The Determination of Copper and Manganese in the Human Liver by Neutron Activation Analysis

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Neutron-activation analysis of copper and manganese was performed both by nondestructive (Cu-66) and chemical separation (Cu-64 and Mn-56) methods and tried to establish the method of choice, Biopsy and autopsy liver samples obtained from 10 cases of Wilson's disease, 5 cases of hemochromatosis and 8 normal cases were studied.

Livevr samples (1~100 mg dry weight) were irradiated for 10 min.~4 hrs. using TTR-1 100KW swimming pool reactor with a thermal neutron flux of 3×10^{11}n/cm^{2}sec.

In chemical separation method, copper was separated with almost 100% yield by internal electro-deposition technique, and then manganese with 90% yield by usual precipitation method. The separation process for copper was completed within 30 min., and for manganese 60 min.

Comparison between destructive and chemical separation methods for copper analysis showed well correlation in Wilson's autopsy liver specimens stored in formalin. However, fresh liver specimens obtained by needle biopsy could be analysed only by chemical separation method, because of the presence of plenty amount of sodium.

The hepatic copper content was found to be abnormally increased in Wilson's disease (88~720μg/g dry weight) and also in hemochromatosis (100~2500μg/g). normal range being 2~μg/g.

The amount of hepatic managnese in hemochromatosis was 4.1~17μg/g dry weight. showing significant increase compared with normal range of 1.1~3.4μg/g.

In conclusion, activation analysis of trace elements in the human liver proved to be useful tool for medical research and diagnosis. By the combination of rapid cehmical separation method with thermal neutron irradiation even the needle biopsy sample (e.g. lmg) could be sucessfully analysed. In Wilson's disease increased level of hepatic copper and in hemochromatosis significant increase in both copper and manganese were observed.

Influence of the Spleen on the Clearance of Intravenously Administreted Colloid

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In ^{198}Au colloid liver scan, the spleen is visualized in various hepatic diseases, and frequently in liver cirrhosis. Different degrees of liver injuries were produced in rats by injection of carbon-tetrachloride, and several experimental studies were carried out on these rats to clarify the cause of splenic visualization.

Au colloid clearance rate became lower after splenectomy and more markedly in $CC1_4$ treat-

ed rats. In normal rats, however, hepatic uptake was not influenced by splenectomy. The uptake of colloid by the spleen and bone marrow was slighty decreased in the group in which colloid was slowly infused into the mesenteric vein compared with that after injection into the penile vein.

After partial hepatectomy, the uptake of colloid by the spleen and bone marrow was not altered, but the hepatic uptake per gram increased two fold. This finding suggests that the spleen and bone marrow have a limited capacity for colloid uptake in the normal state.

In the rats with advanced liver injury, colloid clearance rate was delayed with decreased hepatic uptake, while the spleen and bone marrow were found to have increased capacity to take up the colloid. Between the two groups in which the colloid was infused into the mesenteric vein or into the peripheral vein, no difference was noted in the uptake by the spleen and bone that collateral circulation was present in these CCl_4 treated rats. In these rats, splenic colloid uptake per gram was signicantly elevated.

These studies might suggest that, in liver cirrhosis splenic cisualization in liver scan is due to an increased capacity of the spleen to clear intravascular colloids.

Increased Splenic Blood Flow of Idiopathic Portal Hypertension (IPH) Measured by Kr-85 Clearance Method

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By means of radioactive inert gas, Kr-85, measurement of splenic blood flow was carried out on the 7 cases with idiopathic portal hypertension (I.P.H.), 6 liver cirrhosis, 2 splenomegalic liver cirrhosis, 2 Wilson's disease and control subjects.

Splenic blood flow, expressed as ml per minutes per 100g was determined by a Kr-85 clearance technique after introduction of Kr-85 saline solution through the selective catheterization of splenic artery. Total splenic blood flow was calculated by multiplying splenic blood flow per unit weight by the splenic weight measured after operation or estimated by the splenic scintigram.

The mean splenic blood flow per ml per 100g of tissue was 118.66 ± 29.1 ml/min in control, 115.7 ml/min in IPH, 146.5 ml/min in Wilson's disease, 106.5 ml/min in splenomegalic liver cirrhosis and 65.4 ml/min in liver cirrhosis.

The mean total splenic blood flow was 515.8 ml/min in IPH, 1013 ml/min in Wilson's disease, 604 ml/min in spleromegalic liver cirrhosis and 157 ml/min in liver cirrhosis. The estimated total splenic blood flow in normal cases was 143.3 ± 34.4 ml/min.

Both splenic blood flow per 100g and total splenic blood flow were increased in IPH, splenomegalic liver cirrhosis and Wilson's disease as compared with liver cirrhosis. But the splenic blood flow per 100g of tissue in IPH, Wilson's disease and splenomegalic liver cirrhosis was about the same as controls.

On this report, authors emphasized that increased splenic blood flow seemed to play an important role in the etiology of splenomegally and portal hypertension of IPH, splenomegalic liver cirrhosis and Wilson's disease.