available. Treatment by quinin, intramuscularly or intravenously, usually both, was applied at once. Twelve cases terminated fatally within twenty-four hours. One case terminated in six hours, while two cases lingered for sixty hours. Temperature, when recorded, was always raised from 100° to 105° F. Capillaries and, in severe cases, the arterioles were engorged with infected red blood cells collected at the periphery of the vessels. Prominent and detached endothelial cells, melanin particles, and free parasites, were numerous. The crescent phase of development was not seen, although "dot" forms, fine rings and segmenting forms were present. All degrees of blocking to complete thrombosis with agglutination of the corpuscles and altered staining reactions were observed. Hemorrhages around the smaller blood vessels was met with occasionally, the rupture of vessels in one case having made possible the discharge of parasites into the tissues. This was not usual in rupture of the cerebral capillaries or capillaries in other viscera. The vessels were filled with infected red cells, or in some cases the red cells were seen closely packed about the vessel walls. No infected red cells were to be found in the hemorrhagic zone. The lining endothelial cells showed pigment, in detached phagocytes and free in the lumen. Eleven cases showed nerve cell degeneration, evidenced in abnormalities, loss of Nissl granules, and distortions. In one case where paraplegic symptoms had developed before death the spinal cord showed advanced cell degeneration of the anterior horn cells with typical vascular changes and complete vessels blocking. The spleen, marrow, heart and pancreas showed local accumulations most frequently, the intestines, lungs and suprarenals less frequently. In the liver, kidney and thyroid these accumulations were only occasionally met with. Other conditions recorded were fatty and other degenerations of the cardiac muscles, acute tubal "nephritis," vascular changes in the suprarenals, with tissue necrosis, pulmonary congestion and hemorrhage.

7. NEUROSYPHILIS.

Mehrtens, G. H. THERAPEUTIC MENINGEAL IRRITATION IN NEUROSYPHILIS. [Am. Arch. Neur. and Psych., Oct., 1919.]

Experiments were undertaken to find out if the normal penetration of arsenic into the spinal fluid could be increased by an irritation of the meninges such as must occur in all of the intradural treatments. It was found that irritation of the meninges by intradural injection of the patient's own serum caused a cellular reaction ranging from 100 to 2,300 cells per cubic millimeter of spinal fluid. Simple intravenous injection of 0.6 gm. arsphenamine resulted in a positive test for arsenic in the spinal fluid in 43 per cent. of the cases examined. Complete drainage of the spinal fluid did not increase the number of arsenic penetrations. Intravenous injection of arsphenamine, six hours after meningeal irritation, gave 92 per cent. penetrations, and compared with controls gave three times as strong an average concentration of arsenic.

It would seem that the foregoing results could be applied to the treatment of neurosyphilitics in the following way: (1) Every case should have the benefit of intensive intravenous medication until it is certain that the case belongs among those with impermeable membranes. (2) It would also suggest that for the cases resistant to ordinary therapy, in order to obtain the maximum concentration of arsenic in the spinal fluid, the patient's own blood serum should be injected into the subarachnoid space six hours before the arsphenamine is given intravenously. This serum may be injected as in the Swift Ellis technic, or if necessary, arsenic or mercury may be added according to the Ogilvie or Byrnes technic. This procedure is not more complicated than either of the intradural methods, and has the definite advantage of allowing arsenic to pass from the blood to the spinal fluid in greatly increased concentration. [Author's abstract.]

Mackenzie, Joseph. SYPHILIS OF THE NERVOUS SYSTEM AND ITS TREATMENT. [Glasgow Medical Journal, July, 1919.]

Mackenzie says that gummata, or syphilitic granulomata, may develop in practically any part of the syphilitic subject. In the central nervous system this manifestation of the disease may occur in a variety of forms. In the first place, it may be present in the form of a large tumorlike mass, commencing either in the membranes of the nervous system or in the substance of the brain itself. The symptoms to which it gives rise are those of tumor, but the gumma may be only one or two or more different expressions of infection, and in that case the symptoms may be, and usually are, complicated by those disturbances which depend upon the other manifestations of the disease. For example, one sometimes finds a gumma in a case of general paralysis, or in a case of locomotor ataxia, and in such conditions clinical phenomena of a mixed character would be observed. These gummata, which may be single or occasionally multiple, give rise to progressive symptoms of pressure, which, according to the site of the growth, produce different localizing symptoms. They yield readily to treatment, if treatment has been begun before the pressure effects have produced permanent damage to the important brain structures. They occur in the congenital as well as in the acquired form of the disease, and, as a rule, make their appearance after the second year of its development. Then, again, the gummata may be miliary; that is to say, they may be present in large numbers and dispersed all over the surface of the brain, like miliary tubercles, or scattered here and there along the lines of the main vessels in the substance of the brain. The miliary form of gummatus infiltration occurs late in the disease, and is usually noted for the first time at postmortem, or at a stage when effective treatment is no longer possible. Mackenzie has seen three cases of this type at postmortem, and in each of these, in addition to the miliary gummata, there had