amines. (*Campbell, G. S., and others: Depressed Response to Intravenous Sympathicomimetic 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 µg./l. of adrenaline and 0.3 µg./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.: Fluometric 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 "amphibaric" 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

its degradation, causes accumulation of serotonin. This is associated with agitation, psychosis and other forms of abnormal behavior. Chlorpromazine and Phenergan may produce their tranquilizing effects by specifically antagonizing serotonin. (*Page, I. H.: Serotonin (5-Hydroxytryptamine); Last Four Years, Physiol. Rev. 38: 277 (April) 1958.*)

PITRESSIN SUBSTITUTE Realizing the serious coronary constriction that Pitressin may produce but still needing a hemostatic agent to facilitate uterine operations such as myomectomies, the authors investigated vasopressin, isolated from hog pituitaries. When they injected a solution of 0.2 unit per milliliter into the operative site, they obtained blanching of the uterus for 15 to 20 minutes. Blood loss was remarkably reduced and there were no cardiac, circulatory or other significant complications. (*Dillon, T. F., and others: Vasopressin as Hemostatic in Gynecologic Surgery, Obst. & Gynec. 11: 363 (April) 1958.*)

PLACEBOS An attempt has been made to describe lesser known aspects of the "pharmacology" of placebos by describing the ways in which the clinical use of inert substances may lead to effects which are usually considered to be the exclusive property of active agents. One of the basic indices of pharmacologic activity is the time-effect relationship. Placebos can also show this behavior. A comparison is made between aspirin and a placebo. Although the mean score for the placebo relief was somewhat lower the difference was not statistically significant. Placebos may also show a "build-up" in effect, and there may be a "carry-over" after cessation of therapy. Another general characteristic of drugs is the inverse relationship of their efficacy to the severity of a given complaint. This relationship for placebos has also been apparent. This finding is somewhat at variance with the report of Beecher who found that patients studied early in the postoperative period are handled almost as well by placebo as by morphine, whereas later in the postoperative course morphine performs much better than the placebo. (*Lasagna, L., Laties, V. G., and Dohan, J. L.: Further Studies on "Pharmacology" of Placebo Administration, J. Clin. Invest. 37: 533 (April) 1958.*)

PREANESTHETIC MEDICATION Data obtained in a blind study of morphine, meperidine, alphaprodine, secobarbital and saline solution as preanesthetic medicaments in 1,400 surgical patients showed that secobarbital led to a higher proportion of calm, carefree, yet alert patients than did the narcotics. Undesirable side effects were seen more often after preanesthetic narcotics than with secobarbital. There was little difference in the influence of the various drugs upon satisfactory induction of anesthesia with any given anesthetic agent. Preanesthetic drugs did not appear to influence maintenance of anesthesia, except that respiratory depression was more common if a preanesthetic narcotic had been given. Patients who received preoperative narcotics remained narcotized longer after anesthesia than those who received secobarbital or saline, but they did not complain of pain as often nor appear as restless as the latter group. Of the drugs studied, dosages considered to be equipotent were: morphine, 5 mg.; meperidine, 50 mg.; alphaprodine (Nisentil), 30 mg.; secobarbital (Seconal), 75 mg. (*Eckenhoff, J. E., and Helrich, M.: Study of Narcotics and Sedatives for Use in Preanesthetic Medication, J. A. M. A. 167: 415 (May 24) 1958.*)

NALORPHINE The outstanding differences in the human pharmacology of nalorphine as compared with morphine are: (1) relative low potency of single doses of nalorphine in inducing sedation; (2) lesser degree of pupillary constriction, depression of temperature, depression of respiratory rate, and minute volume after single doses of normorphine; (3) marked accumulation of sedative effects of nalorphine during repeated administration, and (4) relatively slow onset and mildness of the abstinence syndrome after withdrawal of nalorphine. (*Fraser, H. F., and others: Human Pharmacology and Addiction Liability of Normorphine, J. Pharmacol. & Exper. Therap. 122: 359 (March) 1958.*)

LEVALLORPHAN The addition of levallorphan to meperidine, in a 1:100