involvement of this area will show the Argyll Robertson pupil. In a series of 749 cases of neurosyphilis, the authors found the Argyll Robertson pupil present in 38·3%. G. W. T. H. FLEMING.

Concerning the Striatal Localization in Chronic Progressive Chorea. (*Journ. Nerv. and Ment Dis., vol. lxxviii, p.* 470, *Nov.*, 1933.) *Neustaedter, M.*

The author describes the pathological findings in three cases of chorea, two of the Huntington type, in a brother and sister, and one senile arterio-sclerotic.

All his cases showed convolutional atrophy involving the frontal and central gyri and a corresponding diminution of the white matter. The caudate nucleus and putamen were atrophic in the brother and sister. In the other case these nuclei appeared fairly normal. The globus pallidus was considerably shrunken in the brother and sister. Microscopically the cortex, in the brother and sister, showed obvious disorder of the cell arrangement in the third and fourth layers. In the third case all layers presented this disarrangement, and in addition many areas of softening were present. Changes in the small cells of the neo-striatum in the brother and sister were of a chronic degenerative type, while those of the other case were of an acute type due to arterio-sclerotic changes. The pallidal cells were not involved in any case. In the third case the thalami, red nuclei and olives were markedly affected. The cell destruction was of a chronic nature antedating that in the neo-striatum. In the cerebellum the changes in the first two were slight, but in the third case the dentate nuclei showed considerable cell destruction of a chronic type. The author concludes that in all three cases the choreiform syndrome was probably due to the destruction of the small cells of the neo-striatum. The third case supports the views of Jakob and Vogt with respect to the rôle played by the basal ganglia and cerebellum in their relation to the neo-striatum in the production of the choreiform syndrome. G. W. T. H. FLEMING.

A Comparison of the Viscosity of Muscles in Catatonic and Parkinsonian Rigidity. (*Arch. of Neur. and Psychiat., vol. xxxi, p.* 87, *Jan.*, 1934.) *Finkelman, I.*

The author found that the muscles of patients suffering from catatonic dementia præcox possess a higher degree of elasticity and but little internal friction (viscosity). Catatonic rigidity differs in this respect from the rigidity of chronic encephalitis. The difference between the muscle tonus curves of chronic encephalitis and catatonia is evidence that the muscle rigidity in these two conditions is not due to physiological interruption at the same levels. G. W. T. H. FLEMING.

A Contribution to the Study of Late Cerebellar Atrophy with Rigidity [*Contribución al estudio de la atrofia cerebelosa tardía con rigidez*]. (*La Semana Méd., vol. xli, p.* 109, *Jan* 11, 1934.) *Dimitri, V., and Victoria, M.*

The description and discussion of a case leads to the following conclusions: It is possible to have states of plastic rigidity with integrity of the basal ganglia. A cerebellar symptomatology may coexist with Parkinsonian rigidity. But it is also possible to meet with two alternatives: (1) The cerebellar symptoms may be partly or wholly replaced, during the later stages of the illness, by Parkinsonian symptoms; (2) the Parkinsonian syndrome may be replaced, before the end of the illness, by a typical cerebellar syndrome. M. HAMBLIN SMITH.

The Syndrome of the Median Cerebellar Line [*Sindrome de la línea media cerebelar*]. (*La Semana Méd., vol. xli, p.* 2, *Jan.* 4, 1934.) *Obarrio, J. M., Dowling, E., and Pedace, E. A.*

The structures of the middle line of the cerebellum are the seat of a series of new-growths, of characteristic histopathology and of very varied evolution. The clinical picture produced by one of these growths is, with slight variations, always the same; for this reason the title set out above is the most suitable. The syndrome

is so characteristic that it sufficed, without any other aids to diagnosis, for the exact localization of the tumour in a series of 24 patients, all of which cases were verified surgically. M. HAMBLIN SMITH.

A Case of Narcoleptic Cataplexy, Probably of Encephalitic Origin [Cataplexia narcoléptica, provávelmente encefalitica]. (Rev. da Assoç. Paulista de Med., vol. iii, p. 183, Oct., 1933.) Vampré, E., Sobrinho, P., and Ribeiro, J.

Observations upon narcoleptic cataplexy are rare. The authors give a detailed description of a case. They believe that the phenomena presented by the disease depend upon a larval type of epidemic encephalitis. The syndrome consists of a repetition of crises manifested by a general or localized loss of positional tone and of muscular power. It is never easy to eliminate entirely the possibility of simulation. M. HAMBLIN SMITH.

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5. Treatment.

Mechanism of the Action of Calcium and Potassium Salts Injected into the Cerebral Ventricles. (Compt. Rend. Soc. Biol., vol. cxiv, p. 674, 1933.) Stern, L., Rossine, J. A., and Chroles, G. J.

The direct action of calcium on the vegetative nervous centres of the brain consists of an excitation of the parasympathetic centres (vaso-dilator and cardiac inhibitor), but a possible inhibitor effect on the sympathetic centres is not excluded. Potassium excites the vaso-constrictor and cardiac centres and probably indirectly weakens the tonus of the parasympathetic centres.

L. E. GILSON (Chem. Abstr.).

Alkalosis and Excitability of the Cortical Sensory-motor Centres. (Arch. fisiol., vol. xxxii, p. 361, 1933.) Martino, G., and de'Finis, L.

Oral doses of 2–10 grm. of $CaCl_2$ or HN_4Cl have no influence on the excitability of the cortical centres of the dog. Pulmonary hyperventilation after doses of these salts does not cause changes, whereas it increases the excitability in non-treated dogs. The latter effect is probably caused by alkalosis.

A. E. MEYER (Chem. Abstr.).

The Barbituric Acids: Their Toxicology. (Thesis, Lille, 1932.) Desodt, C.

The properties, uses, toxicology and colour reactions of five important barbituric acid hypnotics are reviewed. The original work consists in the application of a new method for isolation of barbituric acids from the urine. To 250–300 c.c. of urine add ·1 volume of 15% $K_4Fe(CN)_6$ and shake, then add ·1 volume of 11·2% $2N(OAc)_2$ and mix with a stirrer. Filter, and if the filtrate is not acid, add a few drops of AcOH. Transfer to a separating funnel, add 75 c.c. of ether, mix carefully at first, then more vigorously, draw off the aqueous layer and repeat the extraction four times. Dry the combined extracts for 20 minutes with anhydrous sodium sulphate and filter through cotton. Evaporate the ether on a water-bath, extract the residue with 10–20 c.c. of boiling ethyl alcohol, filter into a weighed dish and evaporate. A white residue is assumed to be pure barbituric acid, and the percentage is calculated from the volumes of urine and of the various filtrates. If the residue is coloured, it is extracted with 20–30 c.c. of boiling water and treated with animal charcoal for 15 minutes or until the colour is removed, then evaporated and weighed. The recovery of barbituric acid (barbital, rutonal, gardenal, dial and soneryl) is 89–96% of the amount present. Essentially the same procedure is said to be applicable to blood, gastric contents and cerebro-spinal fluid. Daily excretion by hospital patients during daily doses for the period indicated averaged: