VII

SOME PROBLEMS IN THE HISTOPATHOLOGY OF GENERAL PARALYSIS OF THE INSANE

By Dr. A. J. GALBRAITH

DISCUSSION

Dr. Meyer thanked Dr. Galbraith for his excellent review of the state of affairs in the neuro-histology of G.P.I. The review was ungarished and Dr. Galbraith had not fallen into the oft-repeated mistake of workers in a particular field of claiming that that particular field was far advanced and there were important results to report. He had emphasised that although much was known, still very little was known about the really fundamental events leading to these severe lesions in the brain. He had referred to some interesting theories on the damage to the nervous tissue itself, which was such a marked feature in G.P.I. process as distinct from the rest of cerebro-spinal syphilis. He referred to the vascular factor, although present evidence was not sufficient to enable it to be said that this factor is responsible in the majority of cases.

With regard to the Von Braunmuhl theory that the spirochæte introduced a colloidal change in the cerebral tissue, resulting in degeneration, at present this was no more than a theory and it was not easy to believe that this was really the explanation of what happened to the tissue.

Reference was made to the iron reaction, and from the practical point of view this was an important advance, but what did it mean as evidence of pathogenesis? Was it only due to the activity of the spirochæte? If so, why was the same iron reaction not produced in cerebral syphilis and meningo-cerebral syphilis. The iron reaction might be the starting-point for that correlation between the bacteriologist and the neuro-histologist that might lead to further information about the pathogenesis of the G.P.I.

An interesting phenomenon was that of the involvement
of the hypothalamus. Experience showed that there was much marasmus or other change of lipoid metabolism without any nutritional explanation. It was possible that these changes had something to do with hypothalamic lesions. Clinically it was known that in some cases there were disturbances in the genital system, many cases becoming impotent. One naturally thought of tabes, but it was not always present. There was the possibility that such phenomena could be occasionally correlated with hypothalamus lesions.

There was a great opportunity and ample material at Horton at the disposal of Dr. Galbraith for a systematic attempt at correlation of clinical and pathological findings, and Dr. Galbraith was to be congratulated upon the work he was carrying out.

Dr. A. Beck said that as so much stress had been laid upon collaboration between histology and bacteriology, he felt provoked to say something about the problems of the bacteriologist in research on G.P.I. There had been much interesting work but also much disappointment, and no satisfactory answer to the question of the reason why spirochætes under certain conditions produced brain lesions in a certain percentage of cases.

Attempts have been made to produce brain lesions in animals (rabbits and monkeys) by injecting syphilis spirochætes into their brains or great cistern, and the greatest difficulty was experienced in making the spirochætes persist in the brain for longer than a few weeks. It can be said that nobody has yet succeeded in reproducing the human neurosyphilitic lesions in animals. In view of the great susceptibility to even minor degrees of heat of the syphilis spirochaæte the suggestion arises whether the resistance of the normal brain against the spirochætal invasion is not due to its relatively high temperature when compared with other organs. In his recent paper to the Society Professor Bessemans has shown that such relatively small differences of temperature as were produced by replacing the infected rabbit’s testicle into the peritoneal cavity were already sufficient to inhibit the formation of a chancre in it. The differences of temperature between the brain and other organs (as for instance given in Stewart’s textbook of physiology) would be well within the ranges of temperature which Professor Bessemans used in his experiments.
GENERAL PARALYSIS OF THE INSANE

considering this hypothesis it would be necessary to assume that the brain of paralytics is colder than the normal brain and thus offers a more favourable environment to the spirochætes.

He would like to ask the histologists whether in the pre-paralytic stage of the brain there were any signs of vascular lesions which could account for a diminution of the circulatory volume and thereby for a lower temperature of the brain? He would also like to ask whether in the stage between the secondary stage and the definite outbreak of the disease there were any known histological changes, where spirochætes had been found in the brain parenchyma in this stage and whether there was any link between the known early involvement in the nervous system and the later outbreak?

Dr. Beck, referring to the question of trauma in G.P.I., said this had some experimental foundation. It was known that the spirochætes seemed to have a preference for localisation in traumatic areas, and it had also been shown experimentally by Chesney that, by producing lesion in the skin by burns or injection of irritating material, subsequent intravenous inoculation with spirochætes produced lesions specially in the parts that had been damaged. This had led experimenters to try similar methods in the brain, but so far with no positive results.

Dr. Galbraith, in reply, thanked the Society for the kind reception given to his paper, and especially thanked Dr. Nicol and Dr. Meyer, whose help had made his work possible.

There was much more work to be done and he certainly had plenty of material at his disposal; he only wished he had adequate time. To correlate pathological findings in the brain with clinical examination involved a thorough investigation of the brain which might require months of work. At Horton there were eighty brains of G.P.I. cases awaiting this investigation.

He was interested in Dr. Beck's remarks and wished he could answer his questions. His remarks on thermolability of the spirochætes were interesting and might lead to important findings.

Dr. Galbraith did not know of any definite answer to the question whether the brain showed any circulatory defects which might account for the development of G.P.I., but in
his paper he had remarked on the vascular factor. That was evidence of impaired circulation, and it might be that this vascular factor was responsible for the invasion of the brain and development of G.P.I.

With reference to the question as to what changes in the brain had been found in pre-paralytic stages, Spielmyer had described a case of a patient with syphilitic aortitis who did not show any clinical evidence of G.P.I., but in whose brain there were definite findings of the inflammatory changes of G.P.I. This answered Colonel Harrison's question to a certain extent, and Dr. Galbraith did not feel in a position to make any further remarks at present which would be enlightening on the subject.
Some Problems in the Histopathology of General Paralysis of the Insane Discussion

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