bral complications, hemorrhage and softening, for which the kidney sclerosis is only indirectly responsible. Cerebral hemorrhage was found by Frerichs in 11 of 292 uremia cadavers, and 3 in 112 by Rosenstein. Canti has recently published 3 cases of cerebral hemorrhage or softening with azotemia of 0.9 to 1.5 gm. per liter. Meningeal hemorrhage is not exceptional with chronic kidney disease. The latter raises the blood pressure until some vessel ruptures. This explanation of the meningeal reactions in uremia is mechanical rather than toxic, would be verified oftener if the cerebrospinal fluid was examined more carefully at necropsies. Slight xanthochromia may escape detection unless the tube is inspected lengthwise, but this proves the presence of blood, thus adding a fourth type to the three biologic types of uremic meningeal reactions, those with hyperalbuminosis, those presenting merely hypercytosis, or both combined. Acute infectious meningitis in a patient with uremia is often mistaken for uremic meningitis. The pneumococcus is usually involved, but syphilis and tuberculosis may likewise be responsible. In one such case, with positive Wassermann reaction, the convulsions subsided after lumbar puncture, but the headache and torpor did not improve until under treatment for syphilis. When the uremia alone can be incriminated for the meningitis or meningeal condition, the spinal fluid may show merely cytologic and chemical reactions without clinical manifestations, or these reactions may be encountered in the course of neuro-uremia, febrile or not, with headache, vomiting, convulsions, stiffness of the back of the neck, Kernig sign and contracture of the spine. But Roger insists that most of these symptoms are the work of the azotemia rather than any special injury of the meninges. The urea poisons the cortex. He had one case in which extreme retention of sodium chlorid alone, without uremia, was responsible for convulsions, etc. To prove the various points in his article, he cites numerous concrete examples from his own and others' experiences.

Haden, R. L. MENINGITIS. [Arch. Int. Med., Nov. 19, 1919.]

Haden in thirty-one cases of meningitis had twenty-one, or 67.7 per cent., which showed evidence of a generalized infection previous to the localization in the meninges. The mortality of the entire series was 22.6 per cent. During the period of intraspinal treatment alone, and combined intraspinal and intrasubcutaneous treatment, the mortality was 37.5 per cent.; during the period in which intraspinal and intravenous therapy was employed, the mortality was 6.6 per cent. One case in which an active localized infection in the meninges recovered under intravenous therapy is reported.

Lecène, P., and H. Routtier. PROGNOSIS IN CRANIAL TRAUMATISM. [Presse Méd., Nov. 12, 1919.]

Extensive clinical experience during the war makes these authors urge that serious attention should be paid, in the prognosis of cranial

traumatisms, to a pathological state which corresponds neither to an extensive local focus of traumatic encephalitis nor to a meningoencephalic infection, but to diffuse brain lesions of concussion—"commotional"—origin. More or less prolonged unconsciousness, followed by a stage of mental confusion and disturbed tendon and skin reflexes, either diffuse or localized in one segment of the body attend pure brain "commotion." There may be disturbances of vascular tone and of muscular tonicity, the latter at times confirmed by electrical tests. Finally, there are psychic reactions and subjective disturbances the organic nature of which may be shown through the abnormal response of the patient to induced vertigo. Lumbar puncture in these cases revealed many instances of abnormal histochemical conditions in the cerebrospinal fluid. Commotional lesions in the brain itself were found by postmortem histological examination. The prognosis is always difficult, both as regards the immediate vital prospect and the ultimate effects. The observed existence of minute histological lesions in the brain doubtless accounts for the importance and chronicity of certain mental disturbances met with following diffuse traumatism of the brain.

Kreider, N. CRANIAL DEFECT. [Journal A. M. A., Ap. 10, 1920.]

As the result of a kick by a horse, a boy sustained a compound fracture of the frontal bone above the left eye. The fissure in the bone was about 3 inches long, and 1 inch broad, running down at each end to a point. Two large pieces of bone were indriven, causing loss of considerable brain substance. The author, under ether anesthesia, removed about six pieces of the bone. The two large fragments were inserted in a pocket, which was prepared in the left hypochondriac region, and made by slightly curved incision, nearly 3 inches long. The two pieces of skull bone were completely buried, and the incision was closed. The wound in the frontal region was thoroughly cleansed, a pledget of iodoform gauze was inserted in the cavity, and the edges of the wound were brought together, except at the ends. Six weeks after the injury was sustained the two fragments of bone were taken from the pocket and placed exactly in their former positions. A considerable layer of fat had become fastened to the outer side of these bones during their sojourn in the abdominal wall, and this was utilized in sewing them in place with fine catgut. The large scalp flap was then brought over, completely closed with silkworm gut, and a firm dressing applied. The result in this case has been perfect recovery. Kreider points out that the implants should be placed in the hypochondrium with the outer surface of the bones in contact with the fatty tissues in this region. When they are removed, as much of this fatty tissue as possible should be brought out with the bones. When they are replaced, this tissue should be sewed with fine catgut to the epicranial aponeurosis and the implants secured in place.