PERIPHERAL NEURITIS IN EXOPHTHALMIC GOITRE.

BY ARTHUR MAUDE.

IN the paper I read at the opening meeting of the London Medical Society in October, I endeavoured to produce evidence, purely clinical, that peripheral neuritis was a common epiphenomenon in exophthalmic goitre. In the discussion which followed, the only notice which was taken of what was at any rate a novel point, was a direct statement from a well-known neurologist that he did not see any reason for supposing that neuritis was ever present. May I, therefore, ask to submit my views to a more highly trained audience of neurologists in these pages?

I was at the time quite unaware of Professor Greenfield's late researches in the microscopical anatomy of exophthalmic goitre;¹ and it is premature to criticise them in detail, as they are still incomplete. I pointed out, however, that no examinations, after modern methods, have yet been made of the peripheral nerve trunks; and I still look to such examination to show changes comparable to the so-called multiple neuritis produced by various toxic agents.

The symptoms I ground my supposition on are:—

(1) The frequent occurrence of "cramp." Dr. Hector Mackenzie,² who first drew attention to it, found it present to some degree in thirteen out of fifteen instances, a proportion I can verify from my own experience. These painful contractions often assume the form of veritable "tetany," both of hands and feet. It is needless, since the publication

¹ "Bradshaw Lecture," 1893.

² *Lancet*, September 13, 1890.

of Ross and Bury's work,¹ to insist that muscular spasm of this form is due to peripheral irritation. As to the nature of this irritation two views present themselves.

(a) Tetany commonly follows removal of the thyroid, and often the injection of mucin into the circulation of animals (cats: Wagner and Hammerschlag).

It is possible, then, that cramps are in these cases the result of late, excessive disturbance of the thyroid, by which some poison, perhaps mucin, remains in excess in the circulation. The cases in which I have found cramp and tetany present have not all presented large goitres. But this proves nothing, for great functional disturbance may exist without great external increase of the thyroid.

(b) Tetany is frequently associated with disturbances of the alimentary canal, from the formation of a direct toxin, which may be separated from the digestive fluids. Precisely similar disturbances of alimentation are prevalent in Graves' disease. In the few cases in which I have been able to follow out this point, cramps were always associated with gastro-intestinal troubles.

(2) Ordinary sensibility is nearly always increased: "Hyperæsthesia is as universal in the sensory as tremor in the motor nervous mechanism" (Grainger Stewart²).

(3) Symmetrical paresis of the legs is the rule. It rarely amounts to complete paraplegia, and is never associated with rectal or cystic paralysis.

(4) The patellar reflexes are nearly always lessened or entirely abolished (Marie): this is my own experience also. On the other hand, Grainger Stewart states that the myotatic irritability varies much, that "the knee-jerks may be sometimes increased, and ankle clonus even be present, more especially in the cases manifesting a high degree of nervous excitability, but on the other hand, the knee-jerk may be entirely absent."

(5) I find varying degrees of numbness, tingling and pains in the peripheral nerves of the legs, a common cause of complaint.

¹ "Peripheral Neuritis," Ross and Bury, p. 398, 1893.

² *Edinburgh Hospital Reports*.

(6) I would suggest that those forms of localised œdema evidently of neuropathic origin, so frequent in Graves' disease,¹ are, in fact, manifestations of peripheral neuritis; comparable as they are to the conditions described by Dr. Bristowe² as hysterical neuritis, in which œdema was occasionally a prominent feature. The comparison is also striking between these localised œdemas and those also seen in beri-beri, which is clearly a "peripheral neuritis."

We have here, I think, all the clinical attributes of peripheral neuritis in an early stage, but in that stage it seems to stop. I have seen no cases, in record or in my own practice, which point to advanced neuritis. And as this arrest is also a feature of the neuritis of diabetes, I have examined all my cases carefully for glycosuria, with negative results. On the other hand, so many of these patients present gastro-intestinal disturbances that it is possible that the irritating poison may be of alimentary origin; while it may be some substance formed in the thyroid, or which should have been eliminated there.

Dr. Judson Bury informs me (August 29, 1893) that he remembers one case in which in addition to the ordinary tumour there was the most marked fibrillation of the interosseous muscles of the hand. This he adduces, quite correctly, as evidence of peripheral neuritis. He kindly adds: "I quite agree with you as to the probable association of peripheral neuritis and Graves' disease."

The point is, perhaps, worth following out by neurologists, both at the bedside and by microscopical examination."

¹ Maude, *Practitioner*, December, 1891.

² *British Medical Journal*, 1892, ii., p. 1098.