Table II

Age (days)		Cortex weight (g) / Body weight (g) × 100	Cortex weight (g) / Liver weight (g) × 100
1 ante-partum (fetus)	IUGR	3.0	55
	Control	2.2	33
2	IUGR	2.8	74
	Control	2.3	59
5	IUGR	3.1	88
	Control	2.4	69
10	IUGR	3.4	107
	Control	2.6	99
15	IUGR	2.7	84
	Control	2.1	72
20	IUGR	2.0	54
	Control	1.6	42
30	IUGR	1.5	25
	Control	0.9	19

Table II shows the brain/body weight and brain/liver weight ratios from fetal to adult age. It is noteworthy that these ratios are always higher in the stunted animals. We could therefore assume that the severe hypoglycemia found in the IUGR new-borns would be the consequence of the discrepancy between the needs of their developing brain (normal oxygen uptake) and the diminished potentialities of the liver metabolism.

Résumé. Les hémisphères cérébraux des rats normaux et des rats ayant subi un retard de croissance intra-uterin ont une consommation d'oxygène identique à tous les stades du développement. Ces résultats confirment que le cerveau est épargné par l'hypotrophie provoquée pendant la vie fœtale tant au point de vue pondéral que métabolique.

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Influence of Various Intracranial Pressure Levels on the Concentration of Certain Arylethylamines in Rabbit Brain

Impairment of the cerebrospinal fluid outlow from the ventricular system increases the ventricular fluid pressure (VFP) and results in hydrocephalus. The condition is accompanied by thinning of the brain mantle, atrophic changes in the brain tissue, and often distinct neurological symptoms. However, little is known about the extent of damage to the cerebral neuron systems and about such associated chemical disturbances as may be responsible for the neurological symptoms. Dopamine (DA), noradrenaline (NA) and 5-hydroxytryptamine (5-HT) in brain are located in neurons[1] and seem to have a transmitter function[2–4]. Information about changes in the brain concentration of these amines during different intracranial pressure conditions would offer a measure of the neuronal involvement as well as additional possibilities for an interpretation of functional disturbances seen in hydrocephalus.

Material and Methods. 22 rabbits of either sex (2–3 kg body weight) were used. They received standard pellet food (SAN-bolagen, Sweden), turnips, carrots and tap water ad lib. throughout the experiment. Intracranial hypertension was induced in 18 rabbits by intracisternal injection of 0.5 ml kaolin (30 g/100 ml concentration) as previously described[5]. The VFP was recorded in the conscious animals during $^1/_2$–1 h via a pressure cannula inserted into the left lateral ventricle of the brain[5] at different time intervals after the kaolin injection. Control recordings were obtained from 4 non-injected animals.

After recording was completed, the animals were killed by i.v. air and the brain was immediately removed. The concentrations of DA, NA and 5-HT were measured fluorometrically[6–8] in one tissue preparation comprising the telencephalon, mesencephalon and diencephalon (except cerebellum) and one (brain stem) consisting of pons and medulla oblongata. The different amines were determined on one and the same tissue sample, one animal being used for each determination (Figure).

Results. The mean VFP during the recording period was 5 mm physiological saline in the non-injected animals (Figure). 2 days after kaolin injection the VFP had increased to about 50 mm saline (Student's t-test: $p < 0.05$). At 7 days the VFP returned to a level that was not significantly different from that of the controls. The pressure was of about the same magnitude also 30 days after the injection.

As illustrated in the Figure, the concentration of DA in brain (brain stem not analyzed) was continuously reduced upon kaolin injection to a level about 30% below that of the controls ($p < 0.01$). The pattern of changes in the amount of NA and 5-HT in the brain and brain stem preparations resembled each other, but differed from that of DA in the brain. Thus, 2 days after kaolin treatment (i.e. when VFP was increased) the amine concentrations were 23–38% lower than in the untreated controls. The differences in the mean concentrations were significant except for 5-HT in the brain stem tissue (Figure). 5 days later (i.e. when the VFP had normalized) the amine concentrations had returned almost to the control values. The concentrations were similar also 30 days after the injection (Figure).

Discussion. The results have shown that kaolin-induced intracranial hypertension reduces the concentration of DA, NA, and 5-HT in the brain and the brain stem. It can be assumed that these changes selectively illustrate the influence of intracranial hypertension on the neuronal component in the brain. Several explanations to the changes can be offered. They can be the direct results of the mechanical pressure effect on the neurons, or they can be associated with changes such as in the oxidative meta-

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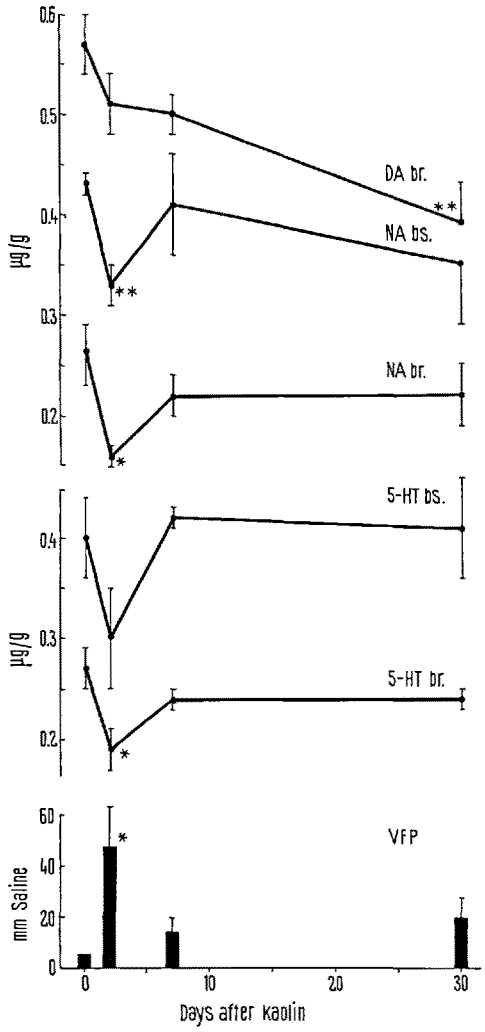

Relation between ventricular fluid pressure (VFP) and the concentrations of dopamine (DA), noradrenaline (NA), and 5-hydroxytryptamine (5-HT) in brain (br.) and brain stem (bs.) at various time-periods after intracisternal injection of kaolin. Mean ± S.E.M. Differences between injected animals and non-injected controls (time 0) according to Student's t-test: * $0.01 < p < 0.05$; ** $0.001 < p < 0.01$.

bolism known to occur in brain during intracranial hypertension[9,10]. The reduction in the amine concentrations may be the consequence of an impaired synthesis and/or an increased turnover and breakdown. The recent finding[11] that homovanillic acid is reduced in the brain during intracranial hypertension incidates that the observed reduction, at least in brain DA, is a consequence of an impaired synthesis. This supports the assumption that the increase in the level of acid amine metabolites found in the cerebrospinal fluid of kaolin-treated animals[12] and hydrocephalic patients[13] reflects a lowered absorption from the ventricular system.

The DA level in the brain showed a progressive fall after the kaolin injection, also when the VFP was normalizing, whereas the reduction in NA and 5-HT was only transient, accompanying the intracranial hypertension. The difference may be due to the different topography of the corresponding neuron systems: the majority of the DA terminals are present in the neostriatum bordering the lateral ventricles. This is consistent with the observations that the structural damage during hydrocephalic conditions affects the periventricular structures more than areas away from the ventricles[14–16].

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Electrolyte Transport in the Seminiferous Tubules of the Rat Studied by the Stopped-Flow Microperfusion Technique

Recently[1] micropuncture and catheterization techniques were used to collect fluid from the seminiferous tubules and rete testis of rats. Three fluids were collected and analyzed: 1. Rete testis fluid; 2. Free-flow fluid, i.e. the fluid which normally lies in the lumen of an undisturbed seminiferous tubule; and 3. Primary fluid, i.e. the fluid which is secreted by the seminiferous tubules after they have first been emptied of free-flow fluid by injection of oil. It was found that primary fluid was extremely rich in potassium (and probably bicarbonate), that rete testis fluid was rich in sodium and chloride and that free-flow fluid had an electrolyte composition intermediate between the other two secretions. To account for the difference between the composition of primary and free-flow fluids it was proposed that the seminiferous tubules secreted only a potassium-rich secretion (the primary fluid) which then mixed by an ebb and flow process with a sodium-rich rete testis secretion to produce an intratubular fluid of intermediate composition (the free-flow fluid).

To test this hypothesis, seminiferous tubules have now been studied by the stopped-flow microperfusion technique[2,3]. Oil was injected into the tubule over a length of 1–2 mm and perfusion fluid was injected into the column so as to split it into two. After a varying interval of time, the sample was re-aspirated for analysis. By this means, a sample of perfusion fluid can be held in contact with the tubule epithelium for any desired length of time without it becoming contaminated with free-flow fluid lying else-