The Action of Potassium on the Superior Cervical Ganglion of the Cat. (*Journ. Physiol., vol. lxxxvi, pp.* 290–305, 1936.) *Brown, G. L., and Feldberg, W.*

Potassium ions liberate acetylcholine from the normally innervated ganglion. A similar property is shown by Rb and to a weak degree by Cs. Sodium and calcium do not have this action. Calcium inhibits the liberation of acetylcholine by potassium. Potassium stimulates the cells of both normal and denervated ganglia to discharge. In large doses potassium has a paralysing effect on ganglion cells.

E. D. WALTER (Chem. Abstr.).

The Liberation of Acetylcholine by Potassium. (*Journ. Physiol., vol. lxxxvi, pp.* 306–14, 1936). *Feldberg, W., and Guimarais, J. A.*

Potassium chloride injected into the arterial blood liberates acetylcholine from the salivary glands, the tongue and the sweat-glands.

E. D. WALTER (Chem. Abstr.).

The Relation between the Alkaline Reserve of the Plasma and the Cerebro-spinal Fluid in Healthy and Pathological Children. (*Pediatria Riv., vol. xliii, pp.* 407–21, 1935.) *Moschino, S.*

In normal children the alkaline reserve of the plasma averaged 56% carbon dioxide by volume and that of the cerebro-spinal fluid 50·7%. Variations in the alkaline reserve of the cerebro-spinal fluid are smaller than in the plasma. In nephritis and meningitis the relation between the alkaline reserves of the plasma and the cerebro-spinal fluid is greatly and not uniformly changed.

MARION HORN (Chem. Abstr.).

The Demonstration of Myelolytic Substances in the Urine and Spinal Fluid in Nervous Diseases. (*Trans. Amer. Neur. Assoc., pp.* 142–4, 1935.) *Weil, A., Luhan, J. A., and Balser, B. H.*

Urine and spinal fluid from cases of nervous diseases were evaporated *in vacuo* at 50° and redissolved in saline to 1/40 (urine) or 1/10 (spinal fluid) of the original volume. The solutions were incubated with rat spinal cord at 37° for 16 hours. When the paraffin-embedded spinal cords were cut longitudinally and stained for myelin sheaths, a destructive action upon the sheaths was demonstrated for the urine and spinal fluid from the majority of cases of disseminated sclerosis, post-encephalitic parkinsonianism and brain tumour (spinal fluid only). The myelolytic action, however, was not entirely specific for nervous diseases, since it was also demonstrated in the urine of cases of liver disease and pulmonary tuberculosis. No myelolytic action was detected in urine from normal controls or from other nervous diseases, i.e., subacute combined degeneration or trauma of the spinal cord, arterio-sclerosis of the brain, and syphilis or tumours of the central nervous system. The myelolytic agents of the urine and spinal fluid were of two types: (1) inactivated by boiling (probably lipolytic enzymes), and (2) not inactivated by boiling. Type (2) was present in the alcohol filtrates after the addition of 9 parts of abstract alcohol to the concentrated fluids, and was precipitated when the alcohol filtrate was poured into four times its volume of acetone. This myelolytic precipitate (yield: 0·2–0·6 grm. per 100 c.c. urine) is phosphorus- and sulphide-free, and, unlike similar but non-myelolytic precipitations from normal urine, gives a strong colour reaction for methylguanidine and urobilin (significant in view of the fact that the metabolic product guanidine carbonate acts destructively upon myelin). In the spinal fluid the myelolytic type (2) was further divided into a fraction precipitated by alcohol and one precipitated by acetone.

MARION HORN (Chem. Abstr.).