after the toxin administration. On an average, the content of serotonin and noradrenaline in pooled brains was about 50 per cent of that of the controls. *Shigella shigae* thus causes a considerable depletion of catecholamines in the brain.

29 Excretion of Vanilmandelic Acid by Psychiatric Patients as Related to Drug Therapy. I. MUNKVAD and A. RANDRUP (Denmark).

The demonstration that several psychopharmaca affect brain amines, seems to furnish a clue for further studies on the modes of action of these drugs. Measurement of the influence of the drugs upon the excretion of the amines and their metabolites in urine may thus be of interest, as this could give some information on the direct or indirect influence of the drugs upon the production and the ways of metabolism of the amines. As it is also possible to measure the urinary excretion products in the clinical situation, we have chosen this procedure for studying the mode of action of some drugs in psychiatric patients.

In earlier publications from this hospital the effect of reserpine and of iproniazid upon adrenaline and noradrenaline excretion was reported. This work has now been extended by measurements of vanilmandelic acid, the oxidized excretion product of these two amines. The vanilmandelic acid is isolated by high voltage electrophoresis at pH 3 and measured colorimetrically. The effects of chlorpromazine (in varying doses), reserpine and tetrabenazine is studied.

30 Suppression by Iproniazide of the Antagonistic Action of Reserpine on Amphetamine "Group Toxicity". B. N. HALPERN and C. BARACCO-DRUDI (France).

Reserpine is known to reduce the "group toxicity" of amphetamine in mice, as does chlorpromazine. It has been found in this laboratory that treatment with iproniazide suppresses the effect of reserpine and raises the amphetamine "group toxicity" in reserpine-treated animals to the level of the controls.

The time interval between the injection of iproniazide and reserpine is an essential factor. In our experiments, iproniazide is injected first, followed by the injection of reserpine, while DL benzedrine is administered, in all cases, 4 hr after reserpine. No inversion of the action of reserpine is observed, if the interval iproniazide-reserpine is less than 4 hr. The inversion is regularly observed during the time interval 4–36 hr.

The effect of chlorpromazine is not affected by pretreatment with iproniazide. The mechanism of the inversion by iproniazide of the action of reserpine on the "group toxicity" of amphetamine in relation with the cerebral metabolism of aromatic amines will be discussed.

31 Studies on the Mechanism of Uptake of Catecholamines by Isolated Brain Tissues. E. O. TITUS and H. J. DENGLER (U.S.A.).

Norepinephrine-^{3}H (NE) in slices of cat cerebral cortex incubated with 5 mμg/ml of the labelled amine in Krebs bicarbonate approaches a steady state concentration approximately 4 times that in the medium after 45–60 min. The uptake mechanism, which operates against a concentration gradient and becomes saturated at levels near 100 mμg/ml may be an active transport. It does not function at 0°C and like active transport of 5-hydroxytryptamine in platelets is inhibited by reserpine and cardiac glycosides. Since administration of 3 mg/kg of phenyl α-methylprophylhydrazine (JB 516) 24 hr before the experiment or preincubation of brain slices in 10^{-6} M JB 516 is without effect on uptake, this amine oxidase inhibitor is used to minimize NE breakdown during transport studies. After 45 min incubations, 70 per cent of the isotope in the medium is NE (determined chromotographically). Isotope emerging from the slice in the steady state is accounted for by the following percentages: NE, 42; epinephrine, 2; normetanephrine 35; acidic products of amine oxidase, 19. Corresponding percentages from slices without JB 516 are: 13·6, 10·7, 6·2 and 70·2, respectively. Since 80 to 90 per cent of endogenous NE does not exchange with isotopic NE in 2 hr, NE taken up by the transport mechanism must equilibrate very slowly or not at all with the stored NE in isolated slices. The data suggest existence of at least two intracellular pools of NE.

32a Teneur en Nor-Adrenaline du Tissu Cerebral d'Animaux soumis a l'action de l'Aminodipropionitrile. M. BEAUVALLET et J. FUGAZZA (France).

En 1952, Delay *et al.* ont constaté que l'aminodipropionitrile:HN = $(CH_2\text{-}CH_2\text{-}CN)_2$ (I.D.P.N.) provoque chez la souris une agitation motrice permanente; l'animal présente une activité généralisée avec forte tendance à tourner en rond.

Les rats soumis à l'injection du même produit réagissent de façon voisine, présentant des troubles de la coordination motrice avec perte d'équilibre.

Dans ce travail, nous avons recherché la teneur en nor-adrénaline du tissu cérébral du rat et de la souris avant et après l'injection d'I.D.P.N.

Les premières expériences ont été faites sur des rats de race Wistar de 60–100 g; 3 groupes de 6 femelles et 1 groupe de 6 mâles reçoivent deux injections intrapéritonéales d'I.D.P.N. à 48 heures d'intervalle. Le tissu cérébral est prélevé dès l'apparition du syndrome excito-moteur. En même temps on prélève le cerveau d'un animal normal de même portée, de même sexe et de même poids.

Les résultats que nous avons obtenus montrent que la teneur en nor-adrénaline du tissu cérébral du rat mâle ou femelle soumis à l'action de l'I.D.P.N. est très voisine de celle de l'animal normal.