

ACIDOSIS Isoproterenol was infused before and following the induction of lactic acidosis and compared with the naturally-occurring norepinephrine. Isoproterenol caused a slight decrease in arterial pressure, lowering of central venous pressure, and an 87 per cent increase in cardiac output 30 minutes following the start of the infusion during acidosis. There was also a decrease in total peripheral resistance and an increase in heart rate. By contrast, norepinephrine caused an increase in arterial pressure, an increase in central venous pressure, but a moderate decrease in cardiac output during acidosis. Total peripheral resistance was increased and heart rate was slowed. (Silberschmid, M., and others: *Isoproterenol and Cardiac Response to Experimental Lactic Acidosis, Surgery* 63: 181 (Jan.) 1968.)

BLOOD GAS CALCULATION A computer program to calculate oxygen saturation, oxygen content, partial pressure of carbon dioxide, base excess, buffer base, standard bicarbonate, and related acid-base variables from data provided by the Astrup method or by Severinghaus electrodes has been developed. No preliminary calculations or corrections are needed. The method is rapid and efficient, and is applicable to adult or fetal blood. (Jalouwayski, A., and others: *A Computer Method for Determination of Acid-Base and Oxygenation Variables in Adult and Infant Blood Samples, J. Lab. Clin. Med.* 71: 328 (Feb.) 1968.)

INCREASED INTRACRANIAL PRESSURE The cardiovascular patterns in eight patients with intracranial injuries were studied, and the data compared with data from 13 healthy subjects. Considerable variability in hemodynamic patterns was observed in the period immediately after injury, suggesting that several factors may be involved in circulatory reactions to head injury. Tachycardia, reduced stroke index, and reduced stroke work were found in the series. Three patients had increased cardiac output and low peripheral resistance. The relation of increased intracranial pressure to hemodynamic events was evaluated in three patients by simultaneous pressure and flow measurements. Increased

intracranial pressure was positively related to changes in cardiac output. Although there were exceptions, the increased pressure was not positively related to arterial pressure or peripheral resistance. (Brown, R. S., and others: *Changes after Cranial Cerebral Injury and Increased Intracranial Pressure, Surg. Gynecol. Obstet.* 125: 1205 (Dec.) 1967.)

SYMPATHETIC ACTIVITY The direct effects of propranolol and its interaction with isoproterenol on heart rate, myocardial contractile force, arterial pressure, aortic flow and atrial pressures were studied in anesthetized dogs in which autonomic tone had been eliminated by sympathetic (epidural) and parasympathetic (atropine) block. Propranolol in doses from 0.01 to 1 mg./kg. caused no significant changes in any of these cardiovascular measurements. Three mg./kg. of propranolol did cause small decreases in heart rate, contractile force and blood pressure, and increased left atrial pressure. The beta-adrenergic effects of 2 µg./kg. of isoproterenol were blocked progressively by increasing amounts of propranolol, and were completely eliminated by the 3-mg./kg. dose. After 3 mg./kg. of propranolol, higher doses of isoproterenol surmounted any effects of the beta-receptor block on heart rate, contractile force and atrial pressure. In addition, high doses of isoproterenol after beta-receptor blockade caused marked rises in mean arterial pressure and in calculated peripheral resistance. These peripheral effects of isoproterenol were not seen in dogs which had received 10 mg./kg. of phenoxybenzamine in addition to the propranolol. (Flacke, J. W., Osgood, P. F., and Bendixen, H. H.: *Propranolol and Isoproterenol in Dogs Deprived of Sympathetic Nerve Activity, J. Pharmacol. Exper. Therap.* 158: 519 (Dec.) 1967.)

BETA-ADRENERGIC BLOCKADE The circulatory effects of 5 to 10 mg. of propranolol administered intravenously were determined in five healthy subjects and in eight patients with verified coronary heart disease, subjected to exercise-induced angina pectoris. The relief of pain during work after beta-blockade could be explained by a 20 per cent decrease in left