

even patches of hyperanæsthesia are very common. Pain in the affected limbs is rare. There is never any atrophy of muscles. The reflexes are increased in cases in which there is much tremor, but diminished often in the paretic cases.

The upper limbs are much more often affected than the lower. The trunk is rarely affected. He draws his cases from the mines of Almaden, and from the felt-makers in Paris.

But in order to study the effect of the poison on the nerves he subjected rats and guinea-pigs to as long a course of mercurial treatment as possible. His experiments were of three classes. (1) Injection of mercurial peptone along the course of a nerve. (2) Injection of the same anywhere in the body. (3) Inhalation of mercurial vapours.

In most cases he got a definite affection of the muscular nerves. This affection was apparently confined to the myeline sheath of the nerves, and therefore differs considerably from the ordinary parenchymate neuritis. It consisted of (1) swelling granulation of the myeline; (2) granular disintegration, followed latterly by (3) atrophy of the myeline. This condition obviously differs from other forms of neuritis, in the fact that only the myeline sheath is affected, the axis cylinder being spared indefinitely. This will explain the great rarity of atrophy in hydrargyrisms, but it is difficult to explain the paresis, at least by the ordinarily accepted views of the functions of the myeline sheath.

The paper is well illustrated by cases and details of experiments.

H. H. TOOTH, M.D.

Chauffard on Sudden Blindness in Lesions of the two Occipital Lobes (*Revue de Médecine*, Feb. 1888).—It is not easy to refer sensory functions to their place in the cerebral cortex, for not only are there few clinical facts to guide us, but experiments on the lower animals are open to obvious difficulties. The localisation of sensory thus affords a great contrast to the localisation of motor functions, which indeed may almost be regarded as a finished work.

In respect to Sight, it has been proved by Allen, Starr, Seguin, and Exner, that bilateral corresponding *hemianopsia* results, without any other sensory or motor symptom, from destruction of part of the occipital lobe. Cases have also been recorded in which in General Paralysis *total blindness* has been noted corresponding with lesions of both occipital lobes. Pflüger relates that a young man received the discharge of a gun in the occipital region, causing immediate blindness and ultimately double optic neuritis.

He died, and at the autopsy grains of lead were found scattered in both occipital lobes.

The author gives four cases, three of which are taken from Berger and Bouveret, in which sudden complete blindness was associated with lesions of these lobes. In his own case, that of an old man of seventy-four, there were three hæmorrhages, one, on the right side, occupying the greater part of the occipital lobe; and two in the left hemisphere. One of these lay in the external capsule, but the larger clot was contiguous to the *pulvinar*, at the very point where, according to Wernicke and others, the fasciculi of the optic tract leave the central ganglia. From this point they pass outside the hinder limb of the lateral ventricle, and finally reach that portion of the cortex which forms the cuneiform lobe and first occipital convolution. In the three other cases, also old men, the occipital regions were affected by softening, from thrombus or embolism. This association of symptoms and lesions accords well with the experimental results obtained in animals by Ferrier and Yeo.

Dr. Chauffard considers that in his case, as in others, simple hemianopsia must have preceded the total loss of sight. Hemianopsia may be often latent or undiscovered. Blindness is thus the result of two seizures on two separate occasions. After the first seizure (hæmorrhage in the left hemisphere) the uninjured centre doubtless took on by substitution more or less of the functions of the other. After the second hæmorrhage (in the right hemisphere), substitution became impossible. It is quite exceptional for the two occipital lobes to be attacked at once. The question arises whether hemianopsia, due to cortical lesion, tends by extension to the companion centre to pass into anopsia. If so, the symptom is one of serious import; for, according to the present author, cases of cortical anopsia invariably prove fatal within a short time. We may note, finally, as diagnostic features, that the blindness is complete and sudden, that "optical memory" is retained, that the fundi are normal, and, what is important, that the pupils are equal, moderately *dilated* (not contracted as in uræmic intoxication), and preserve their reflex excitability to light.

Spillman and Parisot on Peripheral Injury and Tabes (*Revue de Médecine*, March 1888).—In answer to the enquiries, "Can peripheral injuries be the starting-point of Tabes? Are they a sufficient cause?" the authors relate a series of cases in which locomotor ataxy followed the infliction of various injuries. It is not difficult to believe *à priori* that even a small lesion of a

part of the sensory system might lead to an affection generalised over the whole of that system; although it is impossible to assert without evidence from morbid anatomy, that is not at present forthcoming, *how* that generalisation is effected. But, in their opinion, clinical evidence establishes the fact.

The interval between the injury and the first symptoms of tabes varied from a few weeks to as much as eighteen years; and it is worthy of note that, in five out of the fourteen cases quoted, nervous heredity, alcoholism or syphilis were stated to be present. Former writers have noticed an occasional connection between injuries and locomotor ataxy; and Erb (quoting Schulze) cites cases in which it has followed fracture of the thigh or a fall on the abdomen. Petit considers that wounds at a distance "hasten its development," and others have counted *injuries* in a general way as among the causes of tabes. The present writers carry this view much further. Given a personal or hereditary predisposition to nervous affection, they hold that, besides fractures, many slight injuries, such as sprains or wounds of the feet, or even operations in dentistry or for cataract, may be sufficient determining causes. Again, the first symptom of the tabes arises always, in their view, in the injured region. After injury to the left foot, the symptoms commenced after five months with lightning pains in the same limb; after contusion of the abdomen, with gastric crises, two months from the date of injury. This circumstance will often afford a guide in the history of each case to the original exciting cause. Finally it is allowed that diatheses—alcoholism, syphilis or rheumatism, may exert in some of these cases a predisposing influence.

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Raymond on Sweating of the Face (*Arch. de Neurol.*, Jan. 1888).—"The first case recorded is that of a man, aged 31, who noticed in 1880 that sweating of the right side of the face always appeared two or three minutes after he had taken food, and ceased when he left off eating. The sweating extended on to the neck and as far as the shoulder. Hot food induced it most rapidly; movements and the idea of eating had no effect; all other symptoms were absent, except that the right pupil was dilated and not affected by light, and below the right wrist there was difficulty in distinguishing between hot and cold objects. Injections of two milligrammes of pilocarpin produced a local sweating of the face on the right. Neither galvanism, nor atropine, nor ergotine, nor tannin produced any effect on the patient's malady.

The author divides the cases of local sweating into the following