the right iliac fossa; but if we have much ulceration going on we can scarcely avoid having pain, especially if the ulcerative process reaches the serous coat of the bowel, which is here the sensitive membrane, the same as the pleura is the sensitive membrane of the lung, but I can readily believe that in some cases when the lesion is more of a general inflammation and superficial, more of an enteritis pain may be absent.

As to the rash of typhoid, it is an unreliable symptom. I have seen it so marked that it resembled rubeola, save in the crescentic outline of the groups, whilst on the other hand, careful watching failed to reveal a single spot, and in these cases where the rash is typical, the symptoms are generally so well marked, that one does not require the appearance of the eruption to confirm the diagnosis. I think it may be stated generally, that it is in the severer forms of typhoid, the rash is most typical, whilst in mild cases it is most frequently absent.

Another point worthy of attention, is whether or not the typhoid poison may not produce some other disease. In many cases where typhoid appears to be a particularly severe type, the manifestations in the nervous system are also very severe, and perhaps the only marked indications of the disease. If we take those cases, where after the first day or two of illness, coma vigil, or acute delirium marks its advancement, we will find there is little tendency to severe abdominal lesion or symptom; although the patient may linger on for weeks, early death is the rule in these cases. Again, everyone must have noticed the special liability to severe pneumonic complications, where the type of the disease is severe; and this pnumonia also appears early, frequently terminating the case before the abdominal disease has progressed very far. Those cases where pneumonia comes on lateas a pure sequela - are in my experience, rarely well marked cases of typhoid, and in many of them I think there is room for doubt as to the correctness of the diagnosis of typhoid. I remember a case of consolidation of one lung, coming on at the sixth week, during a typhoid and terminating the case, but on post-mortem examination the consolidation proved to be not pneumonic, as thought, but tubercular, and limited to one lung. I do not wish to state that pneumonia cannot be a sequela to typhoid, but that it is more frequently an early

than a late complication. Again I believe it is quite possible to have a septicæmia arise from typhoid. I mean a septicæmia similar in character to that due to direct pus infection, and am of the opinion that many lingering relapses in typhoid are from this cause. We know it is by no means rare to find a suppurating mesenteric gland near to a typhoid ulcer in the bowel, and there can be no reason why pus there should not enter the circulatory system. Again, where ulcerative endocarditis follows upon the disease there is generally evidence of irritating or septic material having entered the blood vascular system.

As to the lesion of softening and pulpy degeneration of the spleen, this is found in many other diseases besides typhoid, and in the latter is often absent; softening of the spleen is the result of high temperature, and should the temperature be low throughout, little change in the spleen need be looked for; it is one of the earliest organs to undergo pyrexial softening, and I do not think it is more predisposed to such change in typhoid than in many other diseases characterized by continued elevation of temperature. It is claimed by some that such tissue change can be entirely prevented by the continued administration of antipyretics, but upon the subject of antipyretics light has yet to dawn; it is a simple matter to reduce the temperature in any disease, but quite another thing to know if such reduction is beneficial; those, who in the administration of antipyretics have in mind the lowering of the temperature only when its continued elevation threatens the integrity of tissue, have grasped the great therapeutic principle underlying their employment, and I would question the soundness of that principle, commonly practised, which interperets the elevation of temperature as fever and the lowering of temperature as its reduction. If diseases of the zymotic type are changes involving the oxidation of morbific matter, I cannot but think that the lowering of temperature may lead to the storing up of that material and in the end to a greater pyrexial increase.

Thus would I outline some of the difficulties which beset us in our studies of typhoid fever, confident that this disease so common in its occurrence, is less thoroughly understood than many other diseases of less frequency, and as Charcot devolved out of those cases commonly called ataxic