Epilepsy may be classified according to three epochs of life: (1) up to the age of 20 years; (2) during adult life; (3) after the beginning of senility. More than three-fourths of the cases occur in the first period. The epilepsies of the first period may be grouped under two heads: primary, or developmental, and accidental. The latter also occurs during the adult period, but with far less frequency. Two kinds of heredity must be considered: similar and dissimilar, the former meaning the transmission of the same disease from parent to child, the latter meaning that alcoholism, insanity, tuberculosis, chorea or a kindred affection in the parent is changed in type in transmission so as to appear as epilepsy in the child. In a study of 1,100 cases of epilepsy the author finds 16 per cent. due to similar heredity, 40 per cent. to dissimilar heredity, such as alcoholism in the ancestors, 16 per cent. in the males, 12 per cent. in the females; insanity in the ancestors 7 per cent., tuberculosis 14 per cent. Of accidental forms 11 per cent. were cerebral palsies; primary or developmental epilepsy after 20 years is probably never met with. After this age it is due to syphilis or traumatism, to alcoholism, ptomain or chemical poison. The writer believes that the sensory or angular cells in the second layer of the brain exercise a power of control over the motor cells in the layer above. When these sensory cells become weakened or destroyed they lose their power of control, and the motor cells explode; that is, they suddenly and violently give off their force at irregular intervals. The structural incompleteness of the sensory cells and consequent failure of inhibition, rather than the over production of nerve force by the large motor cells, is responsible for these periodic discharges.

The writer notes three specific lesions after paroxysmal discharges in uncomplicated epilepsy: (1) a partial disappearance of the tangential fibers of the cortex, "the neurones of association," so-called, and similar to lesions described in cases of general paralysis; (2) an infiltration of nerve cells by leucocytes; (3) the disappearance of the fine granules in the protoplasm of the cell substance of the cortical nerve cells. The latter are probably due to chemical changes dependent on nutritive processes. In treatment the use of bromipin, a brominized oil of sesamum, is advocated.

W. B. NOYES (New York).

PSEUDOMENINGITIS. T. Donath (Deut. med. Woch., April 17, 1902).

The presence of a symptom-complex resembling meningitis, without the demonstrable lesions of this disease at a later autopsy, has sometimes been noted, especially during epidemics of influenza. An unusual case of this kind is reported. The patient, a young man of eighteen, developed eleven days after an osteotomy for genu valgum a sudden rise of temperature, severe cerebral symptoms, loss of consciousness, muscular spasm, retracted abdomen, etc., with death following on the third day. Fat embolus and uremia could be properly excluded, and all the signs pointed to meningitis, possibly tuberculous. Autopsy showed some edema of the pia and surface of the brain, but no signs of any acute inflammatory changes. The influenza bacillus could be readily demonstrated in some of the lobules. The author calls attention to the diagnostic value of lumbar puncture in these cases, when ordinary means are of doubtful value.

JELLIFFE.