SOME POINTS IN THE TREATMENT OF ENTERIC FEVER.

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In response to an invitation from the Editor to write something for this particular number of the Journal, it occurred to me that some notes on a few special points in connection with some well-known disease would probably be more acceptable than the usual article on text-book lines.

I have selected the subject of Enteric Fever for another reason—namely, that it is a malady in which nursing plays such a very important part. When a patient, after three weeks or more of serious illness, ultimately recovers, the nurse, on whom such a great part of the strain of responsibility has fallen—if only for the reason that she is in close attendance for so many long hours at a time—may well feel that the result is to her credit.

But I have often been told by nurses who have not received part of their training in a fever hospital, where some attempt is made not only to employ nurses but also to teach them, that enteric fever is difficult to understand because there are so many *different varieties* of the disease, that an attack in one patient is quite different from what they are told is the same disease in another. This difficulty is quite a real one, but it is due, I think, not so much to the disease itself as to the fact that its pathology has only comparatively recently been put on a sound basis (owing to investigations in the clinical laboratory), and that the results have not yet got into most of the text-books. For this an incubation period of about ten years seems to be the fashion!

Let me, then, briefly describe the nature of the disease itself before going into any question of treatment. Enteric fever is, of course, due to infection of the system with the bacillus typhosus, which is usually swallowed, or may be more rarely inhaled. We need not go into the large question of where the germ usually comes from, but we will start with its arrival in the system of a susceptible person.

It used to be taught that the organisms went straight into the small intestine, where they lodged in the little glands known as Peyer's patches, and gave rise to inflammation, and ultimately ulceration in their midst. Poisonous products, or toxins, were produced at the site of the ulcers (and also in the spleen), which, when absorbed into the system, gave rise to the fever and other constitutional signs which are so well known in an attack of enteric fever. The point about this view is that it is the ulcerated intestine that matters.

Now this is not quite true. As a matter of fact, the bacillus can be found *in the circulating blood* from the beginning of the attack in the vast majority of cases, and it is therefore distributed all over the body from the first. By degrees it disappears from the blood, and is found in the urine, so that in the third week almost all patients are passing typhoid bacilli in the urine daily, and the organisms are very seldom found in the circulating blood after the second week. It is true that the intestine is affected by ulceration in the manner described, but a curious fact is that if we make cultures from the whole length of the intestine in a patient who has died of enteric fever, we find that the bacilli are present in far greater numbers at the commencement of the intestine than at the site of the ulcers themselves. The bile is full of them, so the probability is that they are picked out of the blood partly by the liver, to be discharged into the intestine through the bile, and partly by the kidneys, for excretion in the urine.

We see at once that the ulcerated intestine is not the only part of the body that we have to think about in treating a case of enteric fever, and it is best to regard each attack as a battle between the toxins secreted by the circulating bacilli and the antitoxins manufactured by the patient's own white blood corpuscles.

Now comes the next point. If we do not irritate the intestine, the patient does not get *intestinal symptoms* at all in the vast majority of cases. If we did not know from previous experience, it would be very difficult to say in what part of the body the toxins are being manufactured. It is true that in bad cases we get abdominal distension and diarrhœa, but the point for both physician and nurse to remember is that in any case where these symptoms arise we have to ask ourselves whether they may not be due to our own mistakes in treatment—and they very often are.

Our aim, then, must be to combat the poisoning of the whole system, whilst taking care not to irritate the intestinal ulcers.

How does nature deal with a general infection—*i.e.*, where the circulating blood contains bacilli or their toxins? By the leucocytes, or white blood cells. These sometimes swallow up and destroy the bacilli—the process known as phagocytosis—or, more commonly, they manufacture an antidote—antitoxin—to the poison.

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