hepatocytes was shown to react regularly to changes taking place in the body during temporary ischemia of the limbs, and the response depended on the duration of ischemia. Changes in hepatocyte dehydrogenase activity took the form of an increase in the early stages of the experiments, followed by a marked decrease.

In the postischemic period levels of hepatocyte dehydrogenase activity exceeded the control values only on restoration of the blood flow after ischemia of the limbs for 3 h. Later restoration of the blood flow in the limbs led to a decrease in hepatocyte dehydrogenase activity by a greater degree compared with the corresponding ischemic period.

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SCOPE FOR SOME ENZYMIC TESTS AND FOR RADIONUCLIDE HEPATOGRAPHY IN THE DIAGNOSIS OF EARLY CHANGES IN LIVER FUNCTION

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KEY WORDS: Serum enzymes; liver; tetrachloromethane; hepatogram

The writers showed previously [3] that liver scanning after tetrachloromethane poisoning in rabbits is not the most suitable method of detecting mild and early parenchymatous changes.

TABLE 1. Activity of Serum Enzymes (in I.U./ml) ChE, AlP, Ald, and LAP in Rabbits before and after Tetrachloromethane Poisoning (M \pm m)

		· · · · · · · · · · · · · · · · · · ·		
Exptl. conditions	CLE	AIP	A1d	LAP
Control (normal				
rabbits)	1342 ± 269	33,0±3	142±22	16,5±1,6
48 h after CCl ₄ poisoning	418±43	44,0±7,3	248±42,7	15,0±2,2
poisoning 15 days after CCl ₄ poisoning	287 ± 32	41,0±4,2	42±7,7	13,0±2,1

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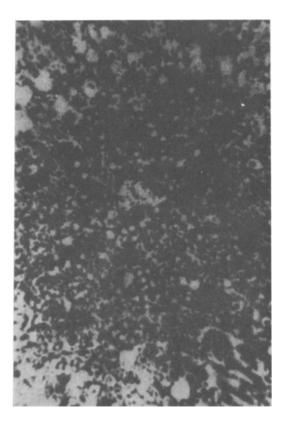


Fig. 1. Histologic section through liver of rabbit No. 9 15 days after tetrachloromethane poisoning: high degree of vacuolar parenchymatous fatty degeneration, affecting mainly cells located in center of liver lobules (intermediate zone). Hematoxylin and eosin, $250 \times$.

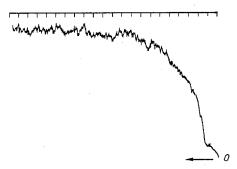


Fig. 2. Hepatogram of same rabbit before CCl4 poisoning: rapid rise of radioactivity in liver with peak accumulation of radioactive colloid after 10 and 12 min, normal phagocytic activity of reticuloendothelial cells. Ordinate, radioactivity (in relative units). 0) Reference point for counting. Speed of automatic writer 15 mm/min, time constant 30 sec.

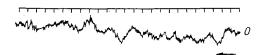


Fig. 3. Hepatogram of same rabbit on 15th day after poisoning: low curve, reduced vascular segment, and prolonged maximum of accumulation of radioactive colloid, exceeding 16-20 min; disturbed phagocytic activity of reticuloendothelial cells. Legend as to Fig. 2.

Besides liver scanning, other radionuclide methods of diagnosis of liver diseases also are used clinically, such as investigation of the hepatic blood flow and of hepatocyte function [1, 6-9]. The Kupffer cells of the liver remove colloidal radioactive gold from the plasma if the particle size in solution exceeds 1 μ . Up to 90% of injected radioactive colloid accumulates in the liver, the rest in the spleen, bone marrow, and other parts of the reticuloendothelial system [11, 12].

In this investigation the sensitivity of some enzymic tests and of radionuclide scanning was investigated for the purpose of detecting the earliest and mildest changes in hepatic function after tetrachloromethane poisoning.

EXPERIMENTAL METHOD

Ten Velikan (Giant) rabbits weighing 3.5-4.5 kg were used. A blood sample was taken from the auricular vein of each rabbit, and activity of the following plasma enzymes was determined: cholinesterase (ChE), alkaline phosphatase (AlP), aldolase (Ald), leucine-aminopeptidase (LAP), using kits of reagents from Boehringer (West Germany). An injection of 196 Aucolloid with activity of 0.75 MBq was given into the auricular vein for isotope scanning of the liver (hepatography). A Picker (USA) scanner, equipped with a suitable collimator for experimental purposes, was used. After normal scans had been obtained, the rabbits were given tetrachloromethane (CCl4) in a dose of 0.1 ml/100 g body weight, mixed with sunflower oil in equal volumes, so as not to irritate the gastric mucosa. CCl4 has a mild anesthetic action on rabbits, and prolonged inhalation of its vapor leads to serious injury. On oxidation of CCl4, phosgene is obtained [4]. Enzyme activity was again determined after 48 h and 15 days and the hepatogram was recorded. By investigation at these two different times it was possible to record the earliest and latest injuries to the liver cell produced by CCl4.

After 15 days the rabbits were killed and pieces of different parts of the liver were removed for histological investigation, by the classical methods.

EXPERIMENTAL RESULTS

It will be clear from Table 1 that after liver damage by CCl₄ considerable changes in plasma enzyme activity were found after 48 h. ChE activity was sharply reduced and this tendency continued until the 15th day. This enzyme is considered to be specific for the liver, for it is synthesized only in the hepatic parenchyma [2, 10]. The degenerative and necrotic changes taking place in the liver cell, revealed by hisotopathologic investigation (Fig. 1) were evidently the main cause of the disturbance of ChE synthesis. Ald activity was considerably increased 48 h after administration of CCl₄, but later it fell although without reaching its initial level. AlP activity also was increased on account of the hepatic isozyme AlP-1 [5].

Changes in LAP activity were very slight and were not statistically significant. These results indicate that the biliary system of the liver is not injured after tetrachloromethane poisoning, for this enzyme is highly specific for that system.

In normal rabbits during the first few seconds of recording the hepatogram a rapid increase of radioactivity was observed in the liver, in the vascular segment of the hepatogram, reflecting entry of the radioactive indicator into the blood vessels of the liver. This was followed in the course of 4-10 min by a slower increase of radioactivity, the parenchymatous segment of the hepatogram, reflecting the rate of absorption of the radioactive colloid in the liver, i.e., the phagocytic activity of its reticuloendothelial cells. Peak accumulation of the isotope and a subsequent plateau were observed after 10-12 min (Fig. 2). No significant changes in the hepatogram were found 48 h after poisoning.

Changes reflected in a lower curve were recorded 15 days after poisoning: a reduced vascular segment and a more prolonged maximum of accumulation of the radioactive colloid, exceeding 16-20 min (Fig. 3). The phenomenon described can be explained by disturbed functional activity of the reticuloendothelial cells as a result of their toxic damage by CCl₄.

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EFFECT OF ACUPUNCTURE ON ACTIVITY OF EXPERIMENTAL HIPPOCAMPAL EPILEPTOGENIC FOCI IN RABBITS

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KEY WORDS: hippocampus; epileptogenic focus; acupuncture; enkephalins.

Acupuncture is being used on an ever-widening scale in clinical practice as one of the most active methods of treatment of various diseases not involving the use of drugs [3, 4]. Acupuncture is used in particular for the treatment of epilepsy, but there are as yet no clinical or experimental data to confirm the antiepileptic effect of acupuncture and to justify its use in paroxysmal states.

The aim of this investigation was to study the effect of acupuncture on activity of experimental epileptogenic foci in model experiments on animals.

EXPERIMENTAL METHODS

Experiments were carried out on 32 chinchilla rabbits, mainly females, weighing 2700-3200 g, anesthetized with pentobarbital. Bipolar chemical electrodes were implanted into the dorsal hippocampus of the rabbits on the left and right sides, in accordance with stereotaxic coordinates. Each chemical electrode consisted of: 1) a cannula-needle 0.8 mm in diameter through which solutions of substance could be injected into the brain, and 2) a nichrome wire (0.25 mm in diameter), insulated with transplant plastic and glued to the cannula. Brain potentials were recorded on an ÉEG4P-02 4-channel electroencephalograph. The experiment in which the EEG was recorded the first time was carried out on the 6th day after the operation. All subsequent experiments with a particular rabbit were done at 2-day intervals. An epileptogenic focus was formed in the rabbit hippocampus by injection of 0.0001 ml (100 U) of a solution of the sodium salt of penicillin through the chemical electrode by

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