epileptics dying in the epileptic condition. Such lesions show themselves under the form of little round cavities, filled with a homogeneous substance scattered about the brain and sometimes in the medulla, as much in the white substance as in the grey, but predominating in the former. They do not show micro-organisms in their midst. They are accompanied by dilatation of the vascular and peri-vascular spaces, and by grave lesions of the nerve-cells. In some cases, the margins of the small cavities are torn, and the surrounding tissue to a very small extent is disintegrated, the cavities being signs of a notable increase in the lymph pressure.

(5) The status cribosus of Vassale is frequent in general paralysis, and in all conditions of wasting of the nervous substance. It consists of degeneration of the myeline sheath of the large nerve-fibres. It is easily found in the pons, and is in the form of small microscopical cavities filled with a hyaline material, in which the axis cylinder of the nerve is found tortuous and altered. To differentiate it from the *état criblé* of Durand-Fardel, the author proposes to call this condition "the punctiform state."

(6) The worm-eaten condition is found in senile dementia only, and then not very frequently. It shows itself as ulcerations in the form of yellow plaques of the grey substance only, and consists in a limited softening of small tracts of the cortex, as a result of sclerosis of the capillaries. It is followed by an active proliferation of the neuroglia, which encloses in its margin the small cavities.

(7) The porous states are the result of *post-mortem* change. They occur in the form of numerous cavities scattered through the whole of the white substance of the cerebrum and cerebellum. They show great variety in size, and are found from the size of a grain of millet to that of a bean. The walls are smooth and white in colour, with margins quite devoid of any membranous covering. The surrounding nervous tissue presents only cadaveric lesions. The walls of the cavities and those of the perivascular spaces are filled with micro-organisms capable of producing gas. The abundant gaseous productions are not able to open a way to the exterior, owing to the resistance of the more superficial strata, and they therefore putrefy with greater delay than the other portions of the brain. The gaseous tension is exercised on the point where these gases originate. It presses on the tissue from within, and thus gives rise to the porous cavities. The extent of the lesions, and, during life, the absence of any signs showing porosities, also the ease with which these cavities are experimentally produced, convey an idea that they are of a cadaveric nature. HAMILTON C. MARR.

The Neuroglia in General Paralysis [*La Néuroglie dans la Générale*]. (*Soc. Clin. Med. Ment., June*, 1908.) *Dagonet, M.*

Dagonet (the author of the *Atlas* published by Baillière, 1897) considers that the neuroglia, owing to its ectoblastic origin, does not become sclerosed as the connective tissue does, but that in general paralytics it hypertrophies and then proliferates, filling up the lacunæ produced by the destruction of the nerve-cells and fibres.

He does not agree with Weigert that the neuroglia is composed of cells and of separate fibres, but considers that the latter are, in fact,

only part of the former. These neuroglial cells show many forms; some are round without any protoplasmic prolongations or fibres, whilst others show either or both. In a section of the cerebral surface of a general paralytic one notices a thickness of the neuroglial bed beneath the membranes, formed by entangled fibres and little star-like cells which invade the molecular stratum. These fibres are thick and rigid, and when they attain the size of 1μ to 1·5 μ they are said to be hypertrophied. In the stratum of the smaller nerve-cells these neuroglial fibres disappear, or, at least, Weigert's method does not stain them, but they exist in small numbers.

In the deeper strata, however, the hypertrophy and proliferation of the neuroglial fibres is remarkable. The cells have a large amount of protoplasm, often two nuclei and very long prolongations, which become attached to the sheath of the vessels. These are the spider cells, and this sclerosis of the neuroglia is called perivascular.

The larger spider cells, the so-called monster cells, have nuclei which attain the size of 8 μ, and their prolongations, having a thickness of 4 μ, appear to be of a horny nature. One sees numbers of these in the neighbourhood of the third ventricle. In the white substance one does not find these spider cells but many Deiter's cells. In the cerebellum there is no neuroglial bed under the pia mater as seen in the cerebrum. The neuroglial fibres are firm, thickened, and one sees them equally in the bed of the granular layer, which in the normal state is not so. In the medulla these neuroglial fibres form an excessive felting around the nerve-cells, and one will notice their unequal distribution in the white matter or in the myelitic foci, and the fasciculated lesions are very frequent, as Westphal has shown.

As regards the granulations on the surface of the ventricles, they are thickened tufts of neuroglial fibres which project into the spaces of the cavities. Weigert says that the ependymal epithelium is wanting at the summit of these granulations, and that this is the primary reason of the lesion, for there is a suppression of the resistance of the ependymal tissue, and the neuroglial mass beneath it, being no longer held in check, proliferates. The author does not agree with this statement, for he can demonstrate slides showing that the granulations are covered with the epithelium of the ependyma, and even some of the ependymal fibres seem to play a part in the formation of these nodules.

These ependymal fibres, seen in the embryonic state, disappear in the normal adult, but reappear in the general paralytic.

SIDNEY CLARKE.

Staining of Blood Films.

To those working on the blood in the insane the following may be of some value, since Leishman's stain is so frequently used.

Colonel Birt, at the Pathological Section of the Royal Society of Medicine (as reported in the *Brit. Med. Journ.*, Nov. 7th, 1908), exhibited blood-films stained with Leishman's fluid, to which an equal volume of glycerine had been added nearly two years previously. Ordinary Leishman's fluid, he observed, underwent deterioration after the lapse of a few weeks, sometimes so great that the nuclei of leucocytes remained colourless. A satisfactory stable staining solution might also