War Medicine Series

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DYSENTERY AND DIARRHOEA IN WARTIME

BY

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Treatment of Bacillary Dysentery

The first thing to be said about treatment is that the sooner the disease is diagnosed and treatment commenced, the better. At the beginning of the last war patients were given opium and opium pills—a pernicious practice; but when the patient is being transported over long distances morphine or tincture of opium may be given to keep him quiet, and to stop the violent peristalsis, dysuria, and tenesmus. “Saline treatment,” introduced by Army surgeons 300 years ago, has still the greatest vogue. Large doses of the salts are given. Some like senna, others rhubarb; but I myself believe that there is nothing better than sodium sulphate, 1 drachm every three hours for the first forty-eight hours and then four times daily until the stools become faeculent, because at first they contain no faeces at all.

Diet is most important. Do not starve your patients. There is no sense whatever in that system, for the upper small intestine is still functioning, and so is the stomach. Not much milk must be given, because it forms into clots and curds, which are passed with difficulty. They should have large amounts to drink—four or six pints of lemonade or barley-water—to replace dehydration. They can be fed on junket, jellies, beef-tea, and chicken broth. In America ice-creams are in vogue. Do not attempt to give anything solid like milk puddings or sago until the stools contain faeces. Above all things, arrowroot is suited for the treatment of bacillary dysentery. The natives of the Pacific have treated dysenteries with arrowroot since time immemorial. The addition of brandy is stimulating and comforting.

Recently it has been shown that blood transfusion is an excellent measure in desperate cases, and has recently been employed in a big outbreak of dysentery in Poland. The other measure is intravenous hypertonic saline with 5% glucose. Then we come to antidysenteric serum, about which a number of doubts have recently been expressed. The real trouble arises from the fact that the antidysenteric serum of the last war was polyvalent, but now we have a concentrated form of Shiga serum. The cases that require serum treatment are Shiga infections. You do not have to wait until your laboratory diagnosis has been perfected before acting, but serum must be given early and in sufficient amount—i.e., 80 c.cm. intramuscularly to adults. The muscles of the buttocks are suitable for the purpose, also the adductor muscles of the thigh, but it should not be injected subcutaneously. Many a soldier has told me he would rather put up with his dysentery than with subcutaneous serum. Serum sickness, which can be obviated by injections of adrenaline, commonly ensues. In severe toxic cases serum can be given intravenously in doses of 50 to 60 c.cm. Anaphylaxis is apt to occur in bacillary dysentery with antidysenteric serum, especially if the patient has been sensitized to it previously, but not many die from this cause. But please do not give everybody antidysenteric serum indiscriminately. The majority of cases get well without it. I think it is a very good rule to say that, if the patient is very toxic, if his pulse rate is about 120, if his temperature is 103° or 104° F., and if the stools number more than twelve in the twenty-four hours and contain a lot of blood and slime, then antidysenteric serum should be given, but otherwise not.

Chronic bacillary dysentery is not so easy to treat. The best method is irrigation of the large intestine with eusol—1 part to 5 parts water. This can be injected into the large intestine, but some of these cases do not respond at all, and a valvular caecostomy may have to be performed to put the bowel at rest. The whole problem of treatment of the bacillary dysenteries, in both the acute and the chronic stages, has now been transformed by the introduction of sulphaguanidine, a sulphonamide which is specific for dysentery bacilli and is given in doses of 6 to 9 g. daily for the first five days; reports on this subject are now coming to hand. It really seems as if this drug was specific, especially for Shiga and Flexner infections. It is also curative in the chronic form.

The best way of diagnosing bacillary dysentery is to examine the exudate under the microscope. Clinicians used to say that if you had to wait for the laboratory to give the correct diagnosis the patient would have either recovered entirely or have succumbed. Nevertheless, a pretty accurate forecast can be made from a study of the cellular exudate. Sigmoidoscopy is a useful adjunct.

In chronic bacillary dysenteries the whole of the mucous membrane is covered with granulation tissue. The normal folds have disappeared and the walls are thickened and stenosed. Therefore stenosis of the bowel is a feature of the post-dysenteric state. This occurs after bacillary dysentery; never after amoebic. Sometimes masses of granulation tissue form and are known as pseudopolypoid, similar to that of chronic ulcerative colitis.

Amoebic Dysentery

Great emphasis has been laid on amoebic dysentery, but it is not anything like the danger it is stated to be. Dobell has claimed that *E. histolytica* infection is the most easily controlled of any in man. This protozoan produces ulcers of the large intestine, but apparently it is a question here of a struggle between the processes of repair and those of destruction. The *E. histolytica* is a parasite of the submucous layer of the large intestine; it lives mostly beneath the surface and not above. It may be looked upon as a sort of intestinal “rabbit,” and the first lesions visible are little superficial abscesses on the mucosa which indicate its burrows. If one of these be incised there exudes some grumous material containing active *E. histolytica*. Although these lesions are scattered throughout the length of the intestine, the intervening mucous membrane is perfectly normal. A familiar form of lesion is known as the “sea anemone” ulcer. It will be observed that fissures traverse the bowel, and there are blood-stained sloughs hanging down like seaweed. Sometimes multiple ulcerations or solitary ulcers of large size are present. The clinical phenomena of amoebic dysentery are produced by local lesions of the intestinal canal. Thus the two main dysenteries are as unlike and apart as the two poles.

There is no mistaking the *E. histolytica* for anything else when it is seen under the microscope. The difficulty always is that it may not be present in the particular portion of the stool when examined. This parasite does not live very long outside the human body—in the tropics perhaps two hours, in this country perhaps three or four. It is most important to examine the stools as quickly as possible. In a fresh specimen the active *E. histolytica* moves, as Dobell says, “like a slug at express speed.” Each individual has the characteristic nuclear granular endoplasm and clear marginal ectoplasm, and it lives upon red blood corpuscles. What the oyster is to the alderman the red blood corpuscle is to the *E. histolytica*! Sometimes as many as 60 or more may be found inside one individual. Then there are in the exudate Charcot-Leyden crystals. But do not jump to the conclusion that these crystals necessarily mean amoebic dysentery: they are found in all kinds of infections of the large intestine, especially ulcerative colitis, but they are certainly common in association with amoebic dysentery.

Amoebic dysentery may produce astonishing situations. In 1937 a man who had had an appendicostomy wound since 1900 was seen. He had been washing himself out daily with 7 pints of fluid, but in the stools one *E. histolytica* was found. He was then rapidly cured by anti-amoebic treatment and the wound rapidly healed. Amoebic dysentery may present

amazingly long incubation periods. There was an old lady aged 74 with a liver abscess for which she had been operated upon. When the stools were examined she proved to have a heavy infection with *E. histolytica*, and the organism was also present in the pus from the liver abscess. On probing into her history it was found that the only time she had been abroad was when, as a bride aged 17, she went out to Bombay for ten days and had had some intestinal disturbance there. The incubation period in her case was approximately fifty-seven years! Another case was that of a man who was playing tennis at Wimbledon and went down with acute dysentery, which was thought to be ulcerative colitis. I could not find the amoebae in the stools; but his intestinal canal was full of typical ulcers, and in scrapings made from them *E. histolytica* was discovered in large numbers. He had lived in Mauritius till he was 4 years of age, and was then 27. Not very long ago a man was sent to me as a case of carcinoma of the rectum. He had a large fungating rectal ulcer. During the last war he had had dysentery on the Somme in 1916, but had not noticed anything until, after eating some fried fish, he passed blood and mucus in the stools and was taken to hospital, where the ulcer was scraped and found to contain *E. histolytica*. In other words, the disease had taken from 1916 to 1937 to develop.

The chronic stage is different from the acute. These people generally suffer from flatulency, from abdominal pain, and from periods of constipation alternating with diarrhoea. The cysts which are found in the chronic stage have four distinctive nuclei; they stain with iodine, which shows up the chromatoid body in their interior. But the cysts of *E. coli* are also found in the stools, and it is necessary to differentiate one from the other. Sometimes four nucleated cysts are present, but the patient does not show any symptoms of acute disease at all. These cases are known as "cyst passers" or "cyst carriers." In India the cyst-carrier rate varies from 60 to 15%, in the United States about 10%. There was a great epidemic of amoebic dysentery in Chicago a few years ago—one of the most remarkable epidemics of all time. Two big hotels were implicated. It was proved afterwards that *E. histolytica* was present in the drinking-water. One of the drains was infiltrating into the main water supply. Thus it was proved that amoebic dysentery is a water-borne disease. The incubation period is anything from 2 weeks to 120 days. We know that the *E. histolytica* may lie dormant for a long time inside the body without producing any outward signs of disease. The chief point is to examine the stools in as fresh a state as possible.

The entry of *E. histolytica* into the bowel is first effected by proliferating in the intestinal mucus. Thence they penetrate into the submucosa and produce typical flask-shaped ulcers. This process is entirely different from that of the bacillary disease.

Amoebic dysentery may give many kinds of clinical pictures. The great majority of the cases are chronic. Often they cannot be diagnosed correctly without the aid of the sigmoidoscope. The ulcers are easily seen just inside the sphincter, and *E. histolytica* can be demonstrated under the microscope in preparations from the lesions. In chronic amoebic dysentery ulcers appear like small craters, and it is known that this parasite may produce extremely minute lesions. One investigator once cut 250,000 sections of a bowel, and in one section only he found a nest of *E. histolytica* in the submucosa. There must be a great many people with these minimal lesions inside them. Many so-called carcinomata of the rectum turn out really to be instances of localized chronic amoebic ulceration.

Infections of the skin with *E. histolytica* have been known since 1892. Many instances of what is known as "synergistic gangrene" of the abdominal wall after operation are really due to cutaneous amoebiasis at the site of a colostomy. Instead of the colostomy wound healing up, the whole abdominal wall becomes gangrenous and amoebae in numbers can be found in the exudate. I recall one case of amoebic infection of the skin in a soldier who had a rectal abscess which had burst and spread, involving the whole of the lower rectum and both buttocks. He was given 8 grains of emetine, when all the sloughs cleared away and he is now perfectly well. He was an old soldier from India, who had never had "dysentery," though cysts of *E. histolytica* were present in the faeces.

Treatment of Amoebic Dysentery

In the treatment of amoebic dysentery, when the disease is acute and the amoebae are present and active in the stools and there is blood and mucus, emetine is the drug of election; but emetine is not everything that has been claimed for it. It kills off the vegetative *E. histolytica* in a strength of one in five million; but it does not entirely eradicate the disease, and it causes these organisms to scuttle away into their burrows.

The dose of emetine for an adult man is 1 grain a day, or 0.04 mg. per kilogramme of body weight. It is given for ten days and no longer. Let me beg of you not to overdo the emetine. This is a toxic drug, which may have an effect on the myocardium, and children may die of acute heart failure from overdosage. It has an affinity for the nerve endings and may produce neuritis, with muscular wasting resembling progressive muscular atrophy. In others it produces desquamation of the skin and cracked nails. It is not wise to say to one's patient, "I will give you 10 grains of emetine and you will be cured." Some people adopt the principle of giving 10 grains a month and repeating this dosage indefinitely. This is bad practice. When there are cysts in the stools you will not get rid of them with emetine therapy by the injection method, but by the oral route by emetine-bismuth-iodide, popularly known as "E.B.I." This has been shown to exert a direct action on the cysts. It is a red powder, and 1 grain is given as the first dose and 2 grains as the second. It is not necessary to use bigger doses than 2 grains. The total amount necessary to cure amoebic dysentery is about 18 to 20 grains. I have effected permanent cures with 18 grains of emetine-bismuth-iodide, and have now treated over 1,000 cases, only three of which have been known to relapse. Many of these cases have been watched for a period of ten to fifteen years.

The drug must be given last thing at night and be preceded by a sedative, the best being luminal. I usually prescribe 1 grain at 8 p.m., another 1/2 grain at 8.30 p.m., and the E.B.I. at 9.30 p.m., not allowing my patient to take anything solid by the mouth after 6.30 p.m. By this means cysts are got rid of; but that is not the whole story. It is not enough by itself to follow this regime, and it should be aided by quinoxyl (anayodin in America and chiniofon in the *British Pharmacopoeia*). These preparations are quinoline-sulphonic acid. When this substance in 2% solution is injected into the bowel in the form of retention enema it heals up the ulcers wherever it comes into contact with them in the rectum and lower sigmoid. A preliminary wash-out of 2% sodium bicarbonate is given in the morning and then 7 oz. of the quinoline solution is run in; the patient should absorb and retain it for six or eight hours. If you give emetine-bismuth-iodide at night and quinoxyl during the day you can cure almost every case. At the end of the ten days' treatment the patient may eat any diet that he may prefer. It takes from ten to twelve days to heal the amoebic ulcers, and, if the treatment is carried out and the patient well nursed, no great inconvenience or undue risks are involved.

A great many other drugs are recommended for amoebic dysentery. Their name is legion. But I do not think any of them approach in efficacy those of which I have spoken. You can be quite certain that when the patient is once cured of his amoebic dysentery and all ulcers have disappeared, further relapses will not occur.

The Minister of Health has appointed a central technical committee to advise on the organization of mass radiography; it will be a subcommittee of the Minister's Standing Advisory Committee on Tuberculosis. Membership is as follows: Chairman, Mr. T. Lindsay (Ministry of Health), Dr. A. E. Barclay, Dr. G. Lissant Cox, Dr. J. Ferguson, Surgeon Commander W. Fitzpatrick, Dr. F. R. G. Heaf, Dr. G. Jessel, Major P. Kerley, Brigadier D. B. McGrigor, Dr. S. Cochrane Shanks, Dr. Norman F. Smith, Wing Commander R. R. Trail, Dr. Harley Williams (secretary), with representatives of the Ministry of Labour and the Department of Health for Scotland. The first sets of apparatus for mass miniature radiography are expected to be available before the end of the year, and the remainder of the 25 already ordered will be in use during 1943. The committee intends to provide a complete scheme for the full employment of these units throughout the country, including adequate technical standards. It has already considered the training of personnel and the allocation of the first sets of apparatus.