156 Experimental Results of in vivo Laser Excited NADH₂-Fluorescence Measurement on Acute Liver Ischaemia with Radical Scavenger Protection

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Complete interruption of blood flow to the liver during surgical intervention for extensive resection and liver tx causes a temporary damage of the liver function. Therefore it is necessary to administer stabilizing drugs.

In order to evaluate cell damage and the deficiency of energy the time-dependent intensity of NADH₂-surface-fluorescence at 470 nm is measured by a newly developed fluorometric device.

Method. Liver ischaemia was induced by crossclamping for 30 min the hilar pedicle in six groups of rats (control; ischaemia alone and ischaemia with radical scavenger), treated by ascorbinic acid, tocopherol, allopurinol, DFR and prop. carnitine.

During ischaemia typical trace curves of fluorescence intensity could be measured whereby the application of radical scavengers shows a marked protective effect on liver tissue. The most durable effect is achieved by DFR and allopurinol as well as by tocopherol.

The laser excited in vivo NADH₂-fluorescence intensity on the liver surface represents an instant sensitive marker of liver cell ischaemia.

157 Hepatic Regeneration in Ischemic Liver: Effect of Cyclosporin A

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Two years ago in Bologna we showed our results on the enhancing effect of CyA on hepatic regeneration. We have tested the ability of this drug to produce the same effect on the regenerative response of the ischaemic liver.

Material and Methods. In 23 female Sprague-Dawley rats (200 g) a 70% hepatectomy was carried out under light ether anesthesia. Liver ischemia was performed in 16 animals by clamping celiac and superior mesenteric arteries and the hepatic pedicle for 15 min just after completing 70% hepatectomy. 7 of these 16 rats were treated with CsA i.p. (20 mg/kg) the day before and 2 h prior to surgery. Animals were sacrificed 24 h after hepatectomy. Hepatocytic DNA content was measured by means of microspectrocytophotometry.

Results are presented in terms of percentage of hepatocytes undergoing regeneration (% R.H.) and mean values of DNA content in regenerating hepatocytes (M). (1) 70% hepatectomy: n=7, % R.H. = 17.83, M=1.64. (2) 70% Hepatectomy plus ischemia: n=9, % R.H. = 5.77, M=1.482. (3) 70% hepatectomy plus Ischemia plus CyA: n=7, % R.H. = 10.80, M=2.311.

Conclusions. (1) ischaemia reduced the percentage of regenerating hepatocytes (p=0.001) but did not affect their mean DNA values (p=0.25). (2) CyA treatment increased the percentage of regenerating hepatocytes (p=0.028) without reaching normal values (p=0.013). (3) It also induced an increase in mean DNA content of regenerating hepatocytes when compared both with control (p=0.0003) and ischaemic groups (p=0.0039).

158 Hypoxia and Reoxygenation after Temporary Occlusion of the Liver Hilus

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In the relevant literature, data about the tolerable limit of liver hilus occlusion vary between 30 and 75 min, but objective criteria for estimating ischemic liver cell damage are lacking. The present study attempts to verify the extent and pattern of such injuries by analysing the changes occurring in macrocirculation and hepatic microcirculation during hypoxia and reoxygenation.

Materials and Methods. 21 mongrel dogs (mean b.w. 25.1 kg) were operated under neuroleptanesthesia. After isolation of the hilar structures, occlusion was performed by tourniquet, in group I for 30 min, in group II for 45 min, and in group III for 60 min. Heart rate (HF), arterial pressure (Part), pulmonary pressure (Ppulm), central venous pressure (CVP), portal venous pressure (PVP), and blood flow in the hepatic artery and portal vein were continuously monitored. Local tissue pO₂ was measured by a multiwire surface electrode (Kessler and Lübbers) on the left hepatic lobe. The parameters arterial and mixed venous blood gases, liver enzymes, and local tissue pO₂ were assessed at fixed intervals before and during the Pringle manoeuvre and after reoxygenation.

Results. Macrocirculatory changes show no significant differences dependent on the duration of hypoxia. The increase of hepatic enzymes was delayed, but significant differences between group II and III become only apparent 24 h after the occlusion. Continuous measuring of tissue pO₂ verified rapid and homogenous