

amines. (Campbell, G. S., and others: *Depressed Response to Intravenous Sympathomimetic Agents in Humans During Acidosis*, *Dis. Chest* 33: 18 (Jan.) 1958.)

ADRENAL INSUFFICIENCY Adrenalectomized dogs had a lower initial mean systolic and diastolic blood pressure in both femoral and pulmonary arteries than did control dogs. There was no significant difference between the adrenalectomized dogs and the control animals in ability to respond intravenously injected norepinephrine by an increase in force of cardiac contraction or by an increase in systemic or pulmonary blood pressure. Myocardial or coronary artery lesions did not occur in any of the animals. (Reidenberg, M. M., Ohler, E. A., and Sevy, R. W.: *Cardiovascular Responses to Norepinephrine in Acute Adrenal Insufficiency*, *Proc. Soc. Exper. Biol. & Med.* 97: 889 (April) 1958.)

CORTISONE Hydrocortisone or corticosterone given for 60 days to normal rats caused an elevation of the total circulating red cell volume from 1.45 to 1.64 times that of normal, untreated controls. There was a significant elevation in erythrocyte, hematocrit and hemoglobin values. (Fisher, J. W.: *Increase in Circulating Red Cell Volume of Normal Rats After Treatment with Hydrocortisone or Corticosterone*, *Proc. Soc. Exper. Biol. & Med.* 97: 502 (March) 1958.)

DIBENAMINE IN SHOCK Development of irreversibility to transfusion in hemorrhagic shock has been shown to be caused by bacterial endotoxins and dibenamine prevents the development of irreversibility. On the basis of experiments on rabbits, it is concluded that dibenamine blocks the action of the toxin, and thereby not only preserves the responsiveness of the circulation, but also prevents the hemorrhagic lesion in the bowel wall, which is characteristic of irreversible shock. (Smiddy, F. G., Segel, D., and Fine, J.: *Proc. Soc. Exper. Biol. & Med.* 97: 584 (March) 1958.)

ADRENALINE AND NORADRENALINE A photo fluorimetric method for

the determination of adrenaline and noradrenaline in peripheral plasma is described. The sensitivity of this technique is 0.2 $\mu\text{g./l.}$ of adrenaline and 0.3 $\mu\text{g./l.}$ of noradrenaline. By use of a differential oxidizing procedure the concentrations of both adrenaline and noradrenaline in a mixture can be determined. Values for these substances in peripheral blood were found to be very much lower when precautions were taken to prevent the disruption of platelets. This observation is in harmony with the view that the platelets may act as vehicles for the transport of adrenaline and noradrenaline. (Robinson, R., and Scott, F. D.: *Fluorimetric Determination of Adrenaline and Noradrenaline in Plasma*, *Biochem. J.* 68: (March) 1958.)

SEROTONIN Serotonin probably participates in a wide variety of metabolic and physiologic functions. The cardiovascular response to serotonin is "amphiphase" and seems to vary with the dose and test animal. Vasodilatation generally follows from slow infusion of small doses and vasoconstriction occurs when larger doses are given quickly. Single intravenous injections given to normotensive and hypertensive patients while causing an increased pulse rate and cardiac output, had a variable effect (pressor, depressor, or biphasic) on the arterial pressure. The pressor effect may be augmented by cocaine, reserpine and pituitrin. The depressor response may be due to release of endogenous histamine by serotonin and/or peripheral inhibition of neurogenic vasoconstriction. The coronary vessels were dilated. Respiration was consistently increased, possibly by stimulation of the chemoreceptors. Pulmonary vasoconstriction always occurred as manifested by a rise in pulmonary artery pressure and a fall in left atrial flow without a concurrent change in aortic pressure and heart rate. Serotonin was the only substance studied that affected the pulmonary vasculature in doses insufficient to affect systemic and arterial pressure. Serotonin is probably formed in enterochromaffin tissue and is found in particularly large quantities in the brain, platelets and gastrointestinal tract. Reserpine seems to cause its release from these sites, while Iproniazid, by interfering with