zone has gone, and no spongiocytes are found in either the glomerular or the fasciculated layer of the cortex. A similar condition is sometimes found apart from motor agitation. Accepting Mulon's teaching that these cortical enclosures are reserves of accumulated lipoids, the writer concludes that intense prolonged motor agitation leads to exhaustion of the labile phosphorated fats of the adrenals.

Boyd, W. A CASE OF ACUTE SUPRARENAL INSUFFICIENCY. [Jour. Lab. and Clin. Med., Dec., 1918, 4, No. 3, p. 133.]

The patient, 38 years of age, a soldier in a military training camp, was found one morning in a semiconscious condition. Admitted to the hospital a few hours later he was sinking into a state of coma. The picture suggested cerebral hemorrhage with limbs rigid, reflexes gone, pupils distinctly dilated, face and hands cyanosed, and a temperature of 99° F. Urine was normal. Cerebrospinal fluid showed no change and gave a negative Wassermann reaction. The patient died two and a half hours after admission.

The necropsy, performed within two hours of death, showed the suprarenals to be the only organs exhibiting any marked pathologic change, being almost entirely destroyed, and converted into structureless, amorphous, yellowish masses, firm in texture, and considerably larger than the original glands. On the right no trace of suprarenal tissue, either cortex or medulla, was found. On the left a narrow strip of gland of about 2 mm. in width was present, consisting entirely of cortex, whose cells contained a lipid substance staining red with Scharlach R, but not anisotropic. The rest of the gland consisted of areas of necrosis surrounded by the endothelial and giant cells characteristic of tuberculosis. There was no trace of medullary substance. A small nodule showing the microscopic structure of a sympathetic ganglion was attached to the caseous mass. The nerve cells and fibers of this ganglion were stained by various methods, while they showed no abnormality, showed some increase of fibrous tissue.

Löwenthal, K. SUPRARENAL APOPLEXY. [Berl. med. Woch., Nov. 25, 1918.]

A patient with double pneumonia and pleural collection developed intense abdominal pain, constipation and slight meteorism, profuse sweating, and a slow, irregular pulse. At autopsy there was found a hemorrhage in both adrenals; the medullary substance was entirely destroyed in one, and greatly involved in the other. The cortex of the glands was preserved. The writer points out the concordance between the clinical signs offered and the physiological data known, which show that the disturbances arising in this case resulted from a lack of tonus of the sympathetic, that is to say, from an adrenalin insufficiency. The differential diagnosis from ileus or peritonitis should be based above

all on the pulse, which in these circumstances is rapid, while in supranrenal apoplexy there is a tendency to slowing of the pulse rate. In the case reported by the writer there were 44 beats to the minute.

II. SENSORI-MOTOR NEUROLOGY

1. PERIPHERAL NERVES.

Tarchetti, C. PERIPHERAL NEURITIS IN CHRONIC ACONITE POISONING. [Gazz. deg. Osped., Aug. 8, 1918.]

Ten cases of peripheral neuritis in men with pulmonary tuberculosis were observed by the author as occurring at a certain hospital. He was able to trace it to a prescription in vogue which contained aconite. The neuritis was due to chronic aconite poisoning, as there have been no further cases of the neuritis during the six years since the aconite prescription was discarded.

Sicard and Roger. LOSS OF ACHILLES REFLEXES IN INTENSIVE ARSENICAL TREATMENT. [Paris médical, June 29, 1918.]

The authors call attention to destruction of the Achilles tendon reflexes as an early sign of chronic arsenical poisoning in paretics subjected to intensive neoarsenobenzol treatment in the daily intravenous dose of 0.3 gram, up to an aggregate dose of twelve to twenty grams. The loss of the reflex indicates a latent arsenical neuritis of the internal popliteal nerve, as yet unaccompanied by disturbances of locomotion, pain or muscular atrophy, yet already resulting in certain quantitative modifications of the electric reactions in the involved muscles. Paretics subjected to such treatment show marked physical and mental improvement, but there is no clinical or humoral cure, the Bordet-Wassermann reaction in the cerebrospinal fluid remaining irreducible.

Preiser, S. A., and Davenport, C. B. MULTIPLE NEUROFIBROMATOSIS (VON RECKLINGHAUSEN'S DISEASE) AND ITS INHERITANCE. [American Journal of the Medical Sciences, Oct., 1918.]

These authors describe the occurrence of von Recklinghausen's disease in a father and son, with the autopsy findings in the case of the father, in whom the disease lasted thirteen years. The presence of the disease in the son was associated with definite mental inferiority and delayed sexual development. The writers give a review and analysis of 243 cases of multiple neurofibromatosis from the literature, and an analysis of thirty cases of the familiar type, with charts of families in which there were two or more affected persons in one family. This establishes the hereditary tendency of the disease, showing the hereditary factor to be dominant, there being something, apparently, in the germ plasm that positively facilitates the production, under appropriate stimulation, of tumors of the nerve sheaths. The tumors at times assume a malignant character.