lung.

Each specimens were weighed. Its radioactivity was counted with well type scintillation counter.

The radioactivity of periarterial and supraclavicular lymph nodes were 15,000~30,000 cpm/gm tissue and 10,000~20,000 cpm/gm tissue respectively.

Lactic dehydrogenase (abbr. LDH)—by Hess, Scarpelli and Pearse's method 1958, glucose 6 phosphate dehydrogenase (G6PD)—Pearse's method 1958 and succinic dehydrogenase (SDH)—by Nachlas et al method 1957 were stained in each tissue along with hematoxyline-eosin staining.

(2) Human Lymph Nodes: Lymphography with <sup>131</sup>I ethiodol (300-1,000 microcuries) was compared with that by usual contrast medium in tumors of bladder, testicles and kidney.

Lymph glands were removed at the time of operation and were stained for same enzymes as in the animal.

Results:

In rabbits, no marked differences were seen in H-E staining between control and <sup>198</sup>Au groups.

Lymph nodes with 10,000-15,000 cpm/gm tisue showed increased dehydrogenase activity in the order of SDH, G6PD and LDH.

The activity decreased in the lymph nodes of radioactivity 20,000-25,000 cpm/gm tissue. Very slight staining of dehydrogenases were noted in the lymph nodes of 30,000 cpm/gm

tissue.

In the human subjects, the activity of all the dehydrogenases increased in each lymph gland. This is probably because of low radioactivity in the lymphatic tissue. If a larger dosis or longer term administration of radioisotope were carried out, The activity of these enzymes would drop down as in the animal.

Discussion and Conclusion:

<sup>198</sup>Au was absorbed in spleen and liver as in lymph nodes. The dehydrogenases in these organs showed same activity changes as in the lymph node. Increased activity in the organs with low radioactivity would be explained as follows;

- (1) temporary stimulation of respiratory or/and glycolytic systems.
- destruction of organelles resulting in release of enzymes.
- relative increase of enzymatic activity due to insufficient substrate supply.

On the other hand, the relationship between phagocytosis and respiratory or glycolytic system should be considered, as Karnovsky reported. However, non-radioactive Au colloid was not used in our experiments. Further investigation is necessary to find out the cause of the increased enzymatic activity.

It would be of some use to know radioactive effect that dehydrogenase activity varies before the usual morphological demonstration shows some changes.

## In Vivo Behavior of <sup>131</sup>I-Urokinase

M. Murakami, K. Kurakane, M. Kuroda, K. Onchi, Y. Koshimura I. Hoshiba and M. Noto

Second Department of Internal Medicine, Kanazawa University School of Medicine

Urokinase (supplied by Midorijuji Co. Ltd.,) was labelled by the modified procedure of Helmkanpf's monochloride method. The free iodine unbound to urokinase was removed against isotonic saline solution for 24 hours at  $4^{\circ}$ C. The specific radioactigity of the labelled urokinase ( $^{131}$ I-UK) was 5 to 2  $_{\mu}$ C per Ploug unit of fibrinolytic activity. Paperelectrophoresis of  $^{131}$ I-UK was carried out in acetate buffer, 0.1M, pH 4.5. Four

bands were stained with nigrosin. No significant differences from unlabelled urokinase could be found. The densitometric peaks paralleled with radioactive peaks.

Urinary urokinase activity was measured by fibrinolytic area on 0.4% bovine fibrine plate of the crude urokinase fraction of 24 hour-urine collection stored at 4°C. The crude urokinase was extracted by the simplified procedure of Sgouris's. The radioactivity of this fraction and the same aliquot of whole urine was counted by well type scintillator.

Whole body longitudinal profile scannings were serially carried out at 5 minutes, 1.5 hours, and 24 hours after the injection of 80  $\mu$ C of <sup>131</sup>I-UK. The profile scannings were also carried out with RISA, <sup>131</sup>INa, and <sup>203</sup>Hg-Neohydrin, and these profiles of the scan were compared with that of <sup>131</sup>I-UK.

Materials were two normal subjects, a case of gout-nephropathy, and a liver cirrhosis with thalassemia.

In normal subjects, the highest peak of the profile of the scan at 5 minutes, was in the region of liver. At 1.5 hours, the peak was divided into two and they removed to kidney and urinary bladder. After 24 hours, there was very low radioactivity remaining in the reigion of thyroid gland and middle part of the body. The profils were similar to that of <sup>203</sup>Hg-Neohydrin except thyroidal uptake, and was far different from that of RISA and/or <sup>131</sup>INa. Urine-excreted radioactivity for 24 hours was 65% of injected activity, but the radioactivity of urokinase-fraction

was very slight (0.23%) and this was negligible.

In a case of liver cirrhosis, the behavior of <sup>131</sup>I-UK observed by the same external tracing was not so different from that of the normal subjects. His urinary excreted radioactivity and urokinase activity were the same as those of the normals.

In a case of gout-nephropathy, the behavior of <sup>131</sup>I-UK traced by the same procedure was much different except at 5 minutes. Urinary bladder peak of the profile scan was found little even after 1.5 hours and radioactivity after 24 hours remaining within his body was considerable. Urinary excretion of radioactivity was slight (6.9%). Urinary urokinase activity was as low as 8 per cent of normals.

From these results, our conclusions at the present time are as follows: (1) kidney plays a much greater role than liver does concerning the metabolism of urokinase. (2) It sems that urokinase is not a substance leaking unaffected through kidney into urine.

## <sup>131</sup>I-Fibrinogen Catabolism, Fibrinogenolysis and Fibrinolysis

M. Murakami, K. Kurakane, M. Kuroda, K. Onchi Y. Koshimura I. Hoshiba and M. Noto

Second Department of Internal Medicine, Kanazawa University School of Medicine

We presented the 1st paper of this study at the 2nd meeting of this association.

In this paper, influence on <sup>131</sup>I-fibginogen survival of fibrinolysis and/or fibrinogenolysis was observed.

Methods: Fibrinogen (supplied by Midorijuji Co., Ltd.) was iodinated by the iodine monochloride method of McFarlene's with a slight modification.

The fibrinogen labelled in this way had the same property as native fibrinogen on paper- and immuno-electrophoresis and thrombin clottability. Specific radioactivity was 80-50  $\mu$ C per mg. protein.

Turnover studies were carried out in 19 materials, including 5 normals and other cases of various diseases.

Pearson's theory was applied to this investigation. Plasama radioactivity was

counted of thrombin clottable fibrin per ml. of plasma. About the same plasma, fibrinogen concentration, thrombin time and euglobulin lysis time were measured.

Restuls: Normal values: plasma disappearance half life of <sup>131</sup>I-fibrinogen was 3.2-4.0 days; turnover rate, 52-mg./dl./day; half life of radioactively remaining in body, 5.2 days; fibrinogen councentration, 310 mg./dl; thromoin time, 20-28 sec; euglobulin lysis time, 15 hours.

In general, plasma disappearance of <sup>131</sup>I-fibrinogen was not influenced by plasma fibrinolytic and/or fibrinogenolytic activity. Between half life, turnover rate, plasma fibrinogen concentration, thrombin time and euglobulin lysis time, no definite correlation could be found.

We, then, grouped the materials into the