than male rats by 40–60%, and at night higher than noon by 16–18 fold in both sexes.

In P treated groups, the reductase activity increased at noon to 180% and 140%, for male and female respectively compared with respective control values. At night the activity of male was not altered significantly, although that of female increased 140%.

In S treated groups, the activity at noon increased to 140% and 180%, in male and female respectively, but decreased at night to 38% and 36%, in male and female respectively.

In D treated groups, the activity of male decreased to 80% and 60%, at noon and night respectively. In female the activity decreased to 40% but at noon increased 2 fold that of control value.

Plasma Pancreatic Glucagon in the Patients with Various Liver Diseases

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Plasma pancretic glucagon concentrations were determined in the patients with various liver diseases. After overnight fasting, the patients and normal subjects received an intravenous infusion of 30 g of l-arginine over a period of 30 min. Blood was withdrawn before and 15, 30, 60, 90, 120 min after the start of the infusion.

Plasma pancreatic immunoreactive glucagon (IRG) was determined by the radioimmunoassay with antiserum 30 K.

In the patients with acute hepatitis and liver cirrhosis, plasma IRG concentration in the basal state was almost three times greater than that observed in the control subjects. In the patients with acute hepatitis, chronic hepatitis and liver cirrhosis, plasma IRG response to arginine was significantly greater than in the control subjects.

In the patients with liver cirrhosis, the prolonged disappearance curve of injected exogenous glucagon was observed.

Correlation between the maximum concentration of IRG after arginine infusion and liver function tests in the patients with chronic liver diseases was studied.

The correlation between response of IRG and concentration of serum albumin was significant. But the correlation between response of IRG and another liver function tests was not significant.

Plasma Prolactin and Breast Cancer

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Plasma human prolactin concentrations were measured using a commercially available radioimmunoassay kit (CIS) in 46 patients with breast cancer and 42 female hospital controlls. Mean plasma human prolactin levels in female controlls 15.20 ± 8.05 ng/m*l*.

In 12 patients with primary breast cancer receiving radical mastectomy and postoperative irradiation (aged 30 to 71 years, mean 48.3 years), mean plasma human prolactin levels were 16.92 ± 12.05 ng/m*l*.

In 34 patients with advanced breast cancer (aged 33 to 60 years, mean 46 years), mean plasma human prolactin were 26.49 ± 26.72 ng/m*l*.

In 13 patients who received oophorectomy (aged 33 to 55 years, mean 43.3 years), mean plasma human prolactin levels were 35.62 ± 37.39 ng/m*l*.

In 17 premenopausal patients (aged 31 to 53 years, mean 41.9 years) mean plasma human prolactin levels were 19.24 ± 10.74 ng/m*l*.

In 11 postmenopausal patients (aged 52 to 72

years, mean 58.8 years), mean plasma human prolactin levels were 19.58±15.99 ng/m*l*.

Thyrotropin releasing hormone (TRH, 500 μg) was injected intramusculary in 25 patients with advanced breast cancer and 10 patients with primary breast cancer. Maximum responses were obsterbed in both groups one hour after injection. Mean value was 89.08±58.65 ng/m*l* and 67.84± 43.92 ng/m*l* respectively.

In generally normal persons, plasma human prolactin dose not exceed the value more than 30 ng/m*l*. Two of twelve patients with primary breast cancer showed high values more than 30 ng/m*l*. Eleven of thirtyfour patients with advanced breast cancer exceeded this value.

These results suggested that some patients had abnormal prolactin secretion.

In five patients who were treated with CB-154, plasma human prolactin levels were measured. After administration of CB-154, plasma human prolactin levels were decreased in all patients than their basal levels. Its mean maximum per cent decrease was 81.0% and its effect continued 10 hours after administration.

L-DOPA also lowered plasma prolactin levels but its effect disappeared four hours after administration.

A Method for Determination of Hypoxanthine-Guanine and Adenine Phosphoribosyltransferase by Electrophoresis and Thine-Layer Chromatogram Scanner

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The importance of salvage pathways for purine nucleotide biosynthesis became strikingly apparent with the discovery of the Lesch-Nyhan syndrome in 1964. The enzyme assay, however, was complicated. A simple method for hypoxanthine-guanine and adenine phosphoribosyltransferase, which was modified from the method of kizaki is presented. Reaction was carried out in a microtube using isotope labelled substrate and terminated by the addition of formic acid. An aliquot of the reaction mixtures was applied on cellulose acetate membrane over standard carrier substances and the products were separated electrophoretically using two buffer systems, 0.1 M borate buffer pH 9.0 and 0.1 M Tris-HCl buffer pH 7.5. The electrophoresis was performed as commonly used in a clinical laboratory, and the cellulose acetate membrane was scanned by thin-layer chromatogram scanner. We could determine these enzyme activities within 2 hours by this method. This simple and rapid method will be applied to the screening study of hyper uric-acidemia in the clinical field.