CURRENT LITERATURE

Coagulation time during hypothermia in man *by John P. Bunker and Robert Goldstein (1958)* : **Proc. Soc. Exper. Biol. & Med. 97,** *199*

Clotting time, platelets, thromboplastin generation, prothrombin consumption, prothrombin time and concentration of prothrombin, proconvertin, accelerator globulin and fibrinogen were measured in ten patients after the slow induction of hypothermia and later during surgery and transfusion. In contrast to previous studies in the dog, uncomplicated hypothermia in man was not associated with a fall in concentration of clotting factors. Moderate disturbances in coagulation with surgery and transfusion during hypothermia were in most regards similar to those observed during surgery and transfusion at normal body temperature. The one significant difference is a decrease in prothrombin consumption.

B. M.

The action of atropin on Pancreatic secretion *by L.C.V. Janqueira, Hanna, A. Rothschild and I. Vugman (1958,)*; **Brit. J. Pharmacol., 13,** *71.*

The action of atropine, in preventing pancreatic secretion in response to a parasympathomimetic drug has been analysed. Atropine did not appear to effect the uptake of glycine by the pancreatic cells or the incorporation of radioactive amino acids into the total pancreatic tissue proteins or into the proteins of the zymogen granules. The rate of amylase re-synthesis in stimulated glands was not affected by atropine. It has been suggested that atropine blocks pancreatic secretion in rats by blocking the extrusion of zymogen granules.

B. M.

Effect of aldosterone monoacetate and desoxycorticosterone acetate on alkaline phosphatase activity in the liver of rats *by Amiya B. Kar and R. P. Dass (1958)* : **Nature (Lond.) 181,** *625.*

Aldosterone monoacetate and DOCA were administered (10 /mg./daily/ S.C./ for 16 days) to two groups of young male rats with suitable controls.

Aldosterone monoacetate produced statistically insignifiicant inhibition of alkaline phosphatase activity in the liver and the histochemical studies also did not reveal any change. With DOCA the enzyme activity was significantly reduced and the histochemical studies corroborated this.

B. M.

Use of hydrocortisone in experimental viper venom poisoning in mice *by R. D. Ganatra, G. A. Dhopeshwarkar, U.K. Sheth and R.A, Lewis (1957)* : **Ind. J. Med. Sci. 11,** *493.*

The LD_{50} of viper venom was determined on mice. Doses of $2LD_{50}$ killed all untreated mice. When antivenom (Haffkine Institute) calculated to neutralize one LD_{50} was administered with $2LD_{50}$ of venom, the mortality was 57%. When hydrocortisone (100 mg./kg.) was given along with the above doses of venom and anti-venom, mortality was 17%. In a group of 30 mice given doses of $2LD_{50}$ of venom and treated with hydrocortisone alone (100 mg./kg.) the mortality was also 17%. Effects of hydrocortisone could not be demonstrated when the dose was reduced to 50 mg./kg.

B. M.

Distribution of Growth hormone among cell fractions isolated from pituitary gland by *E. Reid and Albert Segaloff. (1958)*: **Proc. Soc. Exper. Biol. & Med. 97,** *181.*

Fractions isolated from rat pituitary homogenates by differential centrifugation have been assayed for growth promoting activity by the tibia test. The supernatant fraction contained a relatively high proportion of the activity but no sharp localization was evident. B. M.

Diuretic action of Sodium Nimbidinate *by N. K. Bhide, D. J. Mehta and R. A. Lewis (1958).* **Ind. Jour. Med. Sci. 12,** *141.*

The diuretic properties of sodium nimbidinate obtained from the bitter principles of the oil of neem (Melia azadirachta) seeds have been studied. It has been shown that 20 mg. of sodium nimbidinate i. v. is far more potent

diuretic than 20 mg. of urea. As compared with mersalyl, the effects of both the drugs are of the same order of magnitude occurring with similar promptness with comparable doses. This drug acts primarily on the renal tubules and appears to be excreted in the urine. Given intramuscularly (in animals) the drug is ineffective and has irregular effects when given orally.

B.M.

Toxicity of Sodium Nimbidinate *by N. K. Bhide, D. J. Mehta, Mrs. W. W. Altekar and R. A. Lewis (1958)*: **Ind. Jour. Med. Sci. 12,** *146.*

Sodium nimbidinate has an encouraging margin of safety. Such high doses as a single oral dose of 1000 mg./kg. or repeated injections lead to proliferation of endothelial cells of the glomeruli and cloudy swelling, fatty infiltration and necrosis of the convoluted segment of the renal tubules.

B. M.

Clinical trials with parenteral Sodium Nimbidinate, a new diuretic. Preliminary report *by P.M. Shah, U. K. Sheith, N. K. Bhide and M. J. Shah (1958)*: **Ind. Jour. Med. Sci. 12,** *151.*

Nine cases of congestive cardiac failure with anasarca were studied with i.m. sodium nimbidinate as a diuretic out of which in eight cases a good response was obtained. In one case there was no response. Other than local discomfort and pain, no toxic effects were obtained.

B. M.

The action of Dopamine on the arterial blood pressure of guinea-pig *by O. Hornkiedicz (1958)*: **Brit. J. Pharmacol. 13,** *91.*

The depressor action of dopamine (β-3:4-dihydroxyphenylethylamine) upon the arterial blood pressure of the guineapig has been studied. This effect begins without a latent period. It is often enhanced after i. v. injection of iproniazid (marsilid). The depressor response is sufficiently sensitive to serve as a method of bio-assay of dopamine in microgram quantities. Observations on the depressor action of L-Dopa have also been made. This effect

is also enhanced by iproniazid; it begins after a latent period. Epinine (β–3:4–dihydroxyphenylethylmethylamine) caused a pressor response followed by a fall of arterial blood pressure. No evidence was obtained in support of the suggestion that the two amines which are oxidized at a similar rate by amine oxidase cause a fall of blood pressure after their conversion to an aldehyde by the action of amine oxidase.

B. M.

Changes in the adrenal of hypothyroid (Thiourea induced) rats and influence of testosterone propionate on such changes *by S. N. Roy, J. N. Karkun and R. N. Sur. (1958)*: **Acta Endocrinol, 27,** *216.*

Hypothyroidism was produced in young male albino rats by injection (S. B.) of aqueous solution of thiourea in the short term experiment for 38 days and in the long term experiment for 88 days. In both the experiments testosterone propionate in oil was also given. In both experiments thiourea lead to adrenocortical atrophy. In the short term experiment adrenal cholesterol rose above the normal level while ascorbic acid content showed a decline. In the prolonged experiment both these constitutents showed a fall. Testosterone stimulated the adrenal cortex of thiourea treated rats. The cholesterol content registered a fall while ascorbic acid concentration did not show any appreciable change. The significance of these changes and the mechanism of action of testosterone have been discussed.

B. M.

Hyperlipoprotinaemia and cholesterol deposition at the arteries of I^{131} treated dogs *by Lawrence J. Milet, A. A. Renzi, Norman Weiner, Lesly G. Robinson and Sherman S. Wilson (1958)*: **Proc. Soc. Exper. Biol. & Med. 97,** *56.*

Dogs maintained for a year after I^{131} administration were found to achieve maintenance levels of serum cholesterol in excess of 430 mg.% and comparably elevated concentrations of other serum lipid and lipo-protein parameters. Tissue analysis at autopsy revealed significantly elevated levels of coronary artery, aorta and liver cholesterol in male treated dogs as compared with the control females. Treated female dogs developed higher S_F. 0–12 blood lipo-proteins. No correlation between altered tissue cholesterol content and any of the serum lipid or lipo-protein moieties were observed.

B. M.

Some effects of pentobarbital anaesthesia on renal haemodynamics, water and electrolyte excretion in the dog *by William D. Blake (1957).* **Am. J. Physiol. 191,** *393.*

Studies were made on dogs to clarify the actions of pentobarbital anaesthesia on renal function. The depression of renal function frequently observed when anaesthesia is induced in animals hydrated with water or hypotonic saline solutions was confirmed. When the neurogenically and hormonally mediated components of the response to pentobarbital are respectively blocked by dihydrogenated ergot alkaloids and pitressin infusion, pentobarbital anaesthesia increases sodium excretion rather than decreasing it. Infusion of pentobarbital directly into the renal artery suggests that this natriuresis is caused by a toxic action of the drug on the reabsorptive process. There is also some inhibition of the glucose transport process.

B. M.

Antagonism of depressed extracellular potassium levels against histamine toxicity in mice *by William H. MacMillan (1957):* **Am. J. Physiol. 19,** *583.*

Three groups of white mice were fed low, normal and high potassium diets respectively for 5 weeks. The plasma K levels of the three groups were determined on the 34th day. The LD_{50} s and 19/20 confidence limits for respective groups to i.v. histamine were 240 mg./kg , 189 mg./kg., and 155 mg./kg. respectively. This indicates that a reduction in total body K offers a protective action against the production of histamine death in mice. The results obtained cannot be explained on the basis of delayed circulation time, renal or adrenal pathology ; however, they suggest a possible mechanism to explain the protective action of fasting against histamine toxicity in this species.

B. M.
