Discussion. Quelle que soit la méthode utilisée pour la préparation des synaptosomes on avait constaté un certain degré de contamination par les mitochondries de différentes tailles. La présence de lysosomes n'avait pas été mentionnée jusqu'ici. Nos résultats montrent une contamination importante de la fraction synaptosomale par des lysosomes intacts.

Summary. Subcellular fraction, enriched with synaptosomes, obtained from rat brain has been found contaminated by lysosomes, as evidenced by the high content of acid phosphatase, their biochemical marker.

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Reduction of Hypoxia-Induced Disturbances by Previous Treatment with Benserazide and L-Dopa in Rats

Improvement of the performances during hypoxia after treatment by an inhibitor of dopa-decarboxylase (benserazide) and L-Dopa strongly suggests a role of catecholamines[1]. Central catecholamines may alter either cerebral blood flow and/or vegetative responses induced by hypoxia, since these catecholamines are known to inhibit central pressor effects provoked by stimulation of carotid sinus[2,3]. In this study, we attempted to determine whether hypoxia causes any modification of cerebral blood flow estimated by rheoencephalography, of spontaneous motility, and level of cerebral catecholamines.

Methods. Long Evans female rats, 3 months old, were used. Every measurement was performed in an hypobaric chamber. Each parameter (pO_2, pCO_2, pH, rheoencephalogramm, motility, central dopamine and norepinephrine) was registered at 760 mm Hg, then at a pressure of 500 mm Hg (4200 m), later on at 300 mm Hg (7300 m) and finally when back to atmospheric pressure. Each experiment started 1 h after administration of L-Dopa. 2 groups were studied: a control one (NaCl 0.9%) and a treated one (RO 4 4.602: 50 mg/kg i.p., L-Dopa 100 mg/kg i.p. 1 h later). Each animal's motility was recorded by a photoelectric actimeter. Cerebral flow was registered in anaesthetized rats (pentobarbitone 35 mg/kg i.p.) by means of impedance plethysmography with 2 electrodes fixed on temples. Simultaneously, heart rate was recorded (ECG); pO_2, pCO_2, pH were registered in arterial aortic blood of anaesthetized rats.

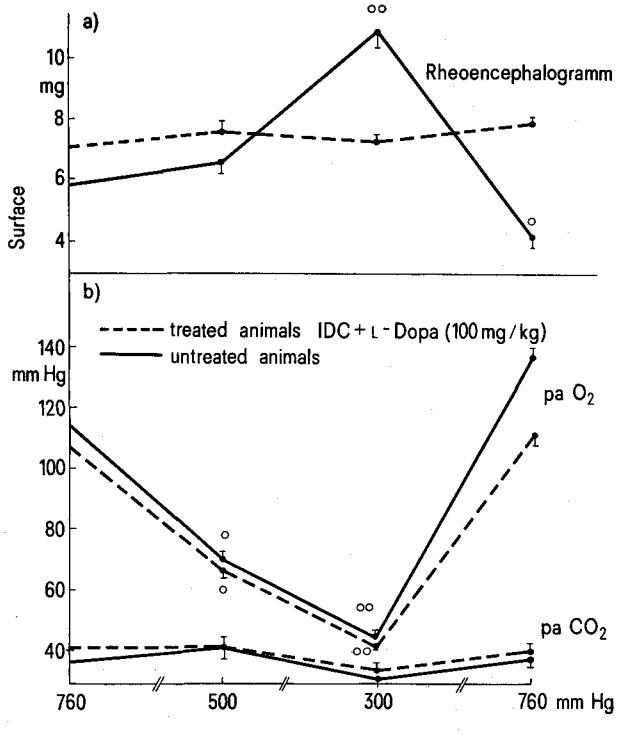

Fig. 1. a) surface of rheoencephalogramms ± SE (mg of paper) measured at variable pressures (760, 500, 300 mm Hg and back to 760 mm Hg) (10 animals per group). b) pO_2 and pCO_2 at he same different values of pressure. $^{\circ}p < 0.05$. $^{\circ\circ}p < 0.01$ (statistical significance calculated by appared couple method and *t*-test inside).

Results and discussion. In control animals, measurements of blood parameters showed that the diminution of pressure chosen (300 mm Hg) induced a sufficient hypoxemia (45 mm Hg) without any modification of pCO_2. Therefore, a normocapnic hypoxemia has been realised and an influence of possible changes of CO_2 on cerebral blood flow seems to be ruled out. But it is important to notice that the blood gas values are not changed after treatment by benserazide and L-Dopa. Thus, the hypothesis of an interaction of the treatment upon animal's ventilation can be rejected.

In the control group, hypoxia induced an elevation of the rheoencephalogramm without any change in heart rate. The return to atmospheric pressure provoked an increase of pO_2 and a decrease of rheoencephalogramm which reached a lower value than the initial one.

Pretreated animals (benserazide + L-Dopa 100 mg/kg i.p.) show very different hemodynamic modifications (Figure 1): these animals' rheoencephalogramms were unchanged. Therefore, administration of benserazide and L-Dopa appeared to abolish interactions of hypoxia on cerebral blood flow. It seems to work as if L-Dopa inhibited the chemoreceptors[2,3]. Administration of benserazide and L-Dopa induced a diminution of motility, which is not observed[4,5] after treatment by benserazide alone. A reduction of motility is also provoked by hypoxia alone. The decrease in the reduction of motility after hypoxia in pretreated rats (benserazide and L-Dopa) might be ascribed to the central effects of L-Dopa[1] (Figure 2).

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Dopamine and norepinephrine brain levels (ng/g of brain) at 760 mm Hg, 300 mm Hg and return to 760 mm Hg

Treatment	760 mm Hg		300 mm Hg		Return to 760 mm Hg	
	DA	NE	DA	NE	DA	NE
Control group	242 ± 58	55 ± 10	227 ± 43	52 ± 14	263 ± 65	36 ± 9
Treated animals (benserazide 50 mg/kg and L-Dopa 100 mg/kg)	3488 ± 648	299 ± 60	1551 ± 566[a]	196 ± 80	2135 ± 700	296 ± 106

[a] $p < 0.05$ (difference between levels at 760 and 300 mm Hg).

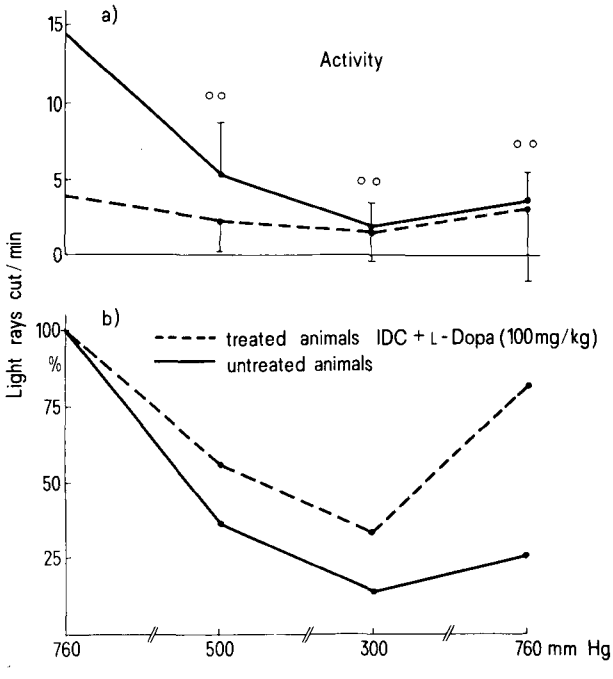

Fig. 2. Spontaneous activity measured at different pressures (10 animals per group). a) The parameters are represented in counts/min (± SE). $^{o}p < 0.05$. $^{oo}p < 0.01$ (appared couples and t-test). b) The parameter was represented in percent of its initial value.

In control animals, dopamine and norepinephrine central levels are unchanged by hypoxia or return to atmospheric pressure (Table) according to previous results[6]. In treated animals, the dopamine level falls down during and hypoxia and the norepinephrine level comes back to its initial values after return to atmospheric pressure. This high level of norepinephrine during post-hypoxic period is probably responsible for return go initial value of the motility[7]. The results of treatment by benserazide and L-Dopa indicate a stabilization of the rheoencephalogramm and a decrease in the reduction of spontaneous motility induced by hypoxia. Overdoses of cerebral dopamine and norepinephrine seem to be the origin of these phenomenons.

Summary. Pretreatment by benserazide (50 mg/kg i.p.) and L-Dopa (100 mg/kg i.p.) in rats induces a reduction of the diminution of motility after hypoxia and a stabilization of cerebral blood flow during and after hypoxia. An overload of cerebral dopamine and norepinephrine seems to be the original process of this phenomenon.

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Prostaglandin-Induced Choleresis in the Rat

Although prostaglandins (PG) affect many biological processes, PG E_2 failed to alter bile flow in the dog[1] and in the isolated perfused rat liver[2]. In view of its ATPase inhibitory properties[3] and the hypothetical role of Na^+-K^+-ATPase in bile formation[4], PG A_1 would appear to be a more likely candidate to influence bile secretion. The effect of PG A_1 on bile flow was, therefore, studied in anesthetized rats. The results demonstrate that intraportal infusion of PG A_1 significantly increases bile salt independent bile formation.

Materials and methods. Studies were performed in 6 male Wistar rats weighing 232 to 263 g which had free access to food (Altromin R) and water. Under pentobarbital anesthesia (5 mg/100 g, i.p.) the femoral vein and artery were cannulated with PE 50 polyethylene tubing for infusion of taurocholate or saline and recording of the blood pressure, respectively. From a midline incision the common bile duct was cannulated with PE 10 tubing just below the bifurcation in order to prevent drainage of pancreatic secretion. Bile was collected in 10 min periods and weighed. For the infusion of PG A_1, a PE 10 catheter was inserted into the portal vein via the gastroduodenal vein. After a control period of 30 min during which 0.9% NaCl (3.3 ml/h) was infused into the portal vein, a solution of PG A_1 was infused for 30 min at a similar rate in a concentration yielding 1 µg PG A_1/min/100 g body wt. The portal vein was chosen as the

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