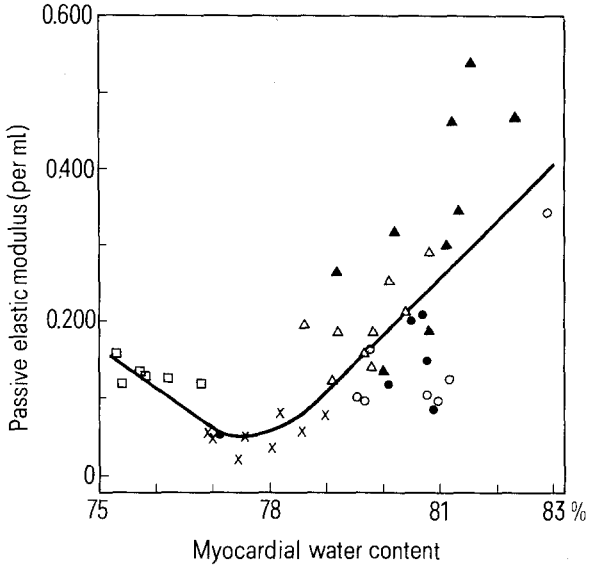

Bilinear correlation ($y = 2.210 - 0.027\ x + 0.072 \cdot \log(\beta \cdot 10^x + 1)$ where $\log \beta = -77.76$; $r = 0.685$) between the values of tissue water content and passive elastic modulus in the left ventricular myocardium. X, control animals; □, pancreatectomized animals suffering from myocardial dehydration caused by hyperosmolality; ○, animals receiving noradrenaline 2 h after coronary artery ligation; ●, animals receiving noradrenaline 48 h after coronary artery ligation; △, animals with empty beating hearts during cardiopulmonary bypass; ▲, animals with empty fibrillating hearts during cardiopulmonary bypass. The last 4 groups of animals were suffering from myocardial oedema.

since ventricular performance was found to decrease in cases where diastolic stiffness increases, i.e. the compliance of the ventricular wall is diminished[8,10-14].

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Changes in brain oxidative metabolism in rats with portocaval shunt

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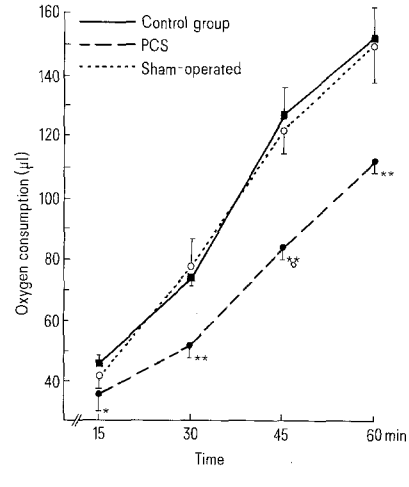
Summary. Oxygen and glucose consumption in brain slices of rats were found to be diminished after 7 days of portocaval shunt. This decrease may be an index of early metabolic alterations produced by the operation.

Hepatic encephalopathy has been classified as acute, chronic (or porto-systemic) and acute-on-chronic[2]. A number of etiopathogenic theories have been proposed for hepatic encephalopathy but many of them have been studied in acute hepatic coma[3] or in acute-on-chronic encephalopathy, clinical or experimental[4].

One of the metabolic abnormalities considered in acute hepatic coma of more than 24 h' duration is the decrease of brain oxygen consumption and glucose utilization[5]. Equally, in the human clinical picture of early chronic portosystemic encephalopathy it has been shown that brain oxygen consumption is reduced and Levodopa treatment produces an increase in brain oxygen consumption and an improvement in the O_2/glucose rate[6].

The object of this paper is to study changes in the cerebral oxidative metabolism after 7 days of portocaval shunt (PCS) in order to study the early variations in control and sham-operated rats.

Material and methods. 18 male Sprague-Dawley rats weighing 250–300 g were used, divided into 3 groups: a) Controls; b) rats after 7 days of porto-caval shunt and c) sham-operated rats. The porto-caval shunt was made by the technique of Arias et al.[7].



Oxygen consumption variations in brain cortex slices of rats, 7 days after portocaval shunt (PCS), compared with control and sham-operated rats. The measures were made 15, 30, 45 and 60 min after the preparation was stabilized. * $p < 0.05$; ** $p < 0.01$.

The forebrain of the animals was rapidly removed and put into a beaker with incubation medium 0-4 °C (Cl = 131.5 mM, PO_4H = 16.58; SO_4 = 1.22 mM; Na = 139.55 mM; K^+ = 4.79 mM; Ca^{++} = 2.73 mM; Mg^{++} = 1.21 mM and glucose = 9 mM). Cerebral slices were made by hand following the McIlwain technique (weight = 50 ± 10 mg). Oxygen consumption was measured, using a manometric technique[9]. Readings were made directly at 15, 30, 45 and 60 min. The glucose consumption was determined by the enzymatic method using glucose oxidase[10]. Statistical comparison of results was made by the Snedecor test[11].

Results. Results obtained are presented in the figure. In PCS rats O_2 consumption is significantly diminished (25-30%) throughout the experiment with respect to controls and sham-operated rats. Differences in O_2 consumption between control and sham-operated rats are not found. Glucose utilization diminishes (55%) with respect to controls and sham-operated rats (0.67 mg glucose/100 mg tissue) in PCS rats.

Discussion. The reduction of cerebral oxidative metabolism in PCS rats 7 days after operation is an index of early energetic alterations produced by PCS in the central nervous system. This decrease may be a consequence of a depletion of catecholamines[12] which could be caused by accumulation of false neurotransmitters. Based on these results, the Levodopa treatment of hepatic coma[6] which increases the catecholamine synthesis, may produce a displacement of false neurotransmitters[4] as well as a cerebral oxygen consumption increase and glucose utilization when Levodopa is administered in early PCS encephalopathy.

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Intestinal glucose absorption in rats after secondary infections with *Nippostrongylus brasiliensis*

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Summary. A challenge infection of *Nippostrongylus brasiliensis* in immune rats resulted in an earlier onset of intestinal glucose malabsorption and increased glucose metabolism compared with rats receiving a primary infection. Intestinal absorption and metabolism recovered to control levels earlier during a secondary infection. The pattern of changes in absorption and metabolism was probably related to host immunological activity.

Studies of host intestinal glucose absorption after secondary infection with intestinal nematode parasites have produced differing results. In guinea-pigs receiving a secondary infection of *Trichinella spiralis* glucose malabsorption and the onset of pathological changes in the gut occurred earlier than after primary infection[1]. In contrast, mice failed to show any change in glucose absorption after secondary infection with *T. spiralis* although their mortality and worm

Glucose absorption by the small intestine was measured in vitro using a technique described in detail elsewhere[5]. The entire small intestine was removed from ether-anaesthetized rats, everted and divided into 3 segments of equal length to facilitate the subsequent procedures. Each segment was made into a sac and filled with test solution (Krebs-Ringer bicarbonate solution containing 10 mM D-glucose and gassed with oxygen containing 5% CO_2) and incubated for 1 h at 37 °C in 30 ml test solution in a 150-ml