

which showed histological evidence of infarct and elevated ^{99m}Tc activity. Yet, there was no consistent relationship between ^{99m}Tc and ^{201}Tl activity. (3) Relationship between ^{99m}Tc -PYP activity and Ca: Ca increased in the segment which showed elevated ^{99m}Tc activity. However, there was no linear relationship between ^{99m}Tc activity and Ca.

In summary, none of these three factors appears

to be solo determinant of ^{99m}Tc -PYP distribution. It seems likely that not only the absolute value of Ca but also composition and physicochemical properties of tissue calcium is important for the accumulation of ^{99m}Tc -PYP. Further study is warranted to make better understanding of relationship between ^{99m}Tc -PYP accumulation and calcium kinetics in infarcted myocardium.

Technetium-99m: 3-Hydroxy 4-Formyl Pyridine: Glutamic Acid Complex. A New Rapid Cholescintigraphic Agent

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Scintigraphic imaging of the hepatobiliary system has been significantly improved with development of Tc-99m labelled compounds.

Tc-99m; 3-hydroxy 4-formyl pyridine (HFP): glutamic acid (G) is a non-toxic radiopharmaceutical that was found to undergo rapid biliary excretion in normal rabbits. A new Tc-99m-HFPG was prepared by heating an aqueous solution (PH 6.5–7.5) of HFP, G, and pertechnetate-99m for 15 min. at 100°C. The yield of Tc-99m-HFPG was in the order of 90–100%. The simple method may be applicable for a kit preparation.

A safety assessment in mice (20 g) was made using HFPG complex without added Tc. In the mice a single intravenous dose of 187 mg/kg was non-toxic and caused no gross behavior or pathologic changes. These doses represent a 1,000–2,000 times excess over the probable human dose in the intended diagnostic application. The biliary trees

and gallbladder were seen within 20 min. of Tc-99m-HFPG injection and by 25 min. marked accumulation of radioactivity was noted in the gallbladder and intestinal tract. While the gallbladder was clearly visualized by 15 min using Tc-99m-HIDA, 40 min: Tc-99m-pyridoxylideneglutamate (PG), 30 min: Tc-99m-pyridoxylideneisoleucine (PI). Blood clearance in the rabbits: The Tc-99m HIDA have lower blood levels than another complexes. The rabbits weighing approximately 2.5–3.0 kg were surgically prepared to allow bile samples to be collected. The cumulative per cent dose in the bile at 1 hour were 38.8% of the injected Tc-99m-HFPG, 42.2% of Tc-99m-PG, 56% of Tc-99m-HIDA, 57.8% of Tc-99m-PI.

In summary, Tc-99m-HFPG appears to be one of the suitable agent of low toxicity for the investigation of biliary tract disorder.

Effect of the Chemical Structure and Plasma Lipoprotein Binding Properties on Adrenal Accumulation of Radiohalogenated Sterols

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The properties of rapid incorporation and long-term retention in adrenal make 19-iodocholesterol (CL-19-I) (Counsell et. al.) and 6 β -iodomethyl-19-norcholest-5(10)-en-3 β -ol (NCL-6-I) (Kojima

et.al.) to be successful adrenal scanning agents. We have undertaken to prepare the following radiohalogen derivatives of cholesterol and to compare their behaviours in animal body: (1) CL-19-

^{131}I , NCL-6- ^{131}I , their 3-acetate, NCL-6- ^{77}Br , ^3H -CL, (II) Cholesteryl- ^{18}F , ^{77}Br , ^{131}I , (CL-3-X), ^3H -CL-3-X, (III) 3-Acetoxy-5-OH-6-(^{18}F , ^{77}Br)-cholestane (6-Halohydrin), 5-Halohydrin, 5-Br-hydrin-3- β -ol.

The radiochemical purity of these compounds was checked by thin layer chromatography.

These compounds were administered intravenously to C3H mice and Wistar rats with the aid of an emulsifier such as polyoxyethylene hydrogenated castor oil. Adrenal, liver, kidney, and blood concentrations were measured at 2, 6 hours and 1, 2, 3, 5, days after injection. Blood of injected rats was divided into several components; RBC, non bound fraction, and plasma protein bound fraction.

The comparison of adrenal uptakes between NCL-6-I and NCL-6-Br, and among CL-3-F, Br, I showed that iododerivatives accumulated significantly higher than bromo- and fluoro compounds. Except for CL-19-I, CL-3-F, and fluoro-hydrin, the adrenal concentrations of all other compounds increased monotonously until 2 or 3 days, thereafter the highest concentrations were retained in adrenal. On the contrary, the adrenal uptakes of CL-19-I and fluoro-compounds reached the peak shortly after injection, then decreased gradually.

In contrast to Counsell's result (J.Nucl.Med; 14, 777, 1973), CL-19-I-3-Ac and NCL-6-I-3-Ac showed almost the same adrenal uptakes as NCL-

6-I in rats. Percent dose per gram of adrenal on 3 days of CL-19-I-3-Ac, NCL-6-I-3-Ac, NCL-6-I, and CL-19-I was $54.4 \pm 5.8\%$, $59.7 \pm 4.6\%$, $50.0 \pm 11.3\%$, and $4.54 \pm 0.97\%$ respectively. Therefore the 3-hydroxy group in cholesterol is not necessary for adrenal accumulation. And 19-nor structure has not always higher affinity to adrenal than 19-methyl structure. In accordance with Szinai et. al. and Yu et. al.'s results, both CL-3-I and CL-3-Br concentrated in the mouse adrenal on day 2, 2.2 times and 1.9 times greater than CL-19-I.

By means of low-speed centrifugation and dialysis of injected rats' bloods, it was shown that all the compounds examined were bound to RBC and non-dialysable protein. Density gradient ultracentrifugation study revealed that such compounds as CL-19-I, MCL-6-I, their 3-acetates, and CL-3-I were not bound to either albumin nor high density lipoprotein, but mainly bound to low density and very low density lipoproteins. The β -lipoprotein binding of halogeno-sterols is the impressive general feature, which is quite different from cholesterol. The fraction of RBC to whole blood activity was quite different among compounds. RBC to whole blood ratio of CL-3-F was about one third of CL-3-I and one fifth of NCL-6-I, for example. We suppose that measuring of β -lipoprotein and RBC bindings of a compound may be useful as an *In Vitro* screening test for adrenal scanning agents.

Rectal Administration of ^{13}N -ammonia in Liver Diseases 2. its clinical use

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Ammonia arising from ingested ammonia salts or produced by bacteria in the gastrointestinal tract is absorbed into the portal circulation and is transported to the liver. Most of the ammonia is removed from the portal blood and converted to urea and glutamine, so that blood ammonia concentrations shouldn't rise appreciably in the systemic circulation. In the presence of liver disease, some of the absorbed ammonia reaches systemic

circulation through portosystemic shunts or as result of impaired metabolism in the liver. Elevated blood ammonia concentrations are frequently found especially in hepatic coma, so that the significance of ammonia as an etiologic factor has been evaluated. In the present study, ^{13}N -ammonia which was produced by NIRS cyclotron was administered intrarectally to 3 controls and 17 patients with liver disease in an attempt to investi-