shock in three. Etiology of this shock syndrome was believed to be similar to that of experimental shock produced with adrenaline and noradrenaline. (*Spoerel, W., and others: Shock Caused by Continuous Infusion of Metaraminol Bitartrate (Aramine), Canad. Med. Ass. J. 90: 349 (Feb. 1) 1964.*)

LEVARTERENOL NECROSIS Evidence suggests that there may be a relation between the lesions produced by endotoxin and alterations in circulating or local levels of levarterenol, or epinephrine, or both. Studies have shown that an intravenous injection of endotoxin, followed by an intradermal injection of epinephrine or levarterenol within the ensuing four hours, produces extensive hemorrhagic necrosis of the skin at the injection site. Endotoxin in some manner alters the response of blood vessels to epinephrine and levarterenol, enabling these hormones to produce necrotizing vascular lesions. Animals in whom an epinephrine tolerance has been developed demonstrate no inhibitory effect on the local necrotizing phenomenon, but generalized reactions, as evidenced by renal cortical necrosis, are inhibited by the previously developed epinephrine tolerance. (*Hall, D., and others: Effects of Epinephrine Tolerance on the Schwartzman Phenomenon, Amer. J. Path. 44: 431 (Mar.) 1964.*)

ALPHA AND BETA RECEPTORS Many substances are known which can block alpha sympathetic receptors, but until recently only one beta receptor blocker had been synthesized (dichloroisoproterenol). A new beta blocker (2-isopropylamino-1-2-naphthylethanol HCl), ("Nethalide") is now available and useful in the investigation of the significance and extent of sympathetic control of cardiac function. Hemodynamic changes following injection of norepinephrine and epinephrine in intact and lightly anesthetized dogs before and after beta adrenergic blockade were studied. Response to injected catechol amines is determined by a complex inter-relationship of various factors controlling circulatory hemeostasis. The ventricle, under the influence of beta receptor blockade, responded to additional pressure load with decreased performance characteristics. (*Kako, K., and others: Cardiovascular Effects of Catechol Amines in Dogs Before and After Beta-Adrenergic Blockade, Naunyn-Schmiedeberg Arch. Exp. Pathol. 246: 297, 1964.*)

CONTROLLED HYPOTENSION Controlled hypotension is accomplished by ganglionic blockade associated with controlled posture and controlled respiration. Ganglion block is usually achieved by intravenous drugs and the hypotension which follows is dependent on posture and respiration, both of which are controlled by the anesthetist. Posture achieves hypotension by pooling blood in vessels of the lower extremities. Veins, which are the capacity vessels of the circulation, must be dilated and capable of accommodating the blood in order to achieve a satisfactory hypotension. Tipping also achieves a gradient of blood pressure throughout the body and the production of "postural ischemia." The cerebral blood flow in the tipped patient may fall to a critical level, and must be watched carefully for clinical signs of cerebral ischemia. Controlled respiration, by raising the intrapulmonary pressure, controls the venous return to the heart and the output of the right heart into the pulmonary circulation. The P_{CO_2} must remain near normal for satisfactory control of blood pressure. Halothane has proved a most valuable addition to the hypotensive technique by increasing the sensitivity to ganglion blocking drugs as well as by its own inherent hypotensive action. The oscillometer is a valuable instrument for blood pressure monitoring. Personal experience is essential to obtain satisfactory and reliable results. (*Enderby, G. E. H.: Controlled Hypotension in Anaesthesia and Surgery, Der Anaesthesist 13: 22 (Jan.) 1964.*)

HEMORRHAGE In a study of seasonal resistance to hemorrhage, over 100 dogs were bled a percentage of their blood volume in the production of a standard shock experiment. A seasonal variation in resistance was confirmed. For example, loss of 39 per cent of the blood volume caused 12 per cent mortality in February and November, but 100 per cent mortality in July. Blood loss was tolerated better in the fall and winter than in the spring and summer. (*Swan, H., and others:*

Effects of Season on Death Due to Hemorrhage, Arch. Surg. 88: 448 (Mar.) 1964.)

HYPERTENSION Polypeptide vasoconstrictor activity was assayed in the peripheral venous blood of 45 hypertensive patients and 20 normal subjects. Average activity was significantly increased in all hypertensive groups, and activity tended to be much higher in patients with renovascular and malignant hypertension than in primary hypertension. There was no correlation between vasoconstrictor activity and renal excretory function. Plasma level of vasoconstrictor activity did not correlate with mean arterial pressure except when malignant or renovascular hypertension was present. A significant inverse relationship was found between the serum potassium concentration and vasoconstrictor activity: as activity levels increased, the potassium fell. Possibly a renin-like substance may be present in various hypertensive states in amounts sufficient to depress activation of the renin-angiotensin-aldosterone mechanism. *(Fitz, A. E., and Armstrong, M. L.: Plasma Vasoconstrictor Activity in Patients with Renal, Malignant, and Primary Hypertension, Circulation 29: 409 (Mar.) 1964.)*

AORTIC CLAMPING In nine cases blood pressure rose by 14 to 64 per cent of the preoperative level when the aorta was clamped. To counteract this rise, 1.03 mg. to 14.09 mg./minute (in a total dose of 32 mg. to 310 mg.) of Arfonad was infused for 21 minutes to 2 hours. These doses kept the blood pressure at +34 to −14 per cent of the preoperative level. It took an average of 30 minutes for the blood pressure to stabilize after removal of the aortic clamps. Conclusions were (1) the maximum total dose of Arfonad should be 300 mg.; (2) infusion should be at 1.5 mg./minute; (3) blood pressure during Arfonad administration should be kept at +25 per cent of the preoperative level, if it rises 50 per cent when the aorta is clamped; (4) the infusion should be started five to 10 minutes after clamping of the aorta, and should be stopped five to 10 minutes before removal of clamps. *(Miyazaki, M.: Hypertension and Anaesthesia, Especially in Cardiovascular Surgery with Circulatory Occlusion, (Japanese), Jap. J. Anaesth. 12: 74, 1963.)*

VALSALVA TEST In normal subjects, a pressure overshoot occurs in both pulmonary artery and peripheral artery together with bradycardia after cessation of straining during the Valsalva maneuver. In patients with right ventricular impairment the pressure overshoot in the pulmonary artery and bradycardia do not occur. If peripheral pressure overshoots and bradycardia does not occur with ordinary clinical measurements, right ventricular impairment can be suspected, but if both peripheral pressure and heart rate changes are abnormal no localization of cardiac disease can be made. *(Malmberg, R., and others: Valsalva Maneuver as a Test of Cardiac Function in Patients with Pulmonary Disease, Amer. Rev. Resp. Dis. 89: 64 (Jan.) 1964.)*

CORONARY VASODILATORS By coronary arteriography, glyceryl trinitrate and erythrityl tetranitrate have coronary vasodilating action even in those patients with coronary atherosclerosis. This does not imply that coronary blood flow is consequently improved. *(Likoff, W., Kasparian, H., Lehman, J. S., and Segal, B. L.: Evaluation of Coronary Vasodilators by Coronary Arteriography, Amer. J. Cardiol. 13: 7 (Feb.) 1964.)*

HEMATOCRIT AND CORONARY DISEASE A study comparing hematocrit values of three groups of normal men with those of 200 patients with myocardial infarction showed that hematocrit value does not seem to be a significant factor in predisposition to acute myocardial infarction, except in conditions in which its value is much increased. *(Conley, C. L., and others: Hematocrit Values in Coronary Artery Disease, Arch. Intern. Med. 113: 170 (Feb.) 1964.)*