

**Bing, R., and Schwarz, L.** STEREOGNOSIS. [Schweiz Arch. f. Neur. u. Psych., Vol. 4, No. 2.]

In this elaborate study the authors show that there is a cortical lesion when the subject is unable to recognize objects by touch, the sensory and perceptive powers being unmodified. When the cortical lesion is diffuse there is loss of secondary tactile identification, but usually it is a symptom of a focal vision. This focal vision is usually at the middle third of the parietal ascending convolution and the parietal lobe on the opposite side from the astereognosis. In one patient the astereognosis of the left hand was so pronounced that, with the eyes closed, the man was unable to tell a coin from a pencil, a watch from a box of matches. Other symptoms also suggested a tumor in the brain; they had come on suddenly and by the tenth day left hemiparesis developed. Consent to proposed operations was deferred and the patient died seventeen days after the onset. On necropsy an abscess was found at the point specified above while the parietal lobe and the supramarginal gyri were intact. The findings confirm Head's statements regarding cortical localization.

**Friedman, E. D.** CORNEAL ANESTHESIA IN HEMIPLEGIA. [June 21, 1919. J. A. M. A.]

In coma of moderate degree E. D. Friedman has found that unilateral corneal anesthesia evidenced by absence of the corneal reflex (winking), is a valuable diagnostic point differentiating hemiplegia from other forms of coma. The lesion must be sensory, for if it were motor, as stated by Milian, the consensual reflex would still be present on the other side. The corneal anesthesia may be due to the presence of sensory fibers in the motor pathways. Diagnosis between hemiplegia and uræmia is important, as it affects the treatment. The reflex is best elicited by carefully passing a small blunt object along the conjunctiva to the corneal margin.

**Jefferson, G.** GUNSHOT WOUNDS OF SCALP: NEUROLOGIC SIGNS PRESENTED. [Brain, 42, 1919, Pt. 2. J. A. M. A.]

The observations recorded by Jefferson are based on a series of fifty-hour unselected average cases of scalp wounds as seen at the base in France. A large number of the patients showed generalized signs; a history of unconsciousness, complete or partial, with vomiting, nausea, headache, and exaggeration of the tendon jerks generally. Only five patients out of the whole series showed no positive neurologic signs at all. In ten more the only symptom complained of was headache, but this was often so severe and the patients were mentally so dull for a short time that it was evident that the brain had received a severe shaking up. One fourth of the patients allege that they were actually unconscious for brief periods, while another fourth were stunned, frequently being knocked down by the impact of the missile. Headache

was present in forty-five cases, definitely absent in six, and not noted in three. Giddiness, was the next most common sign, only being noted on gross changes of posture, and therefore later in convalescence when such active movements began to be attempted. It was never a serious factor. Vomiting occurred in eight cases and nausea was, or had been, present in thirteen. Tendon jerks were exaggerated in twenty cases, and of these eight presented increase of both arm and leg jerks. Increase of the arm jerk always portended a graver injury, and cerebral injury was always suspected when they were active. True ankle and patellar clonus, continuous and regular, was never found, but a few beats, from two or three to six, occurred in seven cases. But when present on one side only, even this mild form has a value. In none of these series was there any injury to the skull, yet there were eleven definite local contusions of the motor cortex, four of the visual, and two more in which a motor lesion was associated with a sensory disturbance of the hand. Three presented Jacksonian seizures, and three were trephined on the neurologic evidence; in two an extradural clot was found, in one, nothing abnormal was noted. There were signs of contralateral injury by contrecoup in four cases.

**Krabbe, Knud H.** A NEW FAMILIAL INFANTILE FORM OF DIFFUSE BRAIN SCLEROSIS. [Brain, Vol. 39, 1916.]

The author describes the histories of five cases and anatomo-pathologically the three cases of a disease which seems heretofore to have been very little described. It shows the following characteristics: it is usually a hereditary disease; it sets in somewhat acutely at about the fifth month in a child who up to then has been quite healthy; it progresses by a chronic course ending with death, five to six months after the onset; universal rigidity of the musculature, violent tonic spasms, probably causing pain, and brought on by touching or noise form characteristic symptoms. As a rule, nystagmus is present, and in the latter stages atrophy of the optic nerve. Periodic elevations of temperature occur without perceptible cause, outside the central nervous system. Finally, extensive paresis and pronounced debility close the scene. The intellect seems scarcely to be developed and the little that is developed seems rapidly to perish. The anatomo-pathological findings are: a marked hardness of the white substance of the brain without alteration of its shape. Microscopical examination of three cases showed relative intactness of cortex and the basal ganglia, the nervous centers of the brain and of the spinal cord; destruction of the medullary sheaths and axis cylinders throughout the white substance of the cerebrum (a 2 mm. layer, however, is preserved immediately under the cortex). Complete destruction of the white matter of the cerebellum and degeneration of the spinal nerve tracts are present. The destroyed tissue is replaced by dense fibrillar glia, in which are seen a considerable number of variously shaped glia-cells, mostly protoplasmic; the