shunned. In no case should tincture of opii should be chosen, and if astringents are imperatively indicated owing to too frequent evacuations, abdominal pain, griping and tenesmus, they should be combined with the saline or the castor oil emulsion or given separately along with the latter. Chlorodyne, Dover's powder, pulvis tannicum, pulvis or tincture catechu may be used alongside the saline mixture, but never alone. *He is a wise physician who uses astringents less and mild purgatives more.*

#### SEPECIFIC DRUGS

##### *Acute amoebic dysentery*

**Emetine** is the active principle of ipecacuanna, first prepared by Pellatier and Magendie in 1817, introduced into medicine only in 1913 by Rogers in our country<sup>1</sup>. Since then it has been a sheet anchor in the treatment of amoebic dysentery. There was a time in our country, not long ago, when it was thought that amoebic dysentery was more common and widespread than bacillary dysentery. This led to the promiscuous use of emetine. But thanks to the Calcutta School of Tropical Medicine, and the successive editors of the *Indian Medical Gazette* who were drawn from the Calcutta School, the position was cleared and the abuse of emetine stemmed effectively. It is now proved beyond any shadow of doubt that emetine is effective only in acute and subacute amoebic dysentery and it should be used only in these conditions. To put it the other way, it is practically of no use in cases of chronic amoebic dysentery and amoebic carriers in whom the chief villain of the piece is the encysted amoeba. It needs also no reiteration that its use in bacillary dysentery is a futile folly and an act of ignorance. Emetine is no doubt lethal to the vegetative forms of entamoeba, but being a potent drug it has to be administered in small and graduated doses in order to eradicate the amoebic infection. The maximum dose for a full course of treatment is about 15 grs., spread over a period of two weeks or three with not more than a grain per injection. In very acute cases, intravenous administration is advocated<sup>2</sup> but the writer feels there is no necessity for such a drastic procedure as in his experience intramuscular injections give equally good results. Thus intravenous injection of the drug is unsafe and unsuited to Indian patients,

while the hypodermic injection cannot be recommended because of the attendant pain and induration, and even of a sterile abscess in some cases. Barnett<sup>3</sup> advocates the combined oral and injection method, and accordingly recommends strongly  $\frac{1}{2}$  gr. of emetine in gelatin capsule by mouth. But this has never been a favourite method with our practitioners, and the writer had not even once adopted it during the last 20 years of his practice. Oral emetine sickens the patient beyond doubt, and there is no imperative necessity for the same. The effect of emetine is enhanced if the patient is given bismuth during the period of injection. At the end of treatment it may be that the stool still shows free entamoeba, but this does not need further course of emetine as the patient's own natural powers of resistance finally exterminate the residual infection<sup>1</sup>. The toxicology of emetine is well worth remembering. It increases peristalsis and lowers the blood pressure. It may give rise to neuritis, mental depression and cardiac irregularities as evidenced from the characteristic "emetine pulse". If the patient shows pronounced intolerance to emetine, it should be stopped and an alternative drug should be chosen.

**Kurchi Extract:** The alternative drug is the standardised preparation of the total alkaloids of kurchi bark. Kurchi bark and seeds enjoy a well-deserved reputation in ayurvedic medicine as a standard treatment for dysenteries in the form of decoction or powders. But its alkaloids were only isolated by Hains in 1853 and by Dutta in 1880. Its chief alkaloid is kurchicine which has a specific action on *E. histolytica*, but the total alkaloids seems to be more useful than kurchicine itself separately. A reliable standardised alcoholic extract of kurchi is now available and is the best form of exhibiting the drug. A sufficient dose of this liquid extract, totalling 3 to 4 grs. of the alkaloids per day, and continued so for a fortnight, is an efficient and dependable amoebicide<sup>2</sup>. Preparations for intramuscular injection are also available, and nearly 30 grains of the total alkaloids form a full course. Tablet preparations are also available, and can be used in patients who refuse to take the liquid extract owing to its extreme bitterness. Compared to emetine, kurchi is rather slow in action, and does not produce that dramatic effect which writer has often experienced with emetine in some cases of acute amoebic dysentery. But in other respects, kurchi is certainly superior to emetine. It is non-toxic,

## SPECIAL SUPPLEMENT TO THE INDIAN MEDICAL JOURNAL *Dealing with the Indian Medical Council Act*

MADRAS

SEPTEMBER 15, 1939

No. 9

### CONTENTS

|                                                                                                 | Page  |                                                                                                                                      | Page  |
|-------------------------------------------------------------------------------------------------|-------|--------------------------------------------------------------------------------------------------------------------------------------|-------|
| Oppose the Indian Medical Council Act by all constitutional means—Dr. D. V. Venkappa            | - 153 | A Model Medical Council for India—Dr. A. Viswanathan                                                                                 | - 157 |
| Twentieth September—Its significance to the Indian Medical Profession—Dr. U. B. Narayana Rao    | - 153 | The British Medical Association Conference—Broadcast by A London Doctor ( <i>Exclusive for the Indian Medical Journal in India</i> ) | - 160 |
| Boycott the Indian Medical Council—An appeal to the medical graduates—Dr. Kunj Behari Lal Varma | - 154 | L. P. H. Officers' Association, U. P.—A Deputation to the Hon'ble Minister of Health                                                 | - 161 |
| Indian Medical Council—A Retrospect—Dr. K. S. Ray                                               | - 155 | Association Notes                                                                                                                    | - 162 |

War News—See page 155

An Appeal to the Medical Profession

### Oppose The Indian Medical Council Act by All Constitutional Means

#### ● Dr. D. V. Venkappa, President, All-India Medical Licentiate's Association

This Special Supplement dealing with the Indian Medical Council Act would perhaps reach the members of the *Association* before the 20th of this month which has been marked by the profession as the Anti-Indian Medical Council Act day. The Editor of the *JOURNAL* has asked me to give a message to the members of the *Association* and others of the profession on the eve of this the 6th annual protest day, so that the fight against this Black Act may continue with redoubled vigour till the wrong is righted. What message can I give except to say, in the words of L'Estrange, that of all injustice, the greatest is that which goes under the name of Law; and of all sorts of tyranny the forcing of the letter of the Law against equity is the most unsupportable. The Indian Medical Council Act is a law and tyranny of this kind. It leaves unrecognised the majority of medical practitioners recognised by provincial statutes. It was passed in the teeth of an almost unanimous opposition. It was conceived in terms of our political slavery, and worked as between the ruler and the ruled. And yet it thrives in our soil! Why? It is because we are divided amongst ourselves. We do not show an united front. That magnificent opposition, witnessed at the time of the passing of the Act, has given place to complaisance, and even to co-operation in the working of this Act. We have materially damaged the cause and obscured the goal of the Indian Medical Profession by such complaisance and co-operation. My message to the profession, at any rate to the members of our *Association*, is therefore to warn them not to become apathetic, not to become luke-warm, not to lull themselves into a false sense of security, and not to allow the poison to become fatal, in a matter so vital to the profession. The success of this fight depends on our strength and unity. Let us no longer think in terms of licentiates and graduates. Let us

### Twentieth September—Its significance to the Indian Medical Profession

#### ● Dr. U. B. Narayana Rao, Editor, Medical Digest and Indian Journal of Venereal Diseases.

The Indian Medical Profession has been observing the 20th September of each year as the day of protest against the Indian Medical Council Act. Five years of persistent agitation has not moved the Government a bit. But concerted action will surely mean success to the profession ultimately. On the eve of this sixth Anti-Indian Medical Council Act day, I appeal to the profession to offer an united front, utilize all possible means and explore all possible avenues of attack. I would suggest a joint committee of the two premier medical organisations of the country to draft an amendment to the Act, acceptable to the majority, and on this basis start a systematic and uniform propaganda through the press and platform, through deputations to Ministers and the Members of the Legislatures, through the provincial medical and legislative councils and through the Congress which commands a majority in the country. I have also a word to say to the present members of the Indian Medical Council. Many of them feel in their heart of hearts the injustice done to the profession by the Act as it stands to-day. Some of them happen to be leading members of the Indian Medical Association who led the frontal attack in 1932-33 against the passing of the Act. I would appeal to them also to do their duty.

never forget that what affects the one will ultimately affect the other. Let us remember always that none can harm us except ourselves. Let us be true to the ideals of the profession which are based on the bed-rock of service, and the concord to relieve suffering by all possible means. Suffering knows no caste or creed, and there should be no such artificial status of high and low among the members of the healing Art.

## Boycott the Indian Medical Council—An Appeal to the Medical Graduates

● Dr. Kunj Behari Lal Varma, Provincial Secretary, All-India Medical Licentiates' Association, U.P., Meerut

The object of creating a Medical Council in any civilised country is to provide a machinery for laying down a standard of medical education to be followed by all medical teaching institutions and to register qualified practitioners and thus protect the public from unqualified persons. It is entirely in public interest that such councils are created. Following this principle the General Medical Council of England (G.M.C.) was established by the Medical Act of 1858. At that time there existed in England, Wales and Ireland Universities which conferred medical degrees and two different corporations which conferred their own diplomas, i.e., Licentiate of the Royal College of Physicians and Surgeons and Licentiate of the Society of Apothecaries. With the passing of the Medical Act, the British Universities as well as these corporations were required to conform to the standard laid down by the General Medical Council, thus covering all the medical teaching institutions. Although so many medical schools and colleges existed at that time which were affiliated to the Universities and corporations referred to above, it was also provided in the British Act that unqualified persons who did not pass any examination or possess any qualification but practised allopathic system of medicine before 1st August 1815 (Section 17 of the British Medical Act) were also to be registered along with the qualified persons. This shows the liberal outlook of a self-governing and self-respecting country towards its nationals.

In her own interest, India also needed a similar Medical Council but the noble attempts of two worthy licentiates, who happened to be members of the Central Legislative Assembly, in bringing forward a bill about ten to fifteen years ago to establish an Indian Medical Council were opposed and suppressed by the Government of India. In 1929 the G. M. C. suddenly refused recognition of medical degrees of some of the Indian Universities on the plea of defective teaching in midwifery when an extensive agitation against this move was started. The British Government of India in order to placate the G. M. C. on the one hand and to appease popular feelings on the other conceived an Indian Medical Council Act which gave birth to a council actually servile to G. M. C., unrepresentative of the profession and with an official majority. An intensive and extensive agitation of the entire medical profession in India was started against the Government Bill, but with official manoeuvres it was enacted and thrust upon the country after some minor alterations.

The chief features of the existing I. M. C. Act are:—  
(1) It has limited its jurisdiction to the 7 or 8 University Medical Colleges and medical graduates number-

ing about 5000, thus covering only a minority of the profession—an abnormality not existing in any other country.

(2) It has excluded from its purview the majority section of the qualified medical profession of India and all the Government and Government-aided medical schools which trained medical licentiates and some of which are as old as the G. M. C. itself—another monstrous absurdity nowhere to be found: a Government denouncing its own institutions numbering 29 and their alumni numbering about 28,000.

(3) The I. M. C. does not maintain a register of qualified practitioners like G. M. C. or like medical councils of other civilised countries—another funny abnormality.

(4) It has provided a few seats for medical graduates on the council thus creating a communal outlook amongst them and dividing the profession.

Thus the Indian Medical Council Act is not like similar acts of other civilised countries including England, but it is more like a magician's wand or a snake-charmer's bag containing a variety of emasculated creatures dancing to the tune and obedient to the behests of their master.

It is a misnomer to call the Indian Medical Council by that name, because it keeps out of its purview the greater part of statutorily qualified medical profession of our country consisting of about 28,000 legally qualified medical licentiates. It therefore does not signify what its name implies.

These medical licentiates, like medical graduates, are registered in every province of India since the passing of the different Provincial Medical Acts as far back as 1915 and are employed by Government as medical officers of dispensaries and of jail and police hospitals and in subcharge of big district hospitals throughout the length and breadth of our country. Some of them in Government service, in spite of handicaps and obstacles, have gained distinction in the profession and have been promoted to Provincial Medical Service; some have also held charge of civil surgeoncies with credit. A large majority of the medical licentiates are engaged in private practice as independent general practitioners, surgeons, physicians, eye surgeons, and bacteriologists etc. Innumerable instances can be quoted where medical licentiates have attained marked distinction due to their professional ability and skill in public service and in private practice. Dr. Mathra Das, Eye Surgeon of world-repute, is a licentiate. There are at present some licentiates in the Punjab who have beat the record for major eye operations. One of them is now holding charge of an important eye centre out of the three eye centres in the whole of the Punjab to which he was specially deputed and selected by the authorities against I.M.S. and medical graduate aspirants for that post. Similarly in the capital of the United Provinces it was a medical licentiate who held charge of an eye hospital for a number of years since it was opened. In general surgery, many licentiates have won the appreciation of the Govt. and the public.

In short, in different branches of medicine and surgery licentiates throughout India have been giving a splendid account of their professional skill and abilities; but as their work lies chiefly amongst the poor and voiceless masses and mostly in the remotest corners of the country, it is not so widely known, but known only to those amongst whom they work in their private practice and to their immediate superiors in Govern-

ment service as was publicly expressed by His Excellency Lord Chelmsford in 1920.

Thus it will be clear that on political considerations alone the Indian Medical Council Act was enacted. It is designed to stifle the majority section of the Indian medical profession. It is only a subordinate body, because the British Medical Council is still the medium for reciprocal registration with foreign countries. It is only meant to proclaim to the world that India with a population of 35 crores had got but a few qualified practitioners and hence was politically

so much backward. It has effectually debared a large body of qualified nationals from sharing their rights and privileges in and outside the country by reserving them for British and Indian vested interests and by curtailing the number of Indian eligibles. That a medical practitioner legally qualified and registered in all the provinces of India should be declared as a non-entity under an Act which has assumed a high sounding, but misleading name of the Indian Medical Council Act is really unthinkable in any civilised and self-governing country. It is possible only in a subject country like India. The most depressing part of it is that the new autonomous Provincial Governments and Provincial Medical Councils should send their nominees to this so called Indian Medical Council while their own registered practitioners are rejected by the latter. Melancholy and sickening is the mentality of those men who take pride in being nominated or elected as members of this monstrous Council and who pass as leaders of the profession. The only action in keeping with our national and professional self-respect is immediate withdrawal of co-operation with the Indian Medical Council by our graduate comrades. Really it is extremely degrading that our own men should adorn this Council and give it an undeserved reputation and a longer lease of life. It has already involved our profession in indignity and disruption at home, and disgrace abroad. It is high time that medical licentiates should adopt more effective methods of fighting this Act. The ceremonial of observing 20th September every year as Anti-Indian Medical Council Act Day by holding protest meetings throughout our country would no longer help them or erase the insult which has been levelled against them and the country. They must come in closest personal touch with the co-operating party, rouse them to the indignity which their alliance with the Indian Medical Council implies, and ask them to boycott it.

May God give us the wisdom to bury the communal outlook and ambition of the now divided profession, and the glory to be the torch-bearer of India's professional honour at home and abroad.

## Indian Medical Council

(A Retrospect)

● Dr. K. S. Ray, M. A., B. Sc., M. B., Ch. B. (Edin.), General Secretary, Indian Medical Association

Although the Indian Medical Council has now been in operation for six years, having come into existence in the year 1933 as the offspring of the Indian Medical Council Act of that year, few of the public as also of the medical profession know what the Council actually is and what are its activities. As one who had an intimate knowledge of the various phases of the evolution of the Indian Medical Council Bill, I propose to deal first with the question as to why the Medical Council was at all brought into existence. It will be necessary in this connection to recall the history of the controversy that raged round the withdrawal of recognition of the Indian medical degrees by the General Medical Council of Great Britain.

Since 1892, the Indian Universities had enjoyed the right of having their medical degrees registered by the General Medical Council of the United Kingdom. The rights conferred by registration were that the holders of Indian medical degrees could practise medicine, surgery and midwifery in Great Britain and Ireland, in certain parts of the British Empire and in certain foreign countries where British degrees so registered are recognised. Conditions after the war, however, when the number of Indians in the medical profession and teaching institutions increased enormously, Indians making their way even into the ranks of the Indian Medical Service, and when the direct control hitherto exercised by Government was relaxed as a result of the constitutional reforms, led to apprehension (engendered mainly, it is believed, by the British members of the I. M. S.) on the part of the General Medical Council as to the efficiency of the standard of teaching in Indian Universities and in 1922 there developed a protracted controversy. An investigation resulted in the G. M. C. intimating that

## WAR NEWS

### AN APPEAL TO THE MEDICAL PROFESSION

As this supplement goes to the press, the information that Germany has begun to attack Poland is given in the newspapers. It is a great pity that the German Chancellor has rejected Chamberlain's repeated offers of peaceful settlement of German-Polish differences. Humanity is thus put in travail—and none can forecast the day of its deliverance. In this hour of trial and of peril to democracies, we the members of the Indian medical profession should rally round the allied banners of democratic countries and help them individually and collectively by prayers for their success, by pecuniary contributions and by organising a medical ambulance to be placed at the service and disposal of the Government of India. In the last war, our Association had sent many men to the colours who distinguished themselves by acts of heroism and valour, and who rendered useful work in field medical service. It should do no less in the face of a greater danger today. 1-9-39.

[Ed. I. M. J.]

because of insufficient practical experience in mid-wifery they would be obliged to discontinue their recognition of the medical degrees of all Indian universities, except Madras at the time, unless the defects were remedied and some guarantees were forthcoming that their minimum requirements would, in future, be fulfilled.

The G. M. C. stated they would continue recognition pending the formation of an Indian Medical Council if a Commissioner of Medical Qualifications and Standards in whom they had confidence were appointed. Col. Needham was occupied in this work from 1922 to 1927 during which period the recognition was not withdrawn, the universities being found fully up to the standard, and there was no reason why the arrangement should not have been continued. But subsequently the G. M. C. considered that it could no longer negotiate with the universities in India individually and insisted on the appointment of a Commissioner as suggested above to be appointed by the Government of India to deal with the universities as a whole. The chief objection to this suggestion was that the medical institutions in this country would not tolerate the idea of a dictator, especially as the G. M. C. insisted upon the appointment of their own nominee, Col. Needham. It was also felt that no one person could be expected to combine in himself all the qualifications required for the post.

Although the proposal to constitute an All-India Medical Council was first mooted by a non-official member of the Council of State, the Hon'ble Dr. Lohokari, beyond circulation of the Bill for public opinion no further attention was paid to it by Government with the result that it made very little progress. But after the withdrawal of recognition of Indian degrees by the G. M. C. public feeling in the country was so much roused that various meetings and conferences were held to consider the steps to be taken by the universities. An inter-universities' conference was held in Bombay in 1930. The conference protested against the action of the G. M. C. and recommended that the standard of qualification should be regulated by an inter-university Board to be duly constituted as early as possible. This proposal was also not given effect to, but instead of that the Government of India, at the suggestion of the G. M. C. tried to foist Col. Needham as an Inspector of the Indian universities. The proposal was naturally opposed by the universities as well as by the I. M. A. and the whole medical profession, and was consequently thrown out by the Legislative Assembly. The G. M. C. then withdrew their recognition of Indian medical degrees. It served only to exacerbate feeling in this country as it was looked upon as a measure of retaliation, since there was nothing to suggest that the standard of medical teaching in the Indian universities had deteriorated to warrant such action. So greatly was the insult felt that, though some advocated

that redress might be had upon appeal to the Privy Council, that privilege was never exercised.

The Government of India, then at the suggestion of the G. M. C., once again brought the proposal for introducing a Medical Council for India to the forefront. The original clauses of the Bill which were circulated for public opinion provided not only for the inclusion of the Licentiate but also for a Central Register. But subsequently when the Bill was finally drafted and introduced in the legislature, the question of the Licentiate was entirely dropped; so also the question of the Central Register. The Government became unduly apprehensive that inclusion of the Licentiate in the Bill might lead to difficulties in obtaining recognition of Indian degrees by the G. M. C. To the deputation of the Indian Medical Association which waited upon Sir Fazl-i-Hossain and of which I was a member, he pointed out that till the local Government said that they wanted one standard of medical education and not two, the question could not be considered on the same basis. So the decisive voice lay with the local Governments who were all agreed on fixing and controlling the University minimum standard of education and not the school standard. Therefore, the proposed Bill, beyond ensuring a uniformity in the standard of higher qualifications had not tried to do practically anything. This, in short, is the history of the exclusion of the Licentiate from the Indian Medical Council.

The Act which thus gave birth to the Council, placed on it responsibility, besides the maintenance of a uniform standard of higher qualification in medicine for all provinces, of settling schemes of reciprocity with the authorities of States or countries outside India which are entrusted by the laws of such States or countries with the maintenance of a register of medical practitioners. While the Act empowered the Council to enter into schemes of reciprocity with those countries which maintained a register of their practitioners, it did not arm the Council with that effective weapon with which such negotiations can be successfully concluded, viz., a register of its own. Needless to say that omission of this important provision has resulted in a compromise of India's honour and dignity, some of the countries with which reciprocity has been established having refused to enter into direct reciprocal relation with India and asked her to come through the G. M. C. This is an anomaly to which pointed attention has been drawn by the Indian Medical Association.

Sections 13 and 14 of the Indian Medical Council Act had conferred upon the Council the right to settle schemes of reciprocity with countries outside British India and to recognise or refuse to recognise such qualifications which the Council considered sufficient or insufficient for the purpose of recognition. But the new Government of India Act has considerably modified these powers so far as

reciprocal relationship with United Kingdom is concerned. The present position is such that even if the Indian Medical Council considers it desirable to remove any British qualification from its schedules it cannot do so, unless the provincial or central legislature passes an Act which debars such degree-holders from practising on the ground of inefficiency of qualification. Even if suitable legislation is passed by the Central and Provincial Governments, there will still lie the right of an appeal with the Privy Council, whose decision is more likely to be in favour of the British Universities. The recent experience particularly in the case of the Calcutta University also raises another question. Although the Medical Council of India recommended recognition of the Calcutta University degree from the year 1930, the G. M. C. refused to accept the recommendation on the ground that the inspector's report contained some adverse findings. The G. M. C. also maintained that they are not bound on the ground of reciprocity to recognise degrees recommended by the Indian Medical Council but they must satisfy themselves as to whether such qualifications are considered sufficient by them. Thus in actual practice the principle of reciprocity is reduced to mockery!

The other unsatisfactory feature of the Act to which also repeated attention has been drawn is its provision relating to the constitution of the Council, the exclusion of the Licentiate who constitute about 3rd of the medical profession in the council has already been referred to at some length, the nomination of the President and the appointment of the Secretary by the Governor-General-in-Council for the first four years. There is a large preponderance of nominated element which has always stood in the way of the reformist zeal and activity of the elected representatives of the profession. Since the creation of two new provinces, there has been distinct accession of strength to the nominated element. On the other hand some of the provinces have not yet been able to introduce registers so that real representatives of the profession might take their seats in the Council and thus counterbalance the strength of the nominated group. The position was such that when after the expiry of first four years, a President had to be elected, all the efforts of the non-officials to have a President elected from amongst them were defeated. It seems to be an anomaly that although the Act provides for an elected President, the Council has not yet been able to get an Indian elected to the office of the President although there is fortunately no dearth of able, qualified and eminent non-official medical men to do honour to this office both by virtue of their long and intimate connections with educational institutions and their deep knowledge and understanding of the peculiar problems of medical education in this country, to mention nothing of their own academic honours and professional distinctions.

## A Model Medical Council For India

(With a criticism of the present Indian Medical Council)

● Dr. A. Viswanathan, L. M. P., Madras

Act No. XXVII of September 1933 ushered into existence a Medical Council for India, ostensibly to establish an uniform minimum standard of *higher* qualifications in Medicine for all the provinces, but in reality to ensure the supremacy of the General Medical Council of the United Kingdom over affairs medical in our country. It was an Act conceived in terms of our political slavery, wrought by the enemies of Indian Medicine and delivered by a Government not based on people's franchise.

The passing of this Medical Council Act in the face of an almost unanimous disapproval by the profession has revealed to its bitter shame, for the hundredth time in its chequered history, that it has still to be, for sometime to come at any rate, under the British ægis, just as in other matters of national importance. No self-respecting Indian can permanently reconcile himself to this status of obscurity, subordination and contempt.

That is the genesis of the protest against this Act, and this protest will continue till the honour and self-respect of our profession are restored. Let not the powers-that-be overlook this aspect, and beguile themselves that we will be satisfied with a few crumbs of amendments. The whole Act must be repealed and drawn afresh on the canvass of Indian nationalism.

What is there to amend in this Act where every word, every sentence and every paragraph in it breathes the spirit of imperialism and reminds us of slavery? Its very preamble is defective. Its scope is vindictively narrow. Its constitution is autocratic. Its composition is a caucedom of sycophants. Its reciprocity is beggary on bended knees. Abroad, all these have put us low in international esteem, and, within, have perpetuated divisions and castes in the profession.

It is not that we quarrel with the standards it has set up. The profession has accepted with one voice and one mind, even without suggestion from outside, the need for a high standard of medical education, not because medical councils of other countries demand it for purposes of reciprocal recognition but because that is good for our people. Already most of the recently elected Provincial Governments have taken measures to enlarge and improve the present standards of medical education under their purview and control. This shows that our statesmen are keenly alive to the needs of a high standard of medical skill for the people of our country.

Therefore, it is not true to say that the opposition of the profession to the Medical Council of India is based on the desire or the need for the continuance of the lower standard. That is our

enemies' argument but not ours. The opposition is based on the following facts which, for brevity sake, are given categorically:

(1) It was conceived by the G. M. C. for the benefit of the G. M. C.

(2) It had treated with contumely a large majority of medical practitioners statutorily recognised by the Provincial Medical Councils.

(3) Instead of recognising the existing practitioners, and making attempts to remodel the medical schools within a reasonable limit of time, it has destroyed their long and hard-earned reputation by their non-recognition.

(4) It is irresponsible to organised medical opinion in this country and acts as a hand-maid to the British Medical Council.

(5) Its preamble, scope, composition and constitution are but contemptible devices of bankrupt statesmanship intended to keep the profession under perpetual tutelage.

Contrasting the Medical Council of India with the General Medical Council of the United Kingdom one will find a wide gulf of difference between the objectives of these councils and the ways by which these are put into practice. The latter is intended for its people while the former is obviously intended to placate the G. M. C. Let the readers closely follow my analyses, and they will find me just and right.

The duties of the General Medical Council are four-fold: (1) It is responsible for registration. (2) It ensures that there is a definite minimum of medical education and examination requirements. (3) It is responsible for the discipline within the profession. (4) It has the exclusive right of publication of the British Pharmacopoeia.

What are the duties of our Medical Council? They are: (1) The maintenance of a uniform standard of higher medical qualifications for the whole of British India. (2) The furtherance of the recognition of these qualifications in States and countries outside British India and obtaining of reciprocal recognition.

Imitation is the best form of flattery, but our Medical Council only flatters without even imitating its master. Where is the urgency to establish uniformity of higher medical qualifications before attempting to establish uniformity of minimum medical qualifications? It is the latter that is vital to the profession as well as to the people and unless a medical council safeguards the indispensable minimum of medical standard and skill it has no right for statutory existence, it cannot call itself by the high-sounding name of a medical council and it has no right to ask of our sympathy and support.

Where, again, is the need to divide India into British and Indian, as if the Indian States form a different political and geographical entity? The Mysore and Osmania Universities are as much Indian as the Bombay or Calcutta Universities. This is a reproach to our national solidarity. We refuse to reconcile to this position.

Why, again, is this unholy haste to gain foreign reciprocity, and what is the result of this enterprise of begging during the few years of its miserable existence? All the countries have replied that unless our qualifications are included in Table 1 of the British Medical Register as published from time to time by the General Medical Council of the United Kingdom they would not be recognised. We refuse to invite and pocket this insult.

Where, again, are the Indian Medical Register and the Indian Pharmacopoeia? Our Medical Councils' *vile mecums* are the British Medical Register and the British Pharmacopoeia. While we respect them we refuse to be over-awed by them.

Let me contrast the composition of both these Councils.

The G. M. C. is composed of 42 members made up as follows:

|                                          |     |    |
|------------------------------------------|-----|----|
| Universities                             | ... | 18 |
| Licensing Corporations                   | ... | 9  |
| Crown                                    | ... | 5  |
| Direct representatives of the profession | ... | 7  |
| Under the Dental Act                     | ... | 3  |

The Crown nominees and University representatives need not necessarily be medical men. (At present there is one lay member sitting in the G. M. C.)

The Medical Council of India is composed of 28 members made up as follows:

|                                                                                 |    |
|---------------------------------------------------------------------------------|----|
| Provincial Government nominations...                                            | 11 |
| University Medical Faculties                                                    | 6  |
| Provincial Medical Councils' representatives other than the medical licentiates | 7  |
| Central Government nominees                                                     | 4  |
|                                                                                 | 28 |

This composition, though apparently sound, would show on ultimate analysis a relative lack of democratic representation as the sources from which it is drawn are more or less from official-ridden bodies, as in reality both the provincial medical councils and the universities are at present. The Council, therefore, is a *de facto* coterie of reactionaries who are accustomed to worship the British God of medical education although they may be Indians by birth and colour.

Apart from this, if the electorates for direct representation of the profession are the Provincial Medical Councils, how and why is it that they are not taken as a whole except the numerically insignificant members of the provincial register? Obviously, the implication of the Medical Council Act is that the majority of the provincially

registered practitioners are not qualified to be entitled to exercise their franchise. We resent this implication which puts a low premium on a qualification recognised by the Provincial Medical Statutes. There cannot be one law for a Province and another for the Centre on the same subject of legislation.

It may be said that the composition of the Indian Medical Council is more or less akin to the General Medical Council, but it is only so apparently. The Universities and the Licensing Corporations of Great Britain are full blown democratic institutions and as such their 27 representatives are the real representatives of the profession. Even so, the British Medical Profession has always been complaining against the smallness of direct representation provided in the Medical Act. More than the members who compose these Councils, it is their attitude that is important: the members of the G. M. C. are genuinely interested in the upkeep of the prestige and usefulness of the British medical profession, but our members are only interested in making themselves pleasant servants of the G. M. C. The profession, as a whole, would ever refuse to harbour this inferiority complex.

Let me contrast the standards between the G.M.C. and the I.M.C.

(1) The G.M.C. lays down university matriculation or entrance examination in the faculties of art or pure science as the minimum preliminary general qualification. The former, that is those who are not science matriculates, have to undergo examinations in physics, chemistry, and biology, while the latter, that is the science matriculates, have to undergo examinations in one or two subjects of general education other than physics, chemistry or biology.

(2) Excluding the pre-registration course, no medical qualification can be obtained in less than 5 years. Usually the course extends to 5½ to 6 years, particularly the examination for the university degrees.

The Indian Medical Council lays down: (1) The Intermediate Examination in the Medical group of a British Indian University which includes a practical test.

(2) The Intermediate Examination in Arts or Science of a British Indian University with Physics, Chemistry and Biology and which shall include a practical test in each of these subjects.

(3) B. Sc. Examination in chemistry of a British Indian University if Physics and Biology have been taken in the Intermediate Examination with a practical test in those subjects.

(4) The Intermediate examination held by the following Boards of High School and Intermediate Education with Physics, Chemistry and Biology which includes a practical test in each of these subjects:—(a) Rajputana, Central India and Gwalior. (b) United Provinces.

(5) Any other examination which in scope and standard is found equivalent to the intermediate Science Examination of an Indian University tak-

ing Physics, Chemistry and Biology including a practical test in each of these subjects.

(6) No medical qualification can be obtained in less than 5 years.

Is it not strange that while in Great Britain matriculation is deemed sufficient for admission to a medical course, it is held to be inadequate for our country?

The reason is obvious: In no other way could the medical schools and the licentiates of these schools could be eliminated from the schedule of medical colleges and qualifications recognised by the Indian Medical Council; in no other way could the G.M.C. hold sway over matters medical in our country without excluding the major part of the profession. Thus it has come to pass that 29 medical schools and nearly 80,000 statutorily qualified medical men have been branded as of a sub-standard quality, merely to please the tin-gods of the General Medical Council. In fact, the standards of medical and of general education, as now prevalent in the medical schools of our country, are by no means inferior to the standards laid down by the G.M.C. for home consumption, although the writer would freely admit the necessity for improving the school standard which could certainly be effected without condemning the whole structure built assiduously during the last 150 years.

It is not necessary for the purpose of this article to go into the details of medical curriculum. It is of the same model as in Great Britain—be it a medical school or a medical college. But there is always this question which has not yet been satisfactorily answered: should the curriculum be so long, so tedious and so hard? Could not the non-essential subjects be reduced to barest minimum. The readers may find in this issue a letter from 'our German correspondent' in which he writes that with effect from April 1939, Germany has introduced a shorter medical curriculum in all her universities. Even in England, there is a loud complaint against the over-crowded and prolonged medical curriculum from persons who are not professional agitators but have a long experience in imparting medical education. It is therefore as plain as crystal that mere lengthening of the course and adding an interminable burden of new and newer subjects are not the yard-sticks of efficient medical education.

#### A MODEL MEDICAL COUNCIL OF INDIA

In devising a model medical council for our country, one cannot but take into consideration

(1) its form of government;  
(2) its extent;  
(3) the present state of medical education. But it is not necessary to go into these details at any length except to state categorically:

(4) that since our future Central Government is bound to be a federation of autonomous provin-

ces and Indian States, the model medical council should also be federal in type;

(5) that its basic constituencies should be the Provincial Medical Councils, re-inforced by representations from the Universities, State Medical Faculties and the Federal Assembly;

(6) that while it should assume control of minimum and higher medical qualifications, it must forthwith recognise the existing qualifications admitted as such by the prevalent provincial statutes; and that if any such qualification lacks the minimum standard, time for improvement should be allowed before it is denied the recognition.

Other factors that should be given equal considerations are:

(7) The model medical council should keep the whole of our country under its purview, and not divide it into British India and Indian States.

(8) It should take note that our country needs more doctors and that quality should be combined with quantity.

(9) It should try to improve the medical educational standards, not by intimidation but by courteous negotiations with the local authorities.

(10) It should not go abegging for reciprocal recognition from other countries, and it should boldly retaliate when circumstances demand such an attitude.

Such should be the background in the construction of a model medical council for our country. The filling up of the details are secondary and can be adjusted from time to time as per the exigencies of circumstances. A rough outline of a model council is given below taking into account our present conditions but it should be mentioned at the outset that it does not claim to be a perfect model and it is certainly subject to the opinion of the organised profession and the statesmen of our country:

(1) It should be enacted by the federal legislature.

(2) Its functions should be:

(a) the establishment of minimum standard of medical qualification, and control and guidance of higher medical qualifications;

(b) compilation of All-India Medical Register on the basis of Provincial Medical Registers;

(c) the hearing of appeals against the disciplinary action taken by the Provincial Medical Councils;

(d) compilation and publication of an Indian Pharmacopœia.

(3) Its composition should be:

(a) two members elected by each Provincial Medical Council;

(b) one member from each of the Provincial Universities with Medical Faculties and of the State Medical Faculties;

(c) one judge of the Federal Court nominated by the Federal Cabinet;

(d) six members to be nominated by the Provincial Governments and Indian States after mutual consultation;

(e) one member nominated by the Federal Assembly after election from among its members.

(4) Its constitution should be:

(a) A President, Vice-President and Secretary elected from among the members for a term of five years.

(b) An executive committee of not more than 15 members elected by the Council for a term of five years.

(c) This executive committee should elect a panel of 4 medical examiners, out of whom two should be sent as such and with whom two other members elected by a Provincial Medical Council should be co-opted within whose jurisdiction the medical examiners have to examine and report.

(d) This executive committee shall prepare by-laws, regulations and recommendations on professional education subject to the approval of the general body of the Council.

(e) The Council and its executive committee should meet at least twice in a year.

(f) Expenditure on the maintenance of the Council shall be met with by equal contribution from all the Provincial Governments, Indian States and the Federal Government.

(5) Relationship with Provincial Medical Councils:

(a) The Medical Council of India shall be the supreme authority on the standards of medical education in our country.

(b) The Medical Council of India shall compile an All-India Medical Register based on the Provincial Medical Registers.

(c) The Provincial Medical Councils shall have the right and exercise the duty of inspecting the medical examinations and courses within their areas, and report the same to the Medical Council of India through the Provincial Governments or Indian States.

(d) The Provincial Medical Councils shall take cognizance of penal cases within their respective areas, subject to the appeal of the All-India Council.

#### BUILDING CASTLES IN THE AIR

The writer realises that the above scheme is like building castles in the air. The circumstances are indeed not propitious for this purpose: we lack the cement, the mortar and the workmen to build it. Others may even laugh at the audacity of the imagination which conceives such a scheme. Still others there will be who honestly believe that the present Medical Council is not after all such a bad affair as is made out in this thesis. There is no doubt that such thoughts lack vision and courage. The profession must, somehow or other, wriggle out of the dreary and drudgery existence which is its lot to-day and must not be poisoned by the slough of despond. This may be considered a foolish scheme to-day but it will prove to be the wisest and the best for a country set for high destiny. No scheme other than the kind suggested will be welcome to a free India.

## The British Medical Association Conference

### By a London Doctor\*

Aberdeen is no mean city. This is the verdict of representative doctors from all parts of the Empire, now in conference at the annual meeting of the British Medical Association in Scotland's Granite city.

The conference has faced a wide variety of problems. First came an international question: should the B. M. A. end its connection with an international body on the grounds that the totalitarian members of this body were using it less as a medium for exchanging views and more as a platform for propaganda. The conference said No: this was not the moment, it said, to extinguish any flicker of internationalism, however dimly the flame may burn.

Then came refugee doctors. The conference approved the admission of fifty refugee doctors from Czechoslovakia, to requalify and later to practise in this country. We could do no less, it was argued for Czechoslovakia, than we did a year ago for Austria. But, it will be noted, permission to practise is confined to this country.

Then came a lead to industry. Industrial accidents cost this country no less than fifty million pounds a year in money alone, and much more in terms of human happiness. Much of this can be saved if the treatment given immediately after an accident is the right treatment.

The conference approved a new code for the training of first aid workers in factories. In it are described courses of first aid training for workers in factories of different sizes and of different kinds—large ones, small ones; iron works, chemical works, and so on. The code aims at both imparting first aid knowledge and at checking excessive zeal: it is first aid manual adapted to the varying needs of industry. To a wage earner, a stiff hand may be a more serious disability than the loss of a leg, and if this report raises the standard of first aid work in factories and workshops, it will be the saving of both money and misery.

A special session was devoted to Empire matter: should the Government be urged to spend more money on the control of leprosy? The general view of the Empire representatives was that leprosy should be considered as part of a general health policy, and not singled out for treatment as an individual disease. It was a nutritional as well as a pathological problem.

From Australia, New Zealand, Jamaica, West Indies, South Africa, India and Ceylon, came greetings to the parent body. We learnt of the nutritional research in India, of National Health Insurance in Australia and New Zealand, and of success in the eradication of malaria in Ceylon.

The representative of Assam sounded a practical note when he urged that the council should remind theatrical companies and concert parties touring in the east, to take the necessary precautions against such diseases as typhoid and cholera.

Then there were some old friends. Should milk be pasturised? Again the conference pronounced an over-

(Continued at foot of next page)

\* A summary of a recent broadcast by the British Broadcasting Corporation in their Empire programme—Exclusive for the *Indian Medical Journal* in India.

## L.P.H. Officers' Association, U. P. A deputation to the Hon'ble Minister of Health

[The following memorandum reveals the miserable plight of the L. P. H. Officers in the United Provinces, even under a popular Government. While the old grievances are unremedied, new grievances are being added. Of the latter, the abolition of 20 posts of Assistant Health Officers and putting the present incumbents on reserve epidemic duty is one for which there is little justification. That it is not an act of economy or of a pressing need for readjustment is obvious. Even public opinion seems to be against this move. We wonder why, then, a popular Ministry should initiate such changes and incur a reputation for being hard on a class of devoted public servants. May we appeal to the Minister of Health to undo this injustice and see that she is not guided by vested interests in matters of this nature.

—Ed., I. M. J.]

The Secretary of the Association presented the following memorandum.

We, on behalf of the U. P., L. P. H. Health Officers' Association, deem it our proud privilege to have the honour of waiting in deputation on you in order to seek redress of an urgent grievance of the members of this class.

At the outset, we beg to offer you our respectful and sincere thanks for having acceded to our humble request to receive this deputation, notwithstanding the numerous pressing calls on your valuable time. We earnestly hope that the question of our grievances, though minor compared to the higher national problems before you, will receive your adequate attention and sympathy and the anomalies affecting the members of our class would be set right in your auspicious regime.

Madam, we will not encroach upon your time by giving a catalogue of our demands which are long overdue but we have now come with a single grievance only. The recent orders of the Government abolishing the post of 20 Assistant Health Officers and placing them on Epidemic Reserve Duty have injured us the most and made us feel like fish out of water. These officers had risen by promotion after working for several years in the Travelling Dispensaries where they spent all their youth with untiring energy in serving the masses living in the remotest parts of the rural areas. In this capacity they were shifted from place to place and district to district like a shuttle-cock. It is really an irony of fate that they have once again been put to unsettled life of Reserve Duty which is likely to last practically the whole length of their service since there is no hope of vacancies by retirement of the seniors who are nearly of the same age and service.

In this connection, Madam, allow us to submit that the consideration of economy which the Government meant by these alterations does not seem to have been

(10) Resolved that on the occasion of transfers of Medical licentiates, who are members of our Assn. a farewell party

be arranged without exception and only the medical licentiates members be invited to attend such farewell parties.

Further resolved that instead of raising special subscription for such farewell parties, the monthly subscription of the Association be raised from -/4/- to -/6/- per month, for meeting the expenses of the parties in addition to the usual expenses of the Branch. It was resolved that the cost of farewell parties should not as far as possible exceed Rs. 10/- per party.

A copy of the above resolution be sent to all the medical licentiates of the district for information and favour of communicating their transfers from the district immediately on the receipt of their transfers.

11. Dr. Dinesh Verma proposed that a clinical meeting be held every quarter in Civil Hospital, if possible, and the L. M. P. in charge of the Civil Hospital be requested to kindly arrange for such meetings.

Further resolved that the present sub-charge M. O. Dr. S. L. Dube be requested to sound the Civil Surgeon and to inform the members of this Branch in the next meeting. The meeting terminated with a vote of thanks to the chair.

R. S. Dr. Brij Mohan Lal Sahib was "At home" to the members of the Association after the meeting. The members enjoyed the refreshments nicely served and dispersed.

Proceedings of a general meeting of Fyzabad District Branch held on March 19, 1939 at the residence of Dr. B. P. Jain at 3 P. M.

The following members were present:—

1. Dr. B. P. Jain, 2. Dr. S. A. Qureshi, 3. Dr. I. N. Sexena, 4. Dr. Dinesh Verma, 5. Dr. Syed Ahmad, 6. Dr. S. L. Basi, 7. Dr. B. L. Sharma, 8. R. S. Dr. B. Brijmohan Lal, 9. Dr. P. J. Chandy, 10. Dr. A. N. Srivastava, 11. Dr. Basantlal. Dr. B. P. Jain in the absence of the President was voted to the chair.

1. The minutes of the last meeting were read and confirmed.

2. Account up to 31st January, 1939 with a balance of Rs 62-7-3 was put up and passed.

3. U. P. Prov. Secretary's circular letter to M. L. As was read and recorded.

4. General Secretary's circular No. 1. was read and recorded.

5. Resolution No. 2 of the standing committee U. P. was read. Dr. B. P. Jain, Dr. Syed Ahmad, Dr. S. L. Dube were requested to become members. It was resolved that Dr. Syed Ahmad be requested to enrol members from outlying places.

6. Report of the working of I. M. J. read and recorded. R. S. Dr. B. M. Lal, President of the Branch arrived at this stage and Dr. B. P. Jain vacated the Presidential Chair in his favour.

7. U. P. Prov. Secy's letter No. 976, dated June 30, 1939 regarding Agra Medical course was read and the suggestions from members were incorporated. The meeting dispersed with a vote of thanks to the chair. Dr. B. P. Jain had arranged light refreshment and tea in which all the members and friends of Dr. Jain had participated.

A Scientific meeting was held at the Leper Home and Hospital Zamureadganj at 5 P. M. on April 5, 1939 under the auspices of the Fyzabad District Branch of A. I. M. L. Association to which all the medical men were invited. Classification of leprosy and its treatment was discussed.

Mrs. and Dr. Chandy were at home to the guests at their residence after the meeting.

A general meeting of Fyzabad District Branch of A. I. M. L. A. was held on May 6, 1939 at the residence of Dr. Mahboob Ali, I. M. D.

Dr. Brij Mohan Lal, R. S. presided over the meeting.

1. Minutes of the last meeting were read and confirmed.

2. Accounts upto 31st March, 1939, were read and passed with a balance of Rs. 69-7-3.

3. Circular No. 15 of G. Secretary was read and resolved that a suitable reply be sent to Dr. J. J. Cursetji C. M. O. Oriental G. S. Insurance Co. to his letter published in the

Insurance World and a copy of the Association's reply be sent to all the insurance companies.

Further resolved that the A. I. M. L. Association should call upon all the licentiates to boycott this company by advising their friends not to have any policy and to get their previous policies in this company paid up.

It was resolved that the General Secretary of A. I. M. L. A. should obtain legal advice and if possible Dr. J. J. Cursetji should be sued for defamation.

4. G. Secy's Circular letter No. 17 was read and the resolution suggested therein was passed.

5. The Branch was informed at this stage of the sad and sudden death of R. S. Dr. Suraj Lal, the late C. M. O. Dhoulpur and it was resolved that this Association expresses its grief and sorrow at his sudden death and offers its condolence to the members of the bereaved family.

6. It was resolved that the monthly allowance of Re. 1/- paid to the two peons upto now for the Association work be raised to Rs 2/- per month from May, 1939.

7. The meeting terminated with a hearty vote of thanks to Dr. Mahboob Ali Sahib for his entertainment on a very lavish scale, which the members enjoyed thoroughly.

### THE ALL-INDIA MEDICAL LICENTIATES' ASSOCIATION

(U. P. PROVINCIAL BRANCH)

#### Appeal for Annual Provincial Contribution

The strength and life of an association is entirely dependant upon the sympathy of its members and its financial position. Specially at a moment when the very existence of our Class and Association is being threatened from all sides, it becomes the moral duty of our members to rise to the occasion and help it with all their mite. Although eight months of the year have passed, the annual contribution of Rs. 2/- per member has not yet been received with few exceptions. Last year the same thing had happened and at last the Treasurer was obliged to issue V. P. Cards to individual members to recover the provincial contribution.

This appeal is being issued to members of the Association and Licentiate brethren in the Province with the request that they will kindly send their Annual Contribution of Rs. 2/- for 1939 by postal money order to the address of Dr. Mannu Lal Sharma, Provincial Treasurer, A. I. M. L. A., Jattiwara Street, Meerut, at their earliest possible convenience and latest by 30th September, 1939, otherwise in the absence of any communication the Treasurer will be obliged to issue V. P. Cards for Rs. 2/4/- from 10th October, 39. It is hoped our brethren will honour the V. Ps. It may be added that in issuing V. Ps. double loss of postage is incurred; and hence, to avoid it, it is earnestly requested that you will help the cause of the Association by sending your contribution voluntarily for which we shall feel highly obliged.

KUNJ BEHARI LAL VERMA

L. M. P.

U. P. Provincial Secretary.

Meerut: 25 Aug. '39.

non-emetic and capable of oral administration.

#### SPECIFIC DRUGS

##### Chronic Amoebic Dysentery

It is safe and prudent to be convinced, at any rate for the time being, that there are no dependable specific drugs for the successful treatment of chronic amoebic dysentery. This by no means excludes the possibilities of a successful cure by the use of the available drugs. What the writer means to convey is that the destruction of the integrity, the tone, and the secretions of the intestinal mucosa and glands is so irreparably advanced that even were the patient rid of amoebic infection, he would still continue to suffer from the physical and functional effects of such destruction. The degree of therapeutic effect in the treatment of chronic amoebic dysentery depends, therefore, upon the degree and extent of the prevailing tissue destruction. If the destruction is repairable by rest, diet and treatment, we can claim success; if not we have to face failure. In fact, the essence of treatment in chronic amoebic dysentery lies in attempting to heal the intestinal ulceration, and only secondarily at the riddance of amoebic cysts.

**Yatren**, from this point of view, is definitely the drug of choice. It is an oxy-quinoline derivative prepared by Bayer's Laboratories with 25% of sodi-bicarb to render it water-soluble. It can be given (1) per os (2) per rectum (3) by combining both the methods. The particular method to be employed depends upon the site of the amoebic ulcers. If they are in the upper part of the large intestines oral method would suffice, if in the lower part rectal instillation is indicated, and if the whole of the large intestines is affected both the routes have to be adopted. The programme of yatren treatment is as follows:—

| Oral Yatren |         |        |            |
|-------------|---------|--------|------------|
|             | 1 pill  | T.D.S. | After food |
| First day   | 1 pill  | "      | "          |
| Second "    | 2 pills | "      | "          |
| Third "     | 2 "     | "      | "          |
| Fourth "    | 2 "     | "      | "          |
| Fifth "     | 2 "     | "      | "          |
| Sixth "     | 3 "     | "      | "          |
| Seventh "   | 3 "     | "      | "          |

Then on alternate days for three weeks. Each pill contains 4 gr. of yatren. (After Muhlens).

The only disadvantage of oral yatren is that it provokes a diarrhoea which adds to the discomfort of the already exhausted patient, and this is due to the idiosyncrasy to iodine contained in yatren. But this diarrhoea is bene-

ficial, and should there be a necessity to check it Muhlens advises the addition of small doses of Tr. of opii. He however does not advocate preliminary emetine.

##### Rectal Yatren

|            | 1 gm. | in | 200 c. c. of water |
|------------|-------|----|--------------------|
| First day  | 1 gm. | "  | "                  |
| Second day | 3 "   | "  | 300 "              |
| Third day  | 3 "   | "  | 400 "              |
| Fourth day | 3 "   | "  | 500 "              |
| Fifth day  | 3 "   | "  | 600-800 "          |

Previous cleansing of the bowel by high wash-out enema is necessary, but in view of the weakened intestinal wall and the possibility of perforation it should be done gently and with greatest care. Yatren by rectum is best given in the evening and it must be retained for at least 6 hours to produce any good results. Failure is usually due to high concentration of the yatren solution. It must not be boiled but dissolved in water at 80 degrees centigrade.

**Emetine Bismuth Iodide** was introduced in 1891, and Dale used it in the treatment of amoebiasis in 1916, but it has never been popular because very few patients tolerate it. The nausea and vomiting it produces is sickening. But its use cannot be avoided as this and other allied preparations are the only drugs available which have got some power, if often unsuccessful, to eradicate the encysted amoeba which are the usual forms met with in carriers and chronic cases. To reduce, therefore, the nausea and vomiting it is given preferably in powder or cachet form\* (2 grs. each) in empty stomach at bed time. Acton and Knowles<sup>1</sup> advocate 1/6th gr. omnopon half an hour before retiring to bed, and to administer E. B. I. (2 grs.) suspended in two drachms of liquid paraffin. The course is repeated everyday for at least 10 days, and the stools examined at the end of the period. Most of the chronic cases may be suffering from coincidental infection due to hæmolytic streptococci, not certainly an unexpected complication if we remember any pathological specimen of a semi-gangrenous gut of chronic dysenteries. In which case, an autogenous vaccine should be administered without any delay. Many cases require at least two courses of E. B. I., combined with autogenous streptococcal vaccine.

**Kurchi Bismuth Iodide** is less toxic and nauseating than E. B. I., and may be preferred in cases where there is an idiosyncrasy for the latter.

\*Acton and Knowles believe that compressed tablets are useless as they are nearly always passed unchanged per rectum.

Emetine periodide is better tolerated but less effective.<sup>7</sup>

Auremetine produces no nausea, vomiting, purgation or depression but the writer has no experience of this drug.

Stovarsol is a stable arsenical compound prepared by the House of May & Baker, and contains 27.2 per cent of arsenic in organic combination. Given in the dosage recommended by manufacturers, its toxicity is low but its effect on cystic amoeba is uncertain. The writer would certainly not advocate it for acute cases of amoebic dysentery but has often used it in chronic cases with definite advantage though its amoebicidal action has not been found to be remarkable. The good it does is probably by its toxic action and thus enhancing the patient's power of resistance. The adult dose is 4 grs. per day, and one must be on the lookout for arsenical poisoning.

Carbarsone is another arsenic compound, and is administered in the same manner and with similar effects like Stovarsol.

Ambiarson is another arsenic compound prepared in our country by the laboratories of the Bengal Chemical & Pharmaceuticals. It has been warmly advocated in the treatment of chronic intestinal amoebiasis by Chopra and Sen<sup>4</sup> and in the writer's opinion it is in no way inferior to Stovarsol or Carbarsone.

#### DRUGS IN THE TREATMENT OF ACUTE AND CHRONIC BACILLARY DYSENTERIES

There are no drugs which are known to act on the organisms of bacillary dysentery in the same manner as emetine or kurchi in amoebic dysentery. The few drugs that are used are mainly directed towards the elimination of bacteria and their toxins by means of purgatives and the alleviation of dysenteric symptoms by means of demulcents. In very mild cases these measures are quite sufficient, but in grave cases of acute bacillary dysentery no time should be lost in the administration of potent polyvalent specific bacteriophages or sera. They should be administered in sufficiently large doses and at the earliest possible time. If so administered, its effects are almost magical, but it would be a folly to leave the case as cured when the urgent symptoms abate after the preliminary serum or bacteriophage. Left in that manner, chronicity is certain, and a chronic bacillary dysentery is indeed a bug-bear of the physician. It is therefore imperative that when the acute

symptoms subside after the bacteriophage or serum treatment the stools should be examined, and if positive autogenous vaccine should be prepared and a full course given till the stools become negative. As in the chronic amoebic dysentery, so in the chronic bacillary dysentery the main line of treatment should be directed towards the healing of the intestinal lesions by the rectal administration of antiseptics. A weak solution of silver nitrate or rivanol 1 in 5000, may be employed for this purpose. Even normal saline or a weak boric or permanganate lotion is equally effective. Besides this, preferably an autogenous vaccine should also be administered.

#### SUMMARY

(1) Methods of approach in the treatment of dysenteries should be clearly formulated in the physician's mind before he rushes to choose an anti-dysenteric drug.

(2) Mild purgatives should form an essential preliminary course in any form of dysentery except in cases which are seen in the stage of extreme dehydration and collapse.

(3) Astringents should be strictly avoided, and if indicated they should be given with or alongside the purgative mixtures.

(4) Demulcents can be freely given in any type or stage of dysentery: they give rest to the ulcers, adsorb bacteria and toxin, and alleviate the excruciating symptoms of tenesmus and griping.

(5) Emetine or kurchi extract is the drug of choice in acute amoebic dysentery. Toxic effects of emetine should always be borne in mind.

(6) Yatren, emetine or kurchi-bismuth iodide and its other preparations; stovarsol and other allied arsenical compounds, are valuable drugs in treatment of chronic intestinal amoebiasis, but the main purpose of treatment should be to restore, if possible, the physical and physiological integrity of the colon already in a state of chronic ulceration.

(7) Potent specific polyvalent bacteriophage or serum should be given early and in massive doses in all cases of seriously acute bacillary dysentery.

(8) In mildly acute cases, a regular saline treatment will do immense good and relieve all the symptoms.

(9) It is highly essential that, in all cases and types of acute bacillary dysentery, a course of autogenous vaccine, prepared from the stool, should be given after the preliminary treatment, lest they become chronic and difficult to deal with later on.

(Continued at foot of next page)

## Amoebic Hepatitis & Liver Abscess

[Hepatic Amoebiasis or Suppurative Amoebic Hepatitis or Tropical Liver Abscess]

• Dr. K. Narayana Murthi, M.D., Research Worker, and Honorary Physician, General Hospital, Madras

The most frequent and dangerous complication of intestinal amoebiasis is the infection of the liver, which goes on to the formation of an abscess, unless prompt treatment is given. Sometime ago, the commonest cause of death in the British army in India, next to typhoid, was liver abscess. For every 8 cases of intestinal amoebiasis one case was found to develop liver abscess. Now the abscess formation is easily preventable by instituting treatment in good time, and by so doing the mortality rate which was about 50 to 60% in the army had been reduced to 2 to 4%. Statistics regarding the incidence of the disease in the civil population in India is not available. The author found, on an analysis of 1265 cases of dysentery treated at the Govt. General Hospital, Madras, during the years 1928—1931, amoebic infection of the intestinal tract in 375 cases, out of which not even a single case was found to develop abscess in the liver. Woodward's analysis of 3,680 cases of post-mortem reports of dysentery collected from various tropical countries revealed 779 cases (21%) of abscess of the liver.

**History.**—The etiology of the tropical abscess of the liver remained unsettled till as late as 1902. Duncan with long Indian experience was under the impression that small multiple liver abscesses were related to dysentery and that large single tropical form was not. Up till then, amoebic dysentery was thought to be unknown in India, and particularly the tropical abscess was admitted to be a distinct disease. The bacillary dysentery was only beginning to be recognised as a specific disease. It was Rogers working in Calcutta proved that liver abscess is due to amoebic infection. In a large series of his cases, clinical and post-mortem, evidence of dysentery could be had in 90% of the cases. Besides this, the abscess was found entirely due to the amoebic infection. It must be remembered that extreme amoebic ulceration may exist without any subjective symptoms whatsoever. Many patients suffering from liver abscess forget or fail to mention that they had suffered from dysentery. It is likely in those cases that they might have had a mild attack of amoebic dysentery with symptoms of simple diarrhoea. In the great majority of the cases the dysentery antedates the abscess. Low cited a case of an abscess in the liver occurring after 20 years of the first attack of dysentery.

Europeans suffer more from liver abscess as compared with the Indians. The relative liability

of European soldier and the Indian soldier was 95.2 to 4.8, in spite of the fact that a large proportion of the Indians were infected with *Entamoeba histolytica*. This is probably due to a congested liver brought about by alcohol. It is of interest to note that the liver abscess is found to occur in abstainers also. On an analysis of nearly 400 cases of liver abscess in Calcutta 70% occurred between the ages of 21 to 40 years, about 5% below 20 years and the same proportion above 50 years. The disease is extremely rare below the age of 10 years. An Egyptian child of 3 months old was reported to have suffered from an amoebic abscess of liver. Manson-Bahr recorded a case of an English girl of 16 years suffering from liver abscess.

The extreme rarity of liver abscess in the temperate zone is due to the infrequent occurrence of amoebic dysentery. Bacillary dysentery however is common but it does not result in liver abscess. Even in India, though a tropical country, the incidence of bacillary dysentery is relatively much greater than amoebic dysentery.

**Pre-suppurative stage of Amoebic Hepatitis.**—Two decades ago patients suffering from liver abscess gave invariably history of having suffered from fever of an obscure nature for months together for which they were treated with quinine. If hepatitis was suspected ammonium chloride was given by mouth. The liver abscess was discovered by aspiration alone or by bursting of the abscess into the peritoneum with a fatal result. A correct diagnosis could be made now by a leucocytic count in such of those obscure cases. In the acute stage of amoebic hepatitis the patient will have high fever

(Continued from previous page)

(10) The main principle in the treatment of chronic bacillary dysentery is the healing of the intestinal ulcers. Hence rectal instillation of weak antiseptics is called for, while autogenous vaccine should in no case be omitted.

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enlarged and tender liver and there may or may not be history of recent attack of dysentery or diarrhoea. The temperature is of an irregular remittent type with profuse perspiration. The enlargement of the liver may be upwards producing difficulty of breathing or it might enlarge below the right costal margin. X-ray will reveal an elevated right dome of the diaphragm; palpation of the abdomen is likely to reveal some tenderness and thickening over the caecum and ascending colon, and occasionally over the large bowel indicating the presence of amoebic ulceration.

In the most acute type it is impossible to tell on clinical grounds whether multiple small liver abscess is present or not. As the treatment in either case is the same this is of no consequence.

In the chronic form, the enlargement of the liver is generally present. Repeated examination of the stool for *E. histolytica* should be made in all forms of hepatitis, as a positive result will be of great value. Tenderness may be absent over the liver area. The insidious cases are very likely to be overlooked until the liver abscess is developed. The chronic cases shade off into more easily recognised sub-acute ones with definite hepatic symptoms. In cases of doubt there need be no hesitation in giving emetine as a diagnostic test as there are many instances on record in which an obscure fever with leucocytosis promptly yield to emetine after having resisted all other forms of treatment for months.

The pre-suppurative stage is found to last 2 weeks in 84 per cent of the cases. Thus there is ample time to detect the disease in the pre-suppurative stage, and the abscess of the liver is easily preventable if the patients come early for treatment.

Unexplained fever with a leucocytosis should be suspected to be due to amoebiasis even in the absence of history of dysentery and of amoeba in the stool.

**Treatment.**—The treatment of amoebic hepatitis is simple and satisfactory, especially since the introduction of emetine. One grain of emetine daily given intramuscularly upto 9 grs. is sufficient to bring down the temperature to normal within a week, and the enlargement of the liver and leucocytosis will subside within almost the same time. To avoid the danger of relapse full doses of ipecacuanha may be given orally for another week to eliminate infection from the bowel.

In a case of acute hepatitis aspiration should on no account be performed until a course of emetine injections are given. It is amazing to note the pain, tenderness and fever are relieved within 3 or 4 days under this treatment. In chronic cases recovery is quite satisfactory but it is less dramatic. Alcohol should be avoided. The convalescence is often surprisingly rapid. The thickening of the large bowel due to ulcers also disappears during the treatment. Saline aperients, rest, low diet, fomentations, dry-cupping and similar measures avert a very grave complication as the formation of an abscess. Harley considers removal of a few ounces of blood by the aspirator needle

as a measure of proved value in hepatitis. But these is no unanimity of opinion.

**Liver abscess.**—This occurs in a patient who comes late for treatment. It is always preventable if a correct diagnosis has been made and the specific treatment is carried out in the early stages. The amoebic abscess is mainly due to *E. histolytica* gaining entry into the radicals of the portal system from the infected sub-mucous coat of the large bowel and passing to the liver as an embolic infection. The fact of the infective focus occurring in the caecum or ascending colon accounts for the occurrence of the abscess in the right hepatic lobe. Direct infection of the anterior surface of the liver may possibly sometimes take place from an ulcer in the hepatic flexure *via* the peritoneal cavity by contiguity. But this by no means constitutes the usual method. In the amoebic hepatitis which is usually a precursor of amoebic abscess, there is a massive portal infection with amoeba or what is otherwise known as portal pyaemia. The great majority of amoebae are destroyed by the tissues and those that survive multiply and cause severe necrosis of the surrounding liver cells which is a starting point of a liver abscess. Some of the amoebae are destroyed by the products of their own activity resulting in sterile pus. Sometimes it is likely that pus gets infected with pyogenic organisms.

**Pathology.**—There is general congestion and enlargement of liver confined to one lobe or to even part of a lobe. Ill-defined anaemic circular patches may be found, destroying the lobular structure of the gland. These anaemic patches contain a reddish gummy pus. When this is squeezed out a ragged abscess cavity may be seen. As the abscess enlarges two or more contiguous cavities might form into a single big abscess cavity. During evolution, the pus in the abscess cavity is reinforced by secondary organisms like streptococci and staphylococci, and thus becomes brownish or greenish in colour.

The naked eye appearance of the pus is pathognomonic. It is chocolate coloured, often streaked or mixed with of blood. Here and there streaks of clear mucoid or yellowish material may be found. It is so thick that it can hardly ever soak the dressings. The pus might be contaminated with bile giving a green tinge to it. It is rarely offensive but if the abscess is very near the colon, the pus might emit faecal odour. Under the microscope the pus reveals blood corpuscles, degenerated liver tissue, oil globules, hamatoidin crystals and occasionally Charcot-Leyden crystals, entamoeba, streptococci, staphylococci, and *B. coli*.

The writer's experience of tropical abscess is that entamoeba is rarely found in aspirated liver pus; since they firmly stick to the wall of the abscess cavity. After the abscess is incised and the matter escapes, the entamoebae are found in great numbers on the 4th or 5th day sticking to the drainage tube. This suggests that the amoeba does not live in the pus but lives in the tissue of the liver damaging the liver substance. The longer the abscess persists the larger will be the size. In

a larger proportion of cases the pus is sterile, but we do come across some secondary organisms occasionally. The cysts of the *E. histolytica* are never found in the abscess of the liver. In some instances the pus does not possess the characteristic chocolate colour. It is yellowish or creamy. This feature is due to the abscess becoming encysted, which is a rare phenomenon. The walls of the cyst are thick, smooth and often fibrous. Sometimes the pus becomes coagified and the cyst shrinks and contracts to a very small size.

Rogers considers that cirrhosis of the liver in India is a sequel to liver abscess. But there is no unanimity of opinion on this finding.

An abscess in the left lobe of the liver is variable in outline, shape, and position, and so it may simulate an enlarged spleen. Few crepitations may be found at the left base of the lung which might sometimes be due to compensatory hypertrophy of the undamaged right lobe of the liver without any abscess formation.

As the case progresses the patient is emaciated with drenching nocturnal sweats. The abscess may point downwards. If the pus from the abscess cavity is not drained, the patient may die of exhaustion. The abscess may burst into the right lung or pleura or end in recovery or death from continued hectic fever, or some inter-current complications. With a liver abscess of long duration, usually there may be seen secondary anaemia and the blood changes may resemble that of pernicious type of anaemia.

It is not unusual that an unsuspected liver abscess is discovered on the post-mortem table. The subjective symptoms are entirely absent. If the abscess is undiscovered, it may rupture into any contiguous organ and thereby produce a spontaneous cure. If it ruptures into the lung the abscess contents may be suddenly coughed up in mouthfuls of frothy pus and blood, and in favourable cases the expectoration gradually becomes diminished. Amoebic abscess bursting through the diaphragm into the lung is liable to be mistaken for pulmonary tuberculosis. In the expectorated pus amoebae are not found, and the only microscopical sign that might be available is the existence of striated muscle fibres from the diaphragm.

Arrest of the discharge or shrinkage of the tumour may not mean recovery, as the cavity of the abscess may get refilled with pus many times before recovery finally takes place. In some cases the expectoration never ceases and causes respiratory distress, temperature and night sweats. Clubbing of the fingers may be noticed in such of those cases. Rupture into the pleura may lead to effusion, which gives all the characteristic signs of empyema. Aspiration above the line of the pus in these cases yield a clear yellow fluid.

Hepatic abscess may rupture into the stomach, causing vomiting of pus, or into the bowel causing diarrhoea and discharge of pus in the stool or it may burst into the peritoneum or into the pericardium with fatal results. Spontaneous rupture of the abscess may take place through the skin of the abdominal wall. This is the most

favourable natural cure of the liver abscess. The skin itself may secondarily be infected with amoebae. Rajan recently demonstrated in our hospital an amoebic ulcer of the skin around the rectum and groin simulating an infective granuloma.

The following table is reproduced from Manson-Bahr, which may interest the readers:—

|                                |     |    |
|--------------------------------|-----|----|
| History of previous dysentery  | ... | 60 |
| <i>E. histolytica</i> or cysts | ... | 45 |
| Shoulder pain—right            | ... | 44 |
| do do left                     | ... | 44 |
| Pyrexia, intermittent          | ... | 77 |
| do remittent-typhoid like      | ... | 8  |
| Sudden onset, rigors           | ... | 4  |
| Pyrexia, absent                | ... | 13 |
| Night sweats                   | ... | 82 |

#### DYSENTERIC INFILTRATION OF THE BOWEL

|                                        |     |    |
|----------------------------------------|-----|----|
| Signoid fluxure                        | ... | 24 |
| Caecum and sigmoid                     | ... | 8  |
| Enlargement of liver upwards           | ... | 24 |
| do do downwards                        | ... | 75 |
| Pain and tenderness in liver           | ... | 57 |
| Displacement of heart                  | ... | 8  |
| Associated signs at base of right lung | ... | 75 |
| Pleuritic rub                          | ... | 12 |
| Rupture of abscess into the lung       | ... | 11 |

**Mortality.**—The mortality rate of 50 to 60 % is due to the now obsolete open operation which allows secondary septic infection to take place, Thurston with a personal experience of 164 cases concluded that "the open operation is to all intents and purposes obsolete except under unusual conditions".

**Diagnosis.**—The most common mistakes that are made in the diagnosis of hepatic abscess are:— (1) The failure to recognise the occurrence of amoebic dysentery in the individual. (2) Misinterpretation of a basal pneumonia on the right side. (3) Prolonged hectic fever mistaken for malaria. (4) Suppurative disease of the liver, pyelophlebitis, suppurating hydatid cyst, inflammation of the gall bladder, sub-phrenic abscess due to ruptured gastric or duodenal ulcer or appendicular abscess of the abdominal or thoracic wall, pleurisy, encysted empyema, pyelitis of the right kidney, ulcerative endo-carditis, kala-azar, splenomedullary leukemia, undulant fever, tuberculosis and any malignant disease. Any of these may be attended with hepatic type of temperature associated with the enlargement of the liver, pain, and tenderness in or about that region. X-ray examination may help in the diagnosis. If the abscess is situated in the centre of the liver, even if it be of a considerable size, no definite information is obtainable by radiogram except perhaps when the abscess is encysted. Aspiration with a needle gives the correct diagnosis.

**Prognosis.**—Favourable prognosis depends on early treatment with emetine and aspiration. If there is no dysentery or other complications and if there is single abscess the prognosis is favourable. In multiple abscesses or in a single abscess with acute symptoms of dysentery or other serious complications the prognosis is bad. If the abscesses are more than two the case is usually hopeless. It is

important to remember that the amoebic infection of the bowel needs thorough treatment, otherwise a recurrence of the liver abscess after a period of as long as 7 years from the formation of the first abscess is likely to occur.

**Treatment:**—Aspiration and giving injections of emetine are the methods of choice in the treatment of liver abscess. If the abscess is solitary, as in the majority it is so, the pus may be evacuated by means of a Potain's aspirator. The evidence at present available points out that this procedure has reduced the mortality rate to about 12 per cent. It must be remembered that hepatic abscess contains rarely half a pint of pus in the cavity, but there may be as much as 6 pints. The absorption of the pus throws considerable strain on the patient's constitution and delay his recovery, if it is not aspirated. Nevertheless it has been found that under emetine treatment one is no longer justified to do the dangerous open operation. If the formation of abscess is diagnosed, several punctures with a thick needle may be necessary before the cavity is struck, and in this case an anaesthetic will be required. Chloroform should be avoided if possible. No drug need be injected into the abscess cavity as we have found that cases with quinine injected into the cavity do just as well as those cases without it. The essential point to be remembered is to give emetine. We should under no circumstances give up the attempt of aspiration unless we make at least six punctures to find out the pus. It should be remembered that the needle should not be thrust for more than  $3\frac{1}{2}$  inches for fear of wounding a large vessel. It is very difficult and often very dangerous to puncture the left lobe and for this reason some surgeons prefer to make an incision under strict asepsis. In these cases, since the abscess cavity is small, the risk of post-operative sepsis is less. If the fever persists and the cavity fills up again aspiration may have to be repeated after a week. As a rule, the pus found at the second aspiration will be considerably less than the first aspiration. Thurston found that a single aspiration was sufficient for 26 cases, two in 16 cases, three in 10 cases, and four, seven and eight respectively in the remaining three cases. The surgical axiom "where there is pus there should be an incision" should not be accepted readily in the case of liver abscess.

The open operations for liver abscess done through trans-peritoneal route, trans-pleural route etc., have all become obsolete nowadays.

It should always be remembered that emetine injections act on *E. histolytica* effectively but after aspiration and emetine injection it is further advisable to clear up the infection in the bowels with 20 to 30 grains of ipecacuanha or with emetine bismuth iodide given internally so as to prevent relapses. In cases of abscess discharging pus through the lungs or not progressing favourably surgical drainage has to be considered. Otherwise, the pus in the lung may induce pneumonia or even thrombosis resulting in the abscess of the lung or of the brain. The indication for

### Bacteriophage therapy in Bacillary Dysentery

• Dr. S. P. De, M. B., Bengal Immunity Research Laboratory, Calcutta

According to the report of the Public Health Commissioner with the Government of India, in 1936, the death rate due to dysenteries and diarrhoeas was 281,666 and this was higher than that due to cholera in the same year. If this be the mortality figure, how many people must be suffering from this disease in our country! It will not be an exaggeration if we suggest that every year several millions suffer from dysentery in India.

It is important therefore that effort should be made to check the high incidence and death rate of this disease.

Dysentery is a condition where the principal symptoms are frequent passage of mucus or blood or both with stools, tenesmus, malaise or fever and in graver cases, toxæmia with its associated symptoms.

These are usually the symptoms met with in the acute cases. There are however sub-acute and chronic conditions where the principal symptoms are usually occasional passage of mucus or blood with stool, indigestion, malnutrition, lethargy, etc.

The etiology of dysentery as met with in general practice is due mainly to two factors: *viz.*,

- (1) Bacillary infection
- (2) Infection with *Eutima*, *histolytica* or other protozoa.

In this paper we will take up the treatment of bacillary dysentery only.

The organisms usually responsible for this condition are numerous, although *B. Shiga* and *B. Flexner* are usually believed to be the commoner ones. It should however be remembered that

performing an open operation depends upon the temperature, the character of expectoration and loss of weight. If the temperature remains normal the pus decreases in quantity and if the body weight is maintained open operation may be deferred with safety.

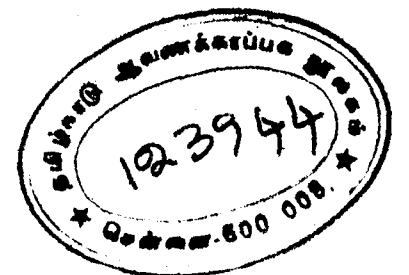
Ipecacuanha and emetine really exert a miraculous effect upon these cases and so render the operation inadvisable.

Treatment of abscess rupturing into any particular serous cavity requires of course an operation immediately, otherwise the patient may die. Under these circumstances the surgeon is justified to undertake this great risk.

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**Treatment of Intestinal Troubles by Specific Bacteriophages**

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**Bi-Dysentery-phage**—A mixture of various races of highly virulent dysentery and para-dysentery phages isolated in India from dysentery cases. Best results are obtained if administered within first 48 hours of the disease. It can also be used as prophylaxis. The method of administration is similar to that of Bi-Cholera-phage.

**Bi-Dysentery-Cholera-phage** (Combined)—A mixture of various races of highly virulent bacteriophage isolated in India from bacillary dysentery and cholera cases. It is useful alike in bacillary dysentery and cholera.

**Bi-Intestiphage**—A mixture of the various races of highly virulent bacteriophages isolated in India from cases of intestinal infection, such as dysentery, para-dysentery, cholera, typhoid, paratyphoid, coli, para-coli, proteus, pyocyanous, enterococcus, streptococcus faecalis etc. Indicated in all cases of summer diarrhoea, acute dysentery, diarrhoea, puerperal diarrhoea, sprue, etc.

**Bi-Typhoid-phage**—A mixture of various races of highly virulent typhoid and para-typhoid phages isolated in India from typhoid and para-typhoid cases. It is indicated in typhoid and para-typhoid fevers.

**Children's Bi-Intestiphage**—(Special for Children's Diarrhoea and Dysentery.) A mixture of the various races of highly potent bacteriophages isolated in India exclusively from cases of intestinal infection of bacterial origin due to highly virulent dysentery and diarrhoea-producing organisms in children and infants.

**Bi-Coliphage**—A mixture of various races of virulent coliphage isolated in India from cases of cystitis, puerperal sepsis and other localised urinary infections. It is also used as local irrigation, besides orally.

*As the Bi-Bacteriophage is destroyed by the HCl of the gastric juice, heat and antiseptics, it should be given by mouth always in an empty stomach with alkaline water, and the internal use of all antiseptics should be suspended during the treatment with phage.*

ADVT.

apart from these two organisms there are many more gram-negative non-lactose fermenters that have been isolated from stools of dysentery cases.

Castellani, Manson-Bahr, Pasricha, De, Trivedi and other workers in the tropics have reported cases of dysentery where organisms other than B. Shiga and B. Flexner were isolated from the stools of dysentery cases. It may be mentioned here that B. Asiaticus, B. Gartner, B. Morgan, B. Carolinus, B. Sonne, B. Pyocyanus etc., have occasionally been found to cause symptoms of dysentery. It is desirable that clinicians should bear this in mind, as they may sometimes be misled by laboratory reports of stool examination where one of such organisms may be mentioned as the organism isolated from the stool. Another idea that is sometimes found to be present is that all acute dysenteries are due to bacillary infection whereas the sub-acute or the chronic conditions are due to protozoal infection. There are cases on record where organisms like B. Shiga have been isolated from stools of chronic dysentery patients. Similarly Entamoeba histolytica has been found in stools of patients suffering from the most severe type of dysentery of sudden onset.

When the question of etiology is being discussed, mention may be made of another condition, viz., summer diarrhoea of infants. This condition is also probably due to bacterial infection. Workers from different parts of the world report that organisms of the nature of B. Morgan have frequently been isolated in such cases.

Thus treatment of bacillary dysentery means devising of methods to deal with the organisms that cause this disease. If the principal agent is not successfully tackled, it is useless to carry out symptomatic treatment only. Methods either to kill the organisms or to enhance the immune bodies to counteract the mischief done by them should be found out. The discovery of a potent anti-toxic serum against B. Shiga made the treatment of this condition comparatively easy. Indeed, if sufficient serum could be given at the earliest opportunity, the death rate is almost nil. But what about the treatment of dysentery due to B. Flexner or the others e.g., B. Asiaticus, B. Gartner, B. Sonne, etc.?

Unless we could find out some specific curative agent against these organisms we would be absolutely helpless in these cases.

The discovery by Twort (1915) and d'Herelle (1917) that in filtrates of stools of dysentery patients certain properties develop by which the causative organism could be broken down or "dissolved" marked indeed a new era in the chapter of treatment of bacillary dysentery. This particular phenomenon was described by d'Herelle as "bacteriophagy" and was said to be due to certain ultra-microscopic living bodies called "bacteriophage". Further studies with bacteriophage showed that these bodies developed in nature *de novo*, side by side with an organism. Thus it developed in the intestines of patients in

the sub-acute or the convalescent stage of the illness. They have also been isolated from samples of river water, pond water, sewage water, etc., in fact from any sample of water or material where some infective organism is also growing.

By improved methods of filtration it has been possible to isolate bacteriophage in its pure form. The phenomenon of lysis is the effect of bacteriophage activity. The *modus operandi* has not yet been clearly understood. Speculative theories try to explain the different stages of action. Of course under special dark ground illumination methods it has now been possible to see the changes that take place in organisms when they are brought in contact with their corresponding bacteriophages. Under such arrangement, we see that when the bacteria are brought in contact with its phage the organisms, after sometime, gradually swell uptill a stage comes when no further swelling becomes possible and the bacterial body wall gives way—the entire organisms breaking down into fragments. It is suggested that this swelling and subsequent breaking down is due to multiplication of the bacteriophage within the cell body—this process continues till an optimum number is reached after which the organism breaks down, releasing the young "corpuscles" which in turn again attack fresh organisms. If this process goes on for sometime, ultimately we get a condition when all the organisms seem to be phage-infected, which naturally means their death. This would result in a complete control over the infecting agent and clinically a cure of the disease.

In bacillary dysentery then, the application of the corresponding bacteriophage seems to be the ideal method of controlling the infection. Whatever be the organism whether B. Shiga, B. Flexner, B. Asiaticus, B. Gartner, B. Sonne, B. Morgan or others, with the corresponding bacteriophage we are in a position to effectively control these organisms.

In clinical practice, thus we have specific agents to control intestinal diseases due to bacterial infections. A word of caution is however necessary here. The selection of the phage should be made carefully in order to get the desired results. If phages are used which contain no "seeds" against the particular infecting organism, as expected, no good result follows. In clinical practice where no laboratory facility exists for immediate examination and culturing of stools, the best practice would be to use a polyvalent phage with a combination of as many different kinds of seeds as possible. An intestiphage with a wide range of activity would be the ideal choice.

In the practical field, the application of bacteriophage therapy has given excellent results in many cases. For the last few years in India, phage has been tried widely not only in sporadic cases, but also in epidemics and the results have been quite encouraging. It has been given in bacillary dysentery, cholera and enteric infections. The most amazing results however have been with bacillary dysentery.

d'Herelle, Asheshov, Morrison and others have tried bacteriophage both as a prophylactic as well as a curative in cholera with good results.

In epidemic dysentery, Morrison obtained encouraging results with bacteriophage.

Col. Chopra in his *Handbook of Tropical Therapeutics* advocates trial with phage in bacillary dysentery.

According to the majority of workers, the dose usually recommended is 3 c.c. in  $\frac{1}{2}$ —1 oz. of alkaline water repeated every 2 hours till symptoms subside. Modification of the dose should be made in the more severe cases. In such cases it is better to repeat the dose every hour.

All other oral medicinal treatments specially of the nature of intestinal antiseptics should be suspended. There is no contraindication to serum, general stimulants, glucose etc., administered parenterally. Another point worth mention is, if the general symptoms do not improve within a reasonable time, say six hours, bacteriophage need not be given any more.

This evidently leads us to another question *viz.*,—the limitations of bacteriophage therapy.

The mode of action of bacteriophage is specific, hence the slightest indiscretion in the choice of the case is sure to result in disappointment. Bacteriophage acts on bacteria, hence it should be only used in bacillary infections of the intestines. Of course where clinical laboratories do not exist many a case of amoebic dysentery is often given bacteriophage. Hence in such cases the failure to respond to phage should be an indication of revising the diagnosis.

Another factor which occasionally results in failure of phage therapy is that the infecting organism may be resistant to the phage given. This natural resistance to a particular seed has enabled us to type organisms according to what is known as "phage types". The best work regards this typing has been with *V. cholera*, partly by Asheshov and partly by Pasricha. This is probably the reason why in some cases of cholera, we find no improvement in spite of giving heavy doses of cholera phage.

In this short summary of bacteriophage therapy in bacillary dysentery, we have tried to discuss the mode of action of bacteriophage in bacillary dysentery. In fact we can now safely rely on bacteriophage as an effective therapy in such conditions, provided the selection of cases and that of the phage are properly made.

*Bacteriophage is a living corpuscular being extremely minute belonging to class protobes (ultraviruses). It is a parasite of bacteria which it destroys while reproducing at their expense. Recovery in acute bacterial disease is not due as hitherto believed to phenomenon of immunity but results from the destruction of pathogenic bacteria by the bacteriophages. Immunity follows recovery, of which it is a consequence, but never precedes it—D'Herelle in his Studies on Cholera.*

## Non-Specific Dysenteries

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It must at the very outset be stated that the term dysentery connotes a group of symptoms (*i. e.*, passage of blood and mucus in stools with or without associated tenesmus) and not a disease *sui generis*. The causative factors of dysentery are manifold and may be broadly classified as belonging to two main groups, the specific and the non-specific. The former group comprises those resulting from protozoal, bacillary and helminthic infections of the intestinal tract. The scope of this paper is to present a comprehensive survey of such of those causes of the dysenteric syndrome as do not fall in the above group. It must however be clearly emphasised that the division between specific and non-specific groups is only arbitrary inasmuch as there are certain types in the latter group on the non-specific nature of which doubt is thrown by some of the present-day workers, *e.g.*, ulcerative colitis which is said to be due to allergy the exact nature of which is not yet known. To a certain extent perhaps it is justifiable to bring in the term pseudo-dysenteries as being equivalent to the non-specific type, not with a view to draw any landmark but to point out the importance of a systematic procedure in a given case so that the more important causes are not lost sight of in elucidating the diagnosis. The classification of such a vague group as non-specific dysenteries presents many practical difficulties. The following however may be of some value:

- (A) Causes in the intestine including rectal and peri-rectal conditions
- (B) Symptomatic haemorrhagic states
- (C) Certain fevers
- (D) Metabolic disorders
- (E) Inorganic poisons:—(1) metallic (2) non-metallic.

### Group (A): Intestinal Causes

In group (A) the causes are of diverse variety and importance. No attempt has been made to further subdivide into acute and chronic as many of the former varieties are merely acute exacerbations of a chronic lesion.

#### (1) ULCERATIVE COLITIS

This is of great importance. This condition though met with quite often in India is not yet so common as in the West where specific dysenteries do not occur in an epidemic form as they do in our country. It occurs in young people and in both sexes alike. Whether it is infective or not is still open to doubt. Certain authorities attribute it to an atypical form of Flexner's bacillus while others opine that the condition is due to a cococcus resembling the pneumococcus. Yet others think that it is the result of an allergic manifestation, although the specific allergen is yet undetermined.

**Pathology.**—The mucous membrane of the colon presents a picture of acute inflammation with minute submucous haemorrhages and patchy necrotic areas, the denudation of which results in superficial ulceration. The ulcers may be miliary or extend superficially with their base being formed by the submucous or muscular coat. In fatal cases the whole colon and rectum are involved, at first presenting minute erosions which subsequently lead to extensive ulceration. The mesenteric glands are inflamed and enlarged. Owing to the friability of the wall of the bowel perforation occurs at times. The mucosa intervening between the ulcers may get swollen giving rise to a condition resembling multiple flat polypi—colitis polyposa. Microscopically it is seen that the mucosa is absent and the submucous coat is infiltrated with rammed cells and polymorphs. Peritoneum over the deep ulcers is thickened.

**Symptoms** are very variable. The condition is rarely of an acute onset with pyrexia and frequent passage of blood and mucus mixed with watery stools. More commonly however the onset is subacute and there is only passage of blood and mucus with little or no watery diarrhoea. Even the few cases of acute onset are now proved to be exacerbations of a pre-existing chronic lesion. There are several small watery stools mixed with blood, pus and mucus occasionally distinguished with the aid of a microscope only. The blood passed is of a bright red colour and at times shows a few clots in it. The mucus may be transparent or opaque according as to whether it is intermixed or not with pus. Rarely there are small blotches of pus itself. Abdomen is not distended and there is no pain apart from a slight colic prior to defecation. Tenesmus is invariably absent being noticed only when the lesion is situated in the anal canal. When localised peritonitis has occurred there is tenderness and rigidity. Patient's appetite is good but owing to the protracted diarrhoea emaciation sets in and the continued loss of blood results in severe anaemia—the haemoglobin percentage being sometimes as low as 20%. The patient wastes to a shadow and generalised oedema and ascites may develop in the terminal stage. Hypochlorhydria has been noticed even in the earliest stages.

**Diagnosis.**—A systematic rectal examination, abdominal palpation and sigmoidoscopy will exclude a malignant growth in the rectum and lower part of pelvic colon. When the growth is too high a careful examination reveals that blood, pus and mucus are seen to come from the inaccessible part. Mucus from the surface of the sigmoidoscope should be examined for amoebic cysts microscopically and cultured for bacteria. Often times the sigmoidoscopic appearance in cases of chronic bacillary dysentery and ulcerative colitis is identical to a perplexing degree. In the early stages the ulcers are so minute that they are indistinguishable without a magnifying eyepiece. When ulcers are more extensive they are seen to possess irregular edges with a greyish floor. A barium meal reveals hypermotility of the colon with loss of its normal haustrum (pipe-stem

colon). In advanced cases the bowel is shortened and the deep ulcers present a feathery, moth-eaten appearance. In the later stages multiple strictures are recognised by sigmoidoscopy and a repeated barium meal is of value in assessing the operability in a given case, but surgical interference is indicated only if stasis is present.

**Complications** are rare if treatment is undertaken early. Localised or generalised peritonitis, polyposis intestini, malignant changes, formation of peri-anal abscess or fistula-in-ano are the most important ones.

**Treatment.**—An early recognition of the condition by sigmoidoscopy is essential for successful treatment. A preliminary saline or 2% soda bicarb. bowel wash followed by tannic acid retention enema  $\frac{1}{2}$ –2 grs. per oz. is advocated by some. The solution is administered slowly through a soft catheter introduced  $1\frac{1}{2}$ " into the anus and the quantity varies with the extent of the lesion from  $\frac{1}{4}$  to  $1\frac{1}{2}$  pints. The enema is retained for  $\frac{1}{2}$  hour. Chopra and Ray recommend rectal instillation of cod-liver oil or emulsion, and they have found it to be beneficial.

Polyvalent anti-dysenteric serum 50–100 c. c. daily for 7–10 days is said to be of some value. Bowel washes with rivanol, 1 in 2000 gradually increased to 1 in 500 is of value. Alternatively silver nitrate 1 in 500 (1 gr. to 1 oz.) may be used. Kaolin and charcoal powders (3 drs. each 4 times daily) serve to relieve flatulence. Rest in bed, low residue diet, vitamins C & B complex, Bayer's Torantil in allergic cases are warmly recommended by Chopra and Ray.

Any septic foci in teeth, tonsils, etc., must be adequately treated. The diet should be bland and consist of conjees (arrow root and sago), butter-milk, custard jellies and fruit juices.

In advanced cases blood transfusion is essential and surgical interference in the form of an appendicostomy or caecostomy with subsequent irrigations through the artificial opening may be necessary. However, with improved methods of early diagnosis there should never occur the necessity for such measures.

#### (2) MUCOUS COLITIS OR MUCO-MEMBRANOUS COLIC

This is a condition characterised by the passage of mucus in the stools occurring in individuals who are chronically constipated. An underlying neurotic factor is also partly responsible by exciting reflex activity of the colon. The mucus may be passed in flakes or as casts. Sigmoidoscopy does not reveal any abnormality in the mucosa.

**Symptoms.**—There is a certain amount of abdominal discomfort associated with a slight tenderness over the left-iliac fossa. Rarely the caecum also may be tender on palpation. Motions are usually passed as scybala coated with mucus. Occasionally the faeces may be intermixed with blood. Membranous casts about an inch and half long are occasionally passed. The passage of intestinal sand is a rare feature and must be distinguished from a similar condition resulting from

over-eating bananas and pears. True intestinal sand consists of calcium salts of palmitic and stearic acids with calcium phosphate at times intermixed. In the long run the patient becomes depressed and hypochondriac.

**Treatment.**—The patient must be discouraged from paying undue attention to the nature of his stools and his nerves must be set right. Abdominal massage is a useful measure in toning up the intestine. Drugs as belladonna and bromides relieve the spasmodic condition of the colon. Intestinal lavage may be undertaken with normal saline or 2% soda bicarb. solution. Medicinal waters of Bath, Harrogate etc., are also used for this purpose. The following prescriptions are of value in relieving pain due to flatulence:

|    |                                 |                     |
|----|---------------------------------|---------------------|
| R/ | Ext. belladonna                 | gr. $\frac{1}{2}$   |
|    | Menthol                         | " 1 $\frac{1}{2}$   |
|    | Ext. valerian                   | " 4 $\frac{1}{2}$   |
|    | " gentian                       | q. s.               |
|    | Fiat pills 3. One pill t. d. s. |                     |
| R/ | Pot. bromide                    | gr. 30              |
|    | Chlorodyne                      | m. 30               |
|    | Tr. hyoscyamus                  | dr. 1 $\frac{1}{2}$ |
|    | Syr. zingiberis                 | " 1 $\frac{1}{2}$   |
|    | Aqua chloroform ad.             | oz. 3               |
|    | F. M. one ounce t. d. s.        |                     |

Galvanic massage may also be tried. Except liquid paraffin all purgatives should be rigorously avoided. The diet should consist of whey, puddings, curds, vegetable soups etc. Ispaghul seeds may be given in the form of sherbet. This is a widely-known indigenous prescription.

### (3) CHRONIC CATARRHAL COLITIS

This is a condition resulting from the abuse of purgatives. The patient presents vague dyspeptic symptoms as abdominal discomfort and fulness. There is no pain or tenderness. The discomfort is worse after meals. The stools are mixed with mucus and occasionally with blood.

**Treatment** is by administration of Tr. belladonna in 30 minims dose thrice daily or of a pill made of codein phosphates ( $\frac{1}{4}$  gr.) and extract of gentian three times a day. B. acidophilus, one dram thrice a day, may also be administered. Sour butter-milk may be of some use in these cases.

### (4) DIVERTICULITIS

This condition is, of late, assuming great importance. True to its name, (diverticula means a way-side house of ill-fame) it is a prelude to many ills. In individuals habitually constipated, particularly the obese where the intestinal walls are more liable to give way in places, these diverticula occur. They often project into the appendices epiploicae and are usually situated in the sigmoid colon, the rectum being invariably free. They are at first constituted by all the three coats of intestine, but later on the muscle fibres atrophy and flask shaped elongations result. Faecal material may get inspired into them giving rise to enteroliths. In chronic cases the symptoms are those of abdominal discomfort, localised tenderness and a colicky pain increasing in severity and frequency. Constipation is marked, and mucus and pus appear in the stools. Visible blood is rare. There is a low grade tem-

perature and leucocytosis. In very chronic cases localised peritonitis may give rise to a palpable mass which may be mistaken for a growth. For diagnostic purposes a barium enema is more valuable than a meal.

**Treatment** consists in withholding all aperients except liquid paraffin and a strict dietetic regime. Liquid paraffin may be given in the night as an enema to be retained till morning. Abdominal massage is contraindicated as the diverticula may rupture. Operative treatment is indicated in acute complications as abscess formation, perforation, etc.

### (5) INTUSSUSCEPTION

This is a condition in which one portion of the bowel gets invaginated into another. It is supposed to result from irregular and violent peristalsis induced by the presence of intestinal polypi, malignant growths, worms, scybala, etc.

The chief varieties are ileo-caecal, enteric, colic and ileo-colic. The first named is by far the commonest met with.

There are two types of this affection—the acute and the chronic. *Acute intussusception* occurs most frequently in infants under two years of age. It is characterised by a sudden onset associated with a severe pain at first localised and paroxysmal but later becoming diffuse and continuous. There is vomiting of a non-faeculent type usually, and discharge of blood-stained mucus the appearance of which has been likened to the red-currant jelly. Tenesmus is also present. Collapse sets in and in fatal cases death results in 24 hours. The issue may be delayed up to a week, death resulting from exhaustion or peritonitis. Abdominal examination does not reveal any marked tenderness or distension. In about 50 per cent of cases the classical sausage shaped tumour can be felt. This follows the course of the intussusception and is curved due to mesenteric traction. There may be a feeling of emptiness in the right iliac fossa—*signe de dance*. Very rarely does a natural cure follow resulting from spontaneous reduction or sloughing of the intussusception. A stricture results in the latter case. Before arriving at a diagnosis a systematic rectal and abdominal examination must be made. Certain cases are met with where the intussusception is actually protruding through the anus, a condition closely simulating prolapse of rectum. The latter is differentiated from the simple fact that the finger cannot be passed up by the side of the protruding mass. The reverse obtains in a true case of intussusception.

**Treatment.**—When the condition has been diagnosed it is of utmost importance that the child be put under the influence of opium to check peristalsis which otherwise would only serve to increase the size of intussusception. In mild cases, air infiltration and enema of warm water or oil introduced by means of a catheter from a height of 1 $\frac{1}{2}$ " above the patient's pelvis may be tried. In most cases such cursory measures prove of no value, and no time should be lost to call for surgical interference.

### (5) CHRONIC INTUSSUSCEPTION

It is more an affection of adults, the onset being insidious and the course variable. About 20% of the cases are secondary to innocent tumours. Constipation is invariably present and the chief complaint is an intermittent colicky pain which increases in severity and frequency as the case progresses. There is passage of blood-stained mucus, especially marked in entero-colic and colic varieties of intussusception. The abdomen is flaccid but presents visible peristalsis and in about 50% of cases a tumour is palpable.

**Treatment** is entirely surgical.

### (6) INTESTINAL TUBERCULOSIS

Primary intestinal tuberculosis is a rare condition in India owing to the universal habit of using boiled milk. Hence the commonest mode of infection is by the swallowing of sputum infected with T. B. It has been observed in about 50% of cases of advanced pulmonary tuberculosis. It is by no means however always a terminal occurrence.

**Pathology.**—The tubercle bacilli invade tubular glands of intestinal mucosa and give rise to inflammatory exudate. Mesenteric glands get involved and undergo caseation. In the intestine the commonest site affected is the ileo-caecal region spreading up and down from thence. By caseation followed by necrosis the classical ragged undermined ulcers form and these extend transversely along the gut (the girdle ulcer). Perforation is rare owing to the adhesion that form externally binding the intestinal coils together. The occurrence of endarteritis obliterans prevents a large hemorrhage. Healing results in stricture formation.

**Symptoms.**—There are recurrent attacks of pain due to spasm of the bowels. The ulcers themselves are insensitise. The involvement and subsequent degeneration of Auerbach's plexus gives rise to hypermotility. Thus there is diarrhoea alternating with constipation. Pus and blood is occasionally passed mixed with the faeces.

### (7) CROHN'S DISEASE OR REGIONAL ILEITIS

It is an affection seen in young adults who present symptoms of chronic intestinal obstruction and are wasted. There is a thickening of the terminal part of the ileum. Although akin to the hyperplastic type of tuberculosis the nature of Crohn's disease is still uncertain. There is a chronic inflammation resulting in the production of a non-specific granuloma. In this condition occasionally there is passage of frequent stools mixed with mucus.

### (8) SYPHILITIC DISEASE OF THE RECTUM

Tertiary syphilitic lesions are uncommonly noticed in the rectum. A gummatous deposit occurs in the submucosa which is followed subsequently by extensive ulceration. The ulcers possess a sloughy base and indurated margins. There is passage of bright red blood alone or mixed with mucus and faeces. This condition is differen-

tiated from malignant disease by its multiple, lesions, a previous history of syphilis, positive Wassermann and the result of biopsy. Stenosis may give rise to stricture formation.

### (9) STERCORAL ULCERS

These occur in individuals afflicted by chronic constipation. The ulcers bleed owing to the irritating nature of the hard scybala which get coated with mucus resulting from chronic irritation.

### (10) POLYPUS RECTI

This occurs commonly in children. They are either adenomata arising from Lieberkuhn's follicles or mere fibrous tissue formations covered over by mucous membrane. Invariably they are solitary. Proctoscopy reveals small cherry-like growth with a long pedicle allowing of free motility.

The symptoms are irritability of the bowel and passage of blood mixed with little mucus *per rectum*.

**Treatment** is entirely surgical.

### (11) POLYPOSIS INTESTINI

It is a condition of rare occurrence characterised by the presence of multiple polypoid growths over the colon and rectum. It occurs in adults and has been observed to be hereditary. They are very liable to go on to malignancy.

**Symptoms.**—There is long-standing diarrhoea with the passage of blood and pus mixed with the stools. When occurring in the very young it leads to infantilism and in adults to cachexia. The presence of tenesmus and abdominal pain is uncommon. Sigmoidoscopy is a valuable aid in differentiating this condition from ulcerative colitis. A barium enema reveals rounded semi-translucent anus.

**Treatment.**—Deep x-ray therapy has been advocated by some. Usually surgical interference is necessary in the form of colectomy.

### (12) PAPILLOMA OF THE RECTUM

It is a rare condition giving rise to irritability and bleeding *per rectum*. It has a tendency to extend and may prove fatal from hemorrhage.

### (13) CANCER COLON

It is a condition most commonly met with in men of cancer age i.e., 45, but occasionally also in young adults. 60% of these growths are situated in the rectum. The disease extends by direct invasion, also by the lymphatic stream and by portal circulation. Pathologically it is an adenocarcinoma which occasionally undergoes colloid degeneration. Clinically it may be of the annular type leading to stenosis or the fungating type in which a cauliflower-like mass projects into the lumen of the bowel. Toxic absorption from the ulcerated surface is always a grave addition to the case. The growth in time involves the surrounding viscera and structures; peri-rectal suppuration and fistula formation also result.

**Symptoms.**—The symptoms are so vague and the onset so insidious that very often a diagnosis is

not made until a period when the prognosis is assuredly gloomy. Hence it is that the early vague symptoms should be paid the attention they deserve and not neglected until it is too late to repent. Increasing degree of constipation and the necessity of greater amounts of aperients should put us on our guard when it occurs in middle aged individuals. There may be constipation alternating with diarrhoea associated with passage of blood and mucus. As the disease progresses the constipation remains obstinate and is unrelieved by enemata or aperients. Bleeding is the earliest sign in rectal cancer while it is a late manifestation in the case of cancer of sigmoid colon. At first there is abdominal discomfort and dragging pain in the rectum which later on develops into true tenesmus. Pain may also radiate to the back and down the legs. Blood is at first passed with the motion but later may be discharged independently. In later stages cachexia sets in. There is a marked anaemia. A careful rectal examination suffices in most cases in arriving at a diagnosis and where it fails sigmoidoscopy will come to the rescue. If still in doubt a biopsy can be done.

*Treatment* is entirely in the province of surgeon.

#### (14) SARCOMA RECTUM

It is an uncommon tumour arising from the submucosa and projecting into the lumen. It is less painful than carcinoma but otherwise presents the same symptoms.

#### (15) FIBROUS STRICTURE OF THE RECTUM

It is a condition usually met with in women over 40 years of age and may result from ulcers following diarrhoea, dysentery, tuberculosis, syphilis, etc. Gonorrhœal proctitis is a rare cause. Pelvic cellulitis and suppuration resulting in adhesions serve to bind the rectum to the sacral wall or constrict it. Recurrent attacks of inflamed piles also lead to stenosis. Post-operative strictures are also occasionally noticed. Chronic fistula gives rise to a stricture as a sequel to inflammatory fibrosis. Radium used for treating cancer of cervix uteri may result in a stricture of rectum. Another cause which deserves more than passing attention is lympho-granuloma inguinale caused by a filterable virus and giving rise to a group of symptoms—the genital-ano-rectal syndrome. Infection spreads up the perineal lymphatics and into the rectum giving rise to a stricture which is very troublesome to treat. Prolonged labour is another cause of stricture rectum.

*Symptoms.*—Constipation is marked with resulting diarrhoea due to irritation caused by the retained faeces. Aperients are always necessary and the motions are characteristic, being like pipe stems. Pain and abdominal discomfort are present with passage of blood and mucus mixed with the stools. Stercoral ulcers, fistula, etc., may result in untreated cases. Rectal examination reveals in the annular variety the feeling of a hole in the turnip or that of the OS of cervix uteri. In certain cases the stricture is more extensive. The gut below the stricture is seen to be ballooned.

*Treatment.*—Diet must be regulated so as not to leave any unnecessary debris. Liquid paraffin and enemata are also useful. Dilatation by bougies may be undertaken. Operative treatment is rarely necessary.

#### (16) PROCTITIS

It is an inflammation of rectum due to presence of piles, foreign bodies, e.g., gall stones, fish bones, etc., give rise to recurring tenesmus, pain of a bearing down character and passage of blood and mucus or muco-pus. Gonorrhœa is often a cause of proctitis, the mode of infection being indirect in women and direct in men. Unclean habits and unnatural sexual practices may lead to this condition in both sexes.

*Treatment.*—Liquid paraffin orally and rectal saline irrigations. Starch and opium enema or morphia and atropine suppositories are valuable in relieving the tenesmus.

#### (17) FISSURE ANUS

It is a triangular-shaped ulcer at the anal margin usually single and posterior, resulting from the stretching of anus by hard faeces. In women fissure may be anterior owing to the weakness of the perineal body. Often the fissure is syphilitic or tuberculous. A sentinel pit may form at the lower end of the fissure and in very chronic cases the fissure is much indurated.

This condition is most agonizing, characterised by a burning pain during and after defaecation radiating to the back and down the legs. Tenesmus is marked and the faeces are streaked with blood and muco-pus.

*Treatment* is by regulating the bowels and introducing a cocaine suppository prior to defaecation. Stretching under anaesthesia, injection of pentocaine around the base of the fissure and like measures may be adopted with success.

#### (18) HAEMORRHOIDS

Internal piles may not give rise to any definite symptoms until hæmorrhage occurs. There is however a sense of fulness about the anus, especially marked before and after defaecation and passage of a little glairy mucus. Later, bleeding occurs and varies in degree from a few drops to an alarmingly large quantity. Severe anaemia results. Blood-stained mucus is frequently passed. The patient is prostrated from the pain and hæmorrhage. Bleeding is beneficial and should not be arrested in conditions of portal obstruction. Diagnosis is never difficult. A visual and digital examination of the rectum always gives definite evidence. Complications such as thrombosis and strangulation occur in neglected cases.

*Treatment.*—The bowels should be kept well open by laxatives like confectio senna or sulphuris or liquid paraffin. The diet should be regulated. Sedentary life and alcoholism are to be avoided. Local applications as ung. gall. c. opio, hamamelis and proprietary preparations as Hadensa, adrenalin in chlorotone ointment, Recto-serol etc., give

considerable relief. Local cleanliness is essential. In protracted cases operative treatment is advisable either in the form of injection treatment or excision.

#### (19) INTESTINAL INFARCTION

This results from embolism or thrombosis of superior or inferior mesenteric vessels. The affection of the latter vessels is of less consequence as they communicate with superior mesenteric vessels above and inferior hæmorrhoidal vessels below. The infarct is situated in ileum or lower part of jejunum and its occurrence is heralded by severe abdominal pain due to sudden localised anaemia or spasm. Bleeding occurs from the bowel and diarrhoea is also present. A fatal issue may supervene owing to gangrene of the affected portion of the gut.

#### Group (B) Symptomatic Haemorrhagic States

##### HENOCH'S PURPURA

It is otherwise styled as anapylactoid purpura, toxic purpura or hæmorrhagic capillary toxicosis. The disease is allergic in nature and the lesions probably result from an abnormal permeability of the capillaries allowing blood and plasma to escape from their walls. The sensitising agent may be either bacterial or dietary.

*Symptoms.*—There is usually a preliminary period of headache, malaise, anorexia and slight temperature. The rectal disease sets in with development of purpuric spots over face and limbs especially marked around hair follicles. In pronounced cases eczemas and wheals develop with erythematous zones around. Bleeding may occur into the muscles, sub-periosteal spaces, viscera etc. The gastro-intestinal symptoms comprise a severe form of colic, passage of blood mixed with stools and bilious vomiting. This condition must be differentiated from acute intussusception. In the latter, purpuric spots on the skin are absent. It is however a paradoxical fact that intussusception may supervene if there is thickening and stiffening of the intestine as in a case of Henoch's purpura. Hæmatoma may be present. Spleen is palpable and urinary changes are present, the urine containing albumin, blood cells and casts. Nephritis may supervene. Slight anaemia and a mild degree of leucocytosis are present. Otherwise there is no detectable abnormality of the blood condition. Prognosis is good except for rare complications as nephritis or intestinal obstruction. Relapses are not uncommon.

*Treatment* is symptomatic and conservative. Complete rest is advised and patient is put on a suitable mixture to alkalise the urine. Diet should consist of milk only. In the chronic stage septic foci are dealt with and desensitisation attempted. The latter should never be done in the acute stage for fear of aggravating the disease. Calcium may be administered orally as lactate or by injection as gluconate. Horse serum in doses from 10-20 c. c. subcutaneously may also be tried.

#### Group (C) Certain Fevers

##### (1) MALARIAL DYSENTERY

In the abdominal manifestation of sub-tertian or malignant Indian malaria there may be a mere blood stained discharge or an actual intestinal hæmorrhage. Petechial hæmorrhages occur into the intestinal mucosa and Manson quotes instances where the parasite was at first discovered in the red blood cells passed *per rectum*. The clinical history and enlarged spleen should suggest malaria. In these cases the outlook is gloomy. The bleeding is supposed to result from damage of capillary endothelium from the action of the malarial parasite on it.

*Treatment* is the same as that for malaria.

##### (2) KALA-AZAR DYSENTERY

In the very advanced cases of kala-azar blood and mucus may be passed mixed with the stools. This condition is attributed to an ulceration of the bowel resulting from the presence of Leishman Donovan body. This statement is substantiated by the observation that L. D. bodies are discovered in the villi of small intestine and polypoid masses of large intestine.

*Treatment* with antimony compounds clear the dysenteric symptoms.

##### (3) PARATYPHOID FEVER

In paratyphoid fever particularly in para-B, infection frequent passage of stools with blood and mucus has been noticed before the actual disease proclaims itself.

#### Group (D) Metabolic Disorders

##### URÆMIA

Owing to the excretion of toxins into the intestinal tract as a result of the suspension of renal functions a severe form of diarrhoea occurs. In cases of chronic uraemia rarely ulceration of the colon has been noticed giving rise to discharge of blood and mucus mixed with watery stools.

#### Group (E) Inorganic Poisons (A) Metallic

##### (i) MERCURY POISONING

Chronic mercurial poisoning occurs chiefly in those engaged in mercury mines and in the manufactures of instruments as thermometers, barometers etc., which involve the constant handling of this metal. Continued administration of calomel or blue pills or using external ointments containing mercury may also give rise to this condition.

*Symptoms* are mainly dyspepsia, anaemia, cachexia and loss of flesh. Lack of oral hygiene results in the appearance of a blue line on the gums. Stomatitis, halitosis and gingivitis are of constant occurrence. Ulceration of the intestine results, and blood and mucus is passed with the stools. Fine tremors, sensory disturbances and

a certain degree of mental symptoms too have been noticed.

**Diagnosis.**—The history and symptoms make it obvious. However a confirmation is made by withholding any mercury that may be being administered, and examining the faeces. The faeces is treated with hydrochloric acid and potassium chloras to destroy organic matter. The resultant solution is electrolysed on which the mercury present gets deposited on a gold kathode. It is dissolved out and chemically analysed.

**Treatment.**—Remove from environment or stop administering mercurial preparations if patient has been having any. Saline aperients are freely used. Hygiene of the mouth and teeth should be specially attended to.

**Acute mercurial poisoning** is rare in India and is usually accidental. The symptoms are those of non-corrosive irritant poisoning. Mercuric compounds are more active than mercurous compounds. There is immediate intense burning pain in the mouth and throat and the mucous membrane over these areas becomes pallid and shrivelled. Abdominal discomfort is present with passage of bloody stools.

## (2) ARSENICAL POISONING

Arsenic is the commonest drug used by murderers in India. It is comparatively tasteless and easily available in any bazaar. It gives rise to abdominal pain, intense thirst, tenesmus and passage of bloody stools. The vomit may also be blood stained.

Other symptoms are anuria, uræmia, exfoliative dermatitis and jaundice. Nervous symptom as collapse, parasthesia, convulsions, delirium, headache or coma may be noticed. Acute arsenical poisoning is differentiated from cholera by the presence of blood in the stools, the pain in the throat and the absence of the characteristic rice water appearance of the stools seen in cholera. Slow arsenical poisoning may result in severe tenesmus and watery stools mixed with blood and mucus. The celebrated Maybrick case reveals the interesting features of chronic arsenical poisoning.

## (B) Non Metallic Poisons

### PHOSPHOROUS AND OLEANDER POISONING

The latter causes severe dysenteric like symptoms. Yellow phosphorous is contained in pastes made for killing vermins and in the material used for tipping lucifer matches. In adults, poisoning is usually suicidal while in children it is accidental. The symptoms are those of any acute irritant poisoning: epigastric pain followed by vomiting and purging mixed with blood. The condition is distinguished by the following characteristic features: garlicky odour of the breath, phosphorescent nature of evacuations, and jaundice. Complications as retention of urine, haematoma, purpura cramps, tetany etc., may occur.

## FOREIGN MEDICAL NEWS

### Our London Letter

(From Our Own Correspondent)

#### ABORTION REPORT

The Report of the Interdepartmental Committee on abortion, published recently in this country, is an interesting document for many reasons. The British medical profession was expecting a definite statement or suggestion as to what constitutes criminal abortion, and what are the ethical and medical responsibilities of the doctor in giving evidence in cases in which criminal abortion is alleged. In this respect, the report is a great disappointment, obviously because the members of the Committee were perplexed at the complexity of the question. The report points out that there were about 40,000 criminal abortions every year. The members of the Committee think that it would be undesirable to authorise the medical officer of health to communicate with the police if there is any suspicion of criminal abortion in the case in which he is dealing. This, of course, protects the profession from future litigation, but it is difficult to think of any other way of getting reliable information unless the Medical Officer of Health gives the information to the police and is protected by statute for having given such information. The growing number of annual criminal abortions is a proof that detection and checking of such abortions would not be easy or possible without information from authorised sources. After all, prevention is better than booking the criminal, and the report does scant justice to this aspect of the problem. On the other hand, one of its recommendations is entirely unwarranted. The Committee suggests that a physician should report the case of therapeutical abortion, and that the police should have access to his records. This

#### Conclusion

The writer likes to reiterate the ambiguous nature of the terms non-specific or pseudo dysentery and hence the necessity for inclusion of such conditions as Crohn's disease which very rarely causes the dysenteric syndrome. For the sake of completeness little amount of latitude has been indulged in presenting this paper. The writer gratefully acknowledges the kind help and encouragement given by Dr. K. Narayana Murti, M. D., in getting this paper ready.

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Does not look like the Committee having confidence in the members of the profession, and it is hoped that this recommendation will not be accepted by the Minister of Health. The report on the whole, though erudite in some portions, is yet a jumble of good and bad recommendations.

#### MEDICINE STAMP DUTIES

A lively and hot debate was witnessed in the Parliament on 22nd June over the question of the repeal of Medicine Stamp Duty. Your Indian readers may not understand what is a Medicine Stamp Duty. It is a tax imposed on all proprietary medicines and has been in the statute book for the last 100 years. It yielded to the Government an annual revenue of about £1,400,000 to £75,000, the latter in the year of maximum evasion. The Chancellor of the Exchequer brought forward a bill to repeal this Duty, ostensibly on the representation of the affected interests. But the profession viewed this repeal in a different light. In a memorandum submitted by the doctor-members of the parliament for the information of its other members, it was forcibly pointed out that if this Duty was repealed it would affect qualified chemists, and also the Health Insurance Service and distribution of dangerous drugs and poisons. Even a large number of Government supporters accepted the rightful contentions embodied in this memorandum. This became quite obvious during parliamentary discussion. Member after member from the Government benches rose to support the amendment of Mr. Graham White to delete the clause. Sir Arnold Wilson who followed Mr. Graham spoke long, and effectively demolished the arguments of the Chancellor of the Exchequer. His speech was enlivened by wit and humour. He told the members that he would not go so far as Oliver Wendell Holmes who said:

"If the whole *Materia Medica* could be sunk to the bottom of the sea, it would all be better for the mankind, and all the worse for the sea."

but rest contented by quoting his own nurse:

"Remember child, that doctors think more men have died from drugs than drink."

The Government, seeing the position, gracefully accepted the deletion of the clause, and saved what would otherwise have been an ugly situation.

#### OPENING OF THE NEW LABORATORIES OF THE IMPERIAL CANCER RESEARCH FUND

The new buildings to provide better facilities for Cancer Research were opened on June 27. Sir Gowland Hopkins traced the origin and growth of Cancer Research in this country, and told how from small beginnings the cancer laboratories have now assumed such proportions which required greater space, more finance and more workers. He briefly reviewed the present position of the cancer problem, and said that with the assurance of fundamental knowledge about the actual nature of cancer, and clinician's skill and equipment, there is every justification for an optimistic outlook.

## Our American Letter

(From Our New York Correspondent)

#### AMERICAN PUBLIC AND EUTHANASIA

The question of mercy death is new to this country. A nation-wide survey by the American Institute of Public Opinion indicated that the country is almost evenly divided on the question of legalizing mercy deaths for hopeless sufferers, of whatever age or station in life, if the patient himself requests euthanasia and if carried under the supervision of the courts.

The question was: "Do you favour mercy deaths under Government supervision for hopeless invalids?"

The vote of the public is: For mercy deaths, 46 per cent; against mercy deaths, 54 per cent. A special survey of doctors two years ago revealed that 53 per cent favoured mercy deaths.

In Great Britain the principle of euthanasia is more familiar than it is in the United States and has more supporters. In a survey conducted by the British Institute of Public Opinion, overseas affiliate of the American institute, 69 per cent or more than two in three, said that they approved the proposal. As envisaged by euthanasia advocates in this country, the plan would call for the concurrence of both the patient and the doctor and a petition for euthanasia would have to be made in the regular courts.

The American survey shows that men are more inclined to favour the idea than women, and that younger persons are more favourable than their elders. The votes of these groups are:

|                     | For | Against |
|---------------------|-----|---------|
|                     | %   | %       |
| Men                 | 49  | 51      |
| Women               | 42  | 58      |
| Persons under 30    | 52  | 48      |
| Persons 30 to 49    | 44  | 56      |
| Persons 50 and over | 41  | 59      |

A retired army major, some time ago, suggested that persons on the relief rolls who are over 70 or 75 years old should be subjected to euthanasia. This remark started a real avalanche of protesting editorials and letters from the public. Every year the newspapers report instances where relatives or doctors have admitted taking the life of a suffering patient when there was no apparent medical remedy for his illness. Such "casual" mercy killings are not contemplated, however, by the Euthanasia Society of America, which has prepared a bill for introduction into the legislatures of various States.

#### NEW TREATMENT FOR MIGRAINE

The treatment of migraine headache by inhalation of 100% oxygen has yielded such encouraging result at the Mayo Clinic, Rochester, Minn. that W. C. Alvarez, of the division of medicine, feels that the method should be placed before the medical profession for further test. In addition to the inhalation mask, all that is required for this method is a tank of oxygen, a reducing valve, and

a flow meter. The oxygen is usually administered at a rate of between 6 and 8 liters per minute. It is essential that 100% oxygen be administered to relieve migraine. Comparatively little oxygen is needed, however, to stop the attacks, and the cost is comparatively low.

Alvarez cites some cases. A very nervous woman who was hardly out of attack of migraine before she went into another, and whose physician refused to take further responsibility for giving large daily doses of gynergen, was given prompt and grateful relief in every attack by inhalation of 100% oxygen. A man endured for many years attacks of extremely severe abdominal migraine; these attacks always prostrated him for a week or more and he did not get much help from gynergen alone, while inhalation of 100% oxygen aborted his attacks. A man with headache and nausea not typically migrainous breathed oxygen only after he had been in a spell for about two days, and the four hours of the treatment did not help him. Proper selection of the patients, therefore, is necessary.

#### OXALIC ACID TO CHECK BLEEDING

The discovery that oxalic acid makes possible the rapid coagulation of the blood was announced and demonstrated at the meeting of the Federation of American Societies for Experimental Biology. The report was given by Dr. A. Steinberg and Dr. W. R. Brown of the research foundation of the Kensington Hospital for Women in Philadelphia. Applied clinically in several hospitals, the oxalic acid treatment was reported successful in almost 1,000 cases of unchecked bleeding resulting from a variety of causes, in which all formerly known means had proved of no avail. With no exception, it was stated, the intravenous injection of three milligrams of oxalic acid, dissolved in distilled water, stopped the hemorrhages in periods of five minutes down to as low as forty-five seconds.

The clinical cases included six cases of hemophilia. The usual clotting time in these cases was from 30 minutes to 2 hours. The injections of oxalic acid reduced this time to 5 minutes, which is about the usual clotting period for normal persons. The treatment was applied "with gratifying results" to a wide variety of cases of excessive bleeding, including post-operative, post-partum and uterine hemorrhages, hematemesis, bleeding gastric ulcers, jaundice, hemoptysis, hematuria, epistaxis, melena neonatorum and other types of hemorrhages and hemorrhagic dyscrasia. The normal human blood contains 5.5 to 7.5 milligrams of oxalic acid per 100 cubic centimeters.

#### ARTIFICIAL INSEMINATION BY EXTRA MARITAL SPERMATOZOA

An extraordinary experiment has been made by Frances I. Seymour, M. D., New York. A married couple Mr. and Mrs. K. had been examined for sterility several times during their eleven years of married life. Both were in excellent health. The

husband's spermatozoa were very active, motility index 18 hours, the wife was normal in every way. All kinds of treatment, diet, change of environment etc., proved useless. The doctor was able, with the consent of all persons concerned (married couple, volunteer women and their husbands) to inseminate 16 women with Mr. K.'s spermatozoa. The women were artificially inseminated monthly for 6 months. No pregnancies ensued.

At the end of this time, consent was received from the participants in the experiment, that now a donor was used whose fertility index was known. The viability index of the donor's sperm (man with an intelligence quotient of 120, father of two children, eminent figure in his profession) was 31 hours. Because of the uncertainty of the exact time of ovulation, it was thought best to have four inseminations each month on the 6, 10, 12, and 14th day of each intermenstrual period, the first day of the period being considered as one. The frequency of these inseminations and the 31 hour viability index of the donor's spermatozoa gave reasonable assurance that the sperm would overlap the periods between inseminations.

The sixteen volunteers and the wife of Mr. K. were artificially inseminated over fourteen months when the rest of the volunteers, including Mrs. K., had become pregnant. The pregnancy was verified after the missing of the next monthly period and a positive Friedman reaction was obtained. The importance of this series is principally that motility and the retrieving of viable spermatozoa after coitus from the cervical canal does not exonerate the man as a cause of sterility. The fertility index of spermatozoa, Dr. Seymour adds, may not be judged by conformity of the count per cubic centimeter, by ascertainable motility or by any of the other physical factors diagnosable by ordinary microscopic examinations.

#### Our German Letter

(From Our Own Correspondent)

##### WHY INCREASED INCIDENCE OF RICKETS?

E. Rominger, ordinarius for pediatrics in Kiel, gave a detailed report on the increased incidence of rickets in Germany as well as in other European countries. The incidence of rickets among the nurslings of the Westphalian industry region for instance is between 37 and 46 per cent, in the city of Dortmund it reaches 55 per cent. In Basel, Switzerland, the incidence was found to be 75 per cent during the winter and 50 per cent during the summer, while from Vienna and Paris an incidence of 40 per cent is reported. The figures are 60 per cent for the winter and 40 per cent for the summer in Kiel. An increased incidence of rickets is even reported in Finland and Norway (where much fish oil is consumed by the population), and in the tropics where an intensive exposure to sunlight would be possible. Also in breast-fed nurslings in several countries, the incidence of rickets has remarkably increased.

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THE results of modern ante-natal care have emphasized the importance of proper nutrition of the expectant mother, in securing a normal pregnancy, labour and puerperium and in endowing the infant with an initially sound constitution.

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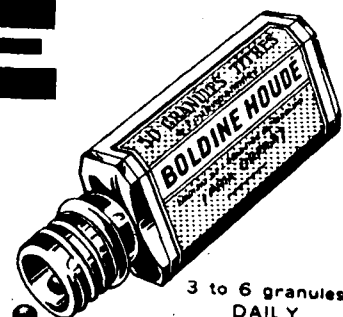
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The author believes, despite all these statistical figures, that the contradictions to the general idea of measures preventing rickets (i.e. sunlight, fish liver oil and breast-feeding) is only apparent. Rickets in the tropics exist only where the children remain confined in houses and huts for long months without ever being taken to the open air. In Northern countries the nurslings are fed too early with cow-milk and milk-gruel diet. And when breast-fed nurslings suffer from rickets, the nutrition of the mothers is frequently entirely improper. The increased incidence of rickets in Central Europe probably is connected with the inadequate dosage of antirachitic remedies such as cod liver oil. The toxicity of modern vitamin D preparations is very low. Remarkable changes do not occur before doses of 200 times the normal doses have been consumed for 4 to 5 weeks. Over-dosage, consequently, is very rare nowadays, while under-dosage is frequent. The correct dosage, however, cannot be laid down in general rules applying to every case, nor can it be left to any lay person.

### SHORTER MEDICAL CURRICULUM

The new medical curriculum which became effective in German Universities on April 1, 1939, has as its stated aim: to cut down the amount of time necessary to turn out a doctor of medicine, and thus to reduce the age at which a man may enter medical practice and found a family. With all time spent in High Schools, Universities, compulsory labour service, military service, internship, specialistic training, and preparation time for panel practice, a general practitioner is about 29 and a specialist about 33 before he is able to start an independent practice. A second reason for the shortening of the medical curriculum is the increased shortage of trained physicians in Germany which is due largely to the exclusion of Jewish practitioners and to the increased need of doctors in the newly built army.

To reduce by two years the time necessary to become a practitioner of medicine, the new curriculum is reduced from the present eleven to ten semesters (four pre-clinical and six clinical). The year of internship is served during and between the clinical semesters rather than after them as was the case so far. This arrangement will deprive German hospitals of the services of graduate interns; the "medicinal praktikanter" had to do the work of staff doctors for one year with no or a very low remuneration. Another half year is saved by reducing the time required for taking state licensing examinations. Throughout his medical course, the German medical student, henceforth, will no longer have any time at all for vacations or leisure periods.

In future less stress will be placed on theoretical training than on practical performance. The courses in zoology, botany, chemistry and physics will be freed of a vast amount of detail, and so will the course of descriptive anatomy while topographic anatomy will be given greater consideration. New items which have been introduced

into the pre-clinical curriculum include lectures on the history and nature of the art of healing, lecture courses in ethnology and policies regarding population, field trips in search of medicinal herbs, to further the new movement for natural therapeutics, as lecture course on the physiology of occupation, sports and aviation, six weeks of practical nursing, and practical experience as a worker in a factory or in a farm. This will last four weeks, and be interposed between the third and fourth semester. To the clinical part of the curriculum have been added courses in radiology, natural therapeutics, occupational diseases, treatment of traumatic injuries, child welfare, eugenics, dental diseases, genetics. Two three-month terms as a famulus, between the sixth and seventh and the eighth and ninth semesters, are required also. It is expected from the authorities that the new medical curriculum will be more in accord with practical needs.

### SOCIAL SIGNIFICANCE OF RHEUMATIC CIRCULATORY DISEASES

Rheumatic heart diseases are particularly frequent among young people and these age groups are socially very important. In a survey of the conditions of school children by Koller, 1 to 2 per cent organic heart diseases were found—and they were almost exclusively of rheumatic origin in this age. The examination of persons for military service in Germany in 1935 showed disorders of heart and circulation (except varicose veins) in 7.6 per cent of all examined persons, 56 per cent of whom were unfit for military service.

Statistics of rheumatic diseases are very insufficient as has been stated by S. Dietrich, II Medical Clinic, Berlin, in his book "Rheumatische Kreislaufschodigungen". The following table shows certain diseases as the cause of invalidity in Germany in 1935.

| Disease                                                              | Percentage of Invalidity | Average Age at Invalidity (Years) |
|----------------------------------------------------------------------|--------------------------|-----------------------------------|
| Diseases of the Heart and Circulation                                | 31                       | 56.8                              |
| Tuberculosis                                                         | 10                       | 42.75                             |
| Acute rheumatic fever, Chronic rheumatism, Gout, Muscular rheumatism | 7                        | 57.6                              |

A disease of the blood circulation is the cause of death of every ninth person deceased between 30 and 44 years of age, and of every fourth person deceased between 45 and 59 years of age. About 25 per cent of all persons in Germany die from some disease of the heart or blood vessels. The conclusion from several facts concerning the incidence of rheumatic circulatory disorders is the following: that about one-third of the mortality from circulatory diseases or about one-twelfth of the total mortality in Germany has been caused by rheumatic troubles. Dietrich considers an early treatment of rheumatic diseases of heart and circulation one of the most important medical tasks to be achieved. A complete cure of rheumatic carditis is undoubtedly possible.

## INDIAN MEDICAL NEWS

(From Our Own Correspondent)

### REPORT OF THE CALCUTTA SCHOOL OF TROPICAL MEDICINE, 1938

This report, published in August, is, as usual, an interesting document indicating as it does some of the recent researches that have been undertaken in this country. Many of the enquiries have not yet been completed while in some tentative conclusions have been drawn. Investigation in leptospirosis in 10 cases revealed that the infecting organism belonged to the same serological type as *L. icterio-haemorrhagiae* but the serum in other cases did not give any significant reaction with classical strain but agglutinated a rat strain recovered from a field rat in a high dilution. This emphasises the need that the agglutination test should be made not only with the strains responsible for the case in the locality, but also with the representatives of the different serological races isolated in the various parts of the world. The school entrusted the preparation of an antileptospiral serum to B. C. P. W. Co. Ltd., and it may be stated that they had already succeeded in immunising a horse against these strains. It is hoped that this serum will soon be put on the market. Investigations into the aetiology of epidemic dropsy reveal that adulteration of mustard oil with Argemone oil may be a possible factor, in that the latter produced typical symptoms of epidemic dropsy. But the matter is still sub-judice. Spectrophotometric and chemical methods have been recently introduced in the school in assaying the vitamin content. Some of the other researches and conclusions are: (1) Indians suffering from gastro-intestinal disturbances, hyperchlorhydria is a more common error than hypochlorhydria. (2) There is very little association between the degree of anaemia and duration of kala-azar infection. (3) The highly refined liver extracts which contain the haemopoietic principle that is deficient in pernicious anaemia, are inactive in many cases of tropical macrocytic anaemia which subsequently respond well to treatment with crude liver extracts. (4) 2 grs. of santalin is as effective as the usually recommended dose of 5 grs. (5) The treatment of hymenolepis Nana with gentian violet has been continued with some success. (6) A number of drugs were tried for the treatment of filariasis, namely, cuprochine, curprion, cibinol, surfen, M. & B. (693), etc., but none of them had any parasitocidal effect. (7) It has been shown that there exists a close relationship between the inorganic phosphates of blood and the carbohydrate metabolism. The phosphate content of the blood was found to be decreased after ingestion of glucose and also after injection of insulin. Above are some of the more outstanding conclusions embodied in the report

and your readers will be well advised to go through it for greater details.

### MALT INDUSTRY IN INDIA

There was an interesting lecture on "indigenous malt products" by Mr. Narayanayya, Research Officer, Agricultural College, Coimbatore on Aug. 1, 1939, and he, according to the report in the *Hindu*, said in the course of his address that "the average children of the poorer classes did not get enough breast-milk for their calorogenic requirements. By virtue of their easy assimilability, malt foods were specially suitable for infants, and could effectively supplement breast-milk. However, the high cost of the imported malt food precluded their use by the poor classes, among whom malnutrition was prevalent. India imported annually 84 lakhs worth of these products, the share of Madras Presidency alone being 18 lakhs." Describing the work done under the scheme to find a better use for the enormous quantities of cholum grown in this province the lecturer added that it was now known (1) that technique of malting could be developed to lower the fat content of cholum malt and to yield a higher 'extract'; (2) that cereals like wheat, ragi and paddy could be malted both on a small scale as well as on a large; (3) that vacuum packing preserved cholum malt for at least six months; (4) that foods of the type of ovaltine, malt extract and Benger's Food could be prepared from cholum malt; and that (5) the blending of cereal malts with and without milk powder offered a great scope in the development of malt industry. Subsequent work demonstrated the possibilities of use of cholum malt in the preparation of malted biscuits, sweets and salted, malt bread, malt cakes etc., which were more palatable and more digestible. The next line of work was the development of cholum and ragi malt industry in the preparation of malt extract which served as a vehicle for the administration of drugs in a palatable form, i. e., in the preparation of therapeutic malt products.

### IMPROVED TECHNIQUE OF GHEE MAKING

Investigations of far-reaching importance to the ghee trade are being conducted at the Imperial Dairy Institute, Bangalore.

A popular method of manufacturing ghee is by curdling of milk by natural souring. This, it has been found, produces at times an undesirable flavour and form under village conditions; the resulting quality is also poor. An attempt has, therefore, been made at the Institute to find out if the addition of citric acid would provide an alternative method. The technique aimed at is so simple that it can easily be adopted anywhere. Citric acid is used for the coagulation of fresh milk. The product is churned and ghee obtained by heating the butter. The ghee has a good flavour and is much superior in quality and quantity to that made by country method. The problem is still being investigated.

Investigations on making ghee direct from cream are also being continued.

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### Composition Data

Scott's Emulsion is based on a biologically standardised cod liver oil, the Vitamin A and D potency of which is fully preserved. The Emulsion also exhibits Calcium and Phosphorus in the form of tonic Hypophosphites. (Note that the calcium is directly utilised in the presence of Vitamin D.)

### Common Indications

Scott's Emulsion is successful in cases of: Anaemia; Amenorrhoea; Avitaminosis; Bronchitis; Catarrh, bronchial and gastric; Coughs, Cold, and Croup; Chlorosis; Debility; Dentition; Eczema; Fevers (after); Influenza; Lupus; Marasmus; Nutritional disorders; Parturition (after); Osteomalacia; Phthisis; Psoriasis; Pyorrhoea Alveolaris; Scrofula; Rickets; Strumous conditions; Ulcers; Tuberculous complications; Whooping Cough.

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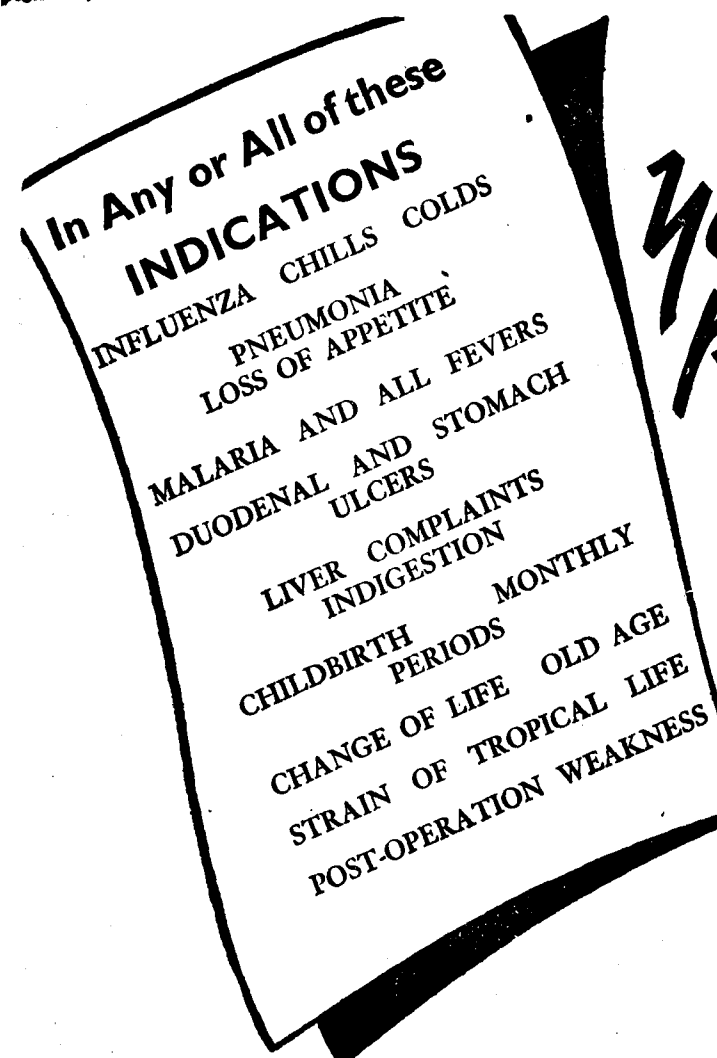
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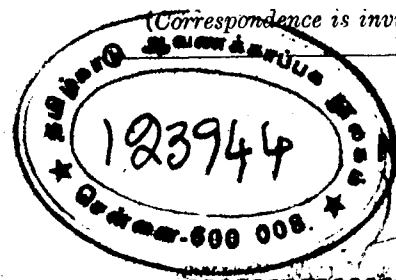
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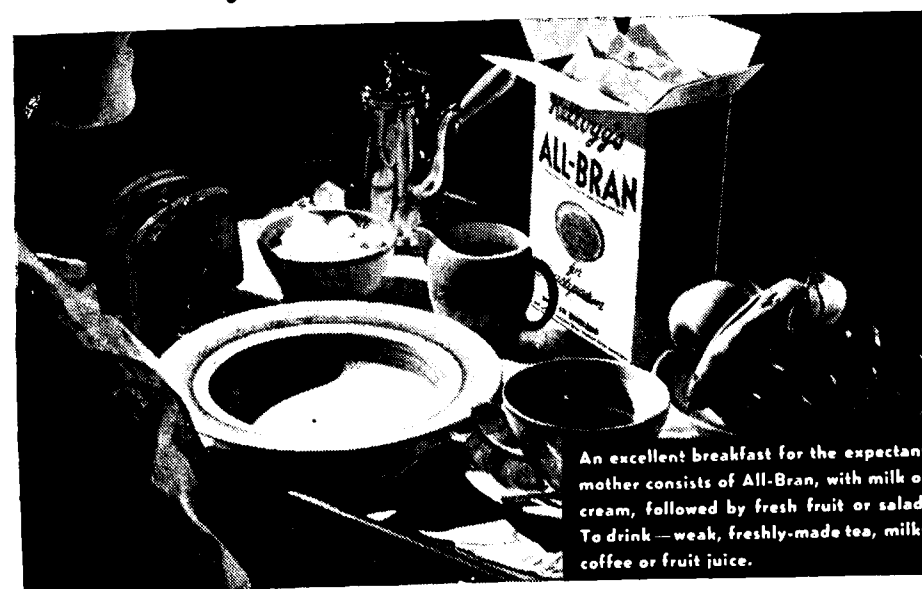
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PRINTED BY MR. A. RANGASWAMY, AT THE PROGRESSIVE PRINTERS, NO. 2/17,  
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