Aneurysms of the Cerebral Vessels. (*Arch. of Neur. and Psychiat., January*, 1929.) *Sands, I. J.*

Cerebral aneurysms may be caused by arterio-sclerosis, septic emboli, or congenital weakness of the vessel-walls. In the vast majority of instances, cerebral aneurysms are recognized only after they have ruptured. The signs of ruptured cerebral aneurysm are those due to the disease causing the aneurysm, those due to pressure or irritation of the surrounding brain structure, and those due to subarachnoid hæmorrhage. In a person suffering from hypertension or arterio-sclerosis, or from a general or local infection, especially infective endocarditis, the sudden onset of headache, nausea, vomiting, unconsciousness and convulsions, and the presence of cervical rigidity and Kernig's sign, disturbances in pupillary reflexes, blurring of the discs, papillœdema, diplopia, paralysis of the cranial nerves and disturbances of deep reflexes, together with a bloody spinal fluid, point to a ruptured intracranial aneurysm. Absolute rest in bed is the most important therapeutic measure.

G. W. T. H. Fleming.

Cerebral Hæmorrhage Consequent on Softening due to Thrombosis [*L'Hémorragie cérébrale massive consécutive au ramollisement cérébral thrombosique*]. (*L'Encéph., June*, 1928.) *Lhermitte and Kyriaco, N.*

The authors cast doubt upon the usually accepted belief that the main cause of large cerebral hæmorrhages is the rupture of a miliary aneurysm. Inspired by the almost forgotten work of Rochoux and the more recent researches of Artobus and Strauss, they have investigated a number of cases, and have come to the conclusion that a considerable number of sudden, large and fatal hæmorrhages occur in parts of the brain where sclerosis and softening have already prepared the way.

A number of cases are given, and the symptoms in life and *post-mortem* findings carefully recorded. R. S. Gibson.

Cerebro-hepatic Syndrome with Cystic Degeneration of the Liver-cell Nuclei [*Syndrome hépato-encéphalique : transformation kystique des noyaux des cellules hépatique*]. (*L'Encéph., April*, 1928.) *Guiraud, P.*

This paper adds a case to those observed and described by Marchand and Courtois. In 1913, at the age of 2 years, the patient had a rather vague, feverish, nervous illness. During the following fifteen years he suffered from epileptiform seizures, and a condition closely resembling the Parkinsonian syndrome. He died at the age of seventeen from pulmonary tuberculosis.

The *post-mortem* findings included extensive degeneration and sclerosis throughout the cerebrum. The most interesting feature, however, was the condition of the liver. Here there was profound lobular disintegration with overgrowth of fibrous tissue and formation of new bile capillaries. There was also extensive cystic formation of a peculiar type.

The author discusses the resemblance to progressive lenticular degeneration, and adduces evidence in support of his thesis that the condition is due to a toxin and is really an infective process.

The article is illustrated by reproductions of miscroscope slides.

R. S. GIBSON.

A Clinical Example of Acute Ataxia [*Un cas clinique d'ataxie aiguë*]. (*L'Encéphale, May*, 1928.) *Nuica, D., and Parvulescu, N.*

The case is recorded of a man who was suddenly seized by complete paralysis of all four limbs, with fever, delirium and profound dysarthria. The acute illness lasted about eight days, after which recovery set in slowly, and eventually became complete.

The authors quote records of similar cases, and consider that the syndrome is liable to occur after certain infectious diseases, such as pneumonia, diphtheria, smallpox, enteric and puerperal fevers. In the majority of cases the prognosis appears to be good.

R. S. GIBSON.

Ocular Symptoms in Tabes. (*Riv. di Pat. Nerv. e Ment., September–October*, 1927.) *Santonastaso, A.*

The author describes at length 16 cases of " superior " tabes and 34 cases of dorsal tabes.

The pupil showed changes in 94% of cases: absence of the reaction to light was less frequent in superior tabes (81%); unequal pupils were present in 56%, myosis in 25% of dorsal tabes, loss of accommodation in 25% of superior and 17% of dorsal, mydriasis in 6% of superior tabes, atrophy of all or part of the iris in 6% of superior and 14% of dorsal tabes.

Vision was impaired in 94% of superior and 70·6% of dorsal tabes. The fundus oculi showed many early ophthalmoscopic lesions. In 60% of cases there was a primary optic atrophy. The field of vision showed alterations in 87% of superior tabes and in 76% of dorsal tabes. Bitemporal hemianopsia was only found in superior tabes. Central scotomata were found in 18% of superior tabes and in 12% of dorsal tabes.

G. W. T. H. FLEMING.

The Relation of Neuro-Recurrences to Late Syphilis. (*Arch. of Neur. and Psychiat., January*, 1929.) *Moore, J. E.*

Neuro-recurrences occur in at least ·2% of patients with early syphilis and occur more often in males than in females. They are limited to patients with early syphilis who have been inadequately treated, and do not appear late in the course of the infection. The type and severity of early lesions play no part in the production of neuro-recurrences. They are rare after bismuth treatment. The Wassermann in the blood is frequently negative at the time of appearance of a neuro-recurrence. The spinal fluid at the time usually gives positive tests, but does not show anything characteristic. The immediate clinical response to treatment is usually satisfactory, except in lesions of the auditory nerve; here