Gowers (*Diseases of the Nervous System*, vol. ii.) alludes to similar cases, but mentions that, as diphtheria is known sometimes to accompany typhoid fever, so a paralysis in reality of diphtheritic nature may be observed as a sequence of such a compound attack; and the question may be raised whether H. did not have diphtheria. There is no proof that he did have throat affection of any kind; and, on the other side, he was, during the whole time, under skilled attendants in a hospital, and nothing was ever noticed or complained of which led to an examination of the fauces. It may, therefore, I think, be safely held that no diphtheria complicated the case. I would also recall, as strongly corroborative of the same view, that there was no disturbance of accommodation, and no albumin in the urine.

Although actual observations are not very numerous showing presence of the lesions of peripheral neuritis, and the absence of change in the spinal centres in cases in which post-typhoid paralysis has existed, yet they are sufficient to substantiate the occurrence of such a disorder. Indeed, in the light of the somewhat remarkable observations recorded by Pitres and Vaillard (*Revue de Médecine*, t. v., 1885), we may well be surprised that the clinical evidences of neuritis are not more frequently met with. These writers made careful and extensive histological examinations of peripheral nerves in various parts of the body in cases of typhoid fever which had proved fatal from various causes, but in none of whom there had, at any time, been evidences of nervous lesion or disorder. The result was, in every case, to find microscopical evidence of well-marked changes in the structure of most of the nervous trunks and their branches, whilst the actual nerve-roots and the spinal centres remained unaltered. And the question is raised, "How frequently in reality does such neuritis occur?" In the examples recorded there had been nothing from a clinical standpoint to suggest its existence, and they were selected quite by chance. Does it occur both in fatal cases and in those mild or more severe cases which end in recovery? We are led to think the latter possibly true, owing to the frequency with which more or less marked evidences of sensory, motor, or trophic disturbances are met with as sequences of typhoid fever.

Is this neuritis set up by the elevation of temperature? or by the general derangement of nutrition? Or may the typhoid poison act directly upon the nervous fibres, irritate these, and ruin their structural integrity—determine, in fact, a parenchymatous neuritis? This neuritis, it is stated, attacks not only the superficial branches which innervate the skin, but also the larger and deeper trunks. May it not, therefore, be that the disturbances in them are to blame for many of the symptoms generally observed in typhoid fever: cutaneous hyperæsthesia, muscular hyperæsthesia, wandering or localized pains in the limbs, etc., symptoms which are often attributed, perhaps rather gratuitously, to irritation of the