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. (Siberschmid, 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. (Jelovayski, 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: 1305 (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
ventricular work. This decrease was related to fall in heart rate, since stroke volume and systemic arterial pressure were unaltered. No changes in acid–base balance or the respiratory quotient which would indicate metabolic disturbances during work of short duration were found. (Aström, H.: Haemodynamic Effects of Beta-adrenergic Blockade, Brit. Heart J. 30: 44 (Jan.) 1968.)

CATECHOLAMINES In dogs subjected to standardized shock, the pressor responses to injected norepinephrine, epinephrine and tyramine were markedly reduced immediately after reinfusion of blood. In most experiments, the responses to norepinephrine and to epinephrine gradually returned toward pre-hemorrhage levels over a period of several hours, whereas the responses to tyramine declined further. The content of catecholamines fell markedly in heart, spleen and adrenal six hours after reinfusion, whereas that in the aorta did not. Changes in the ratio of norepinephrine to epinephrine indicated tissue uptake of epinephrine from the adrenal discharge. (Calcoet, D. N., and Lum, B. K. B.: The Effects of Hemorrhage Shock upon Blood Pressure Responses to Adrenergic Agents and upon Tissue Catecholamines, J. Pharmacol. Exper. Therap. 159: 74 (Jan.) 1968.)

RESUSCITATION The entire house staff and registered nurse staff participated in a resuscitation training program. A special cart with supplies to care for any emergency was prepared. The cart is stored in the Emergency Room. The resuscitation team consists of the Emergency Room intern and nurse who bring the cart, the surgical, medical, and anesthesiology residents who are on call. The resuscitation team arrived on an average of three minutes after the call was made. The team was called about twice weekly, and 89 per cent of the calls were considered to be necessary. Initial success was achieved in one-third of the attempts at resuscitation. Long-term success was achieved in 12 per cent of the resuscitation attempts. (Benfield, J. R., and Hickey, R. C.: Cardiopulmonary Resuscitation at University of Wisconsin, Arch. Surg. 96: 664 (March) 1968.)

PLASMA VOLUME IN HYPERTENSION Plasma volume values in 37 male patients with uncomplicated essential hypertension were compared with similar measurements in 20 normal men. Plasma volume was lower in hypertensive individuals than in normal subjects. This relationship was true whether body weight, surface area or height was used as the reference index. These results contrast with reports of expanded plasma volume in primary aldosteronism and renovascular hypertension, and stress the importance of diagnostic grouping in evaluating the hypertensive state. (Tata, R. C., Frohlich, E. D., and Dusan, H. P.: Plasma Volume in Men with Essential Hypertension, New Engl. J. Med. 278: 762 (March) 1968.)

Respiration

COMPLIANCE Lung, thoracic, and total respiratory compliances were measured and found to be normal in patients with obstructive lung disease in whom respiratory muscle activity had been eliminated by use of muscle relaxants. In most cases, more air could be put into the lungs by a ventilator than the patient could inspire when conscious. The pressure required to inflate these lungs passively was not excessive. Maximal negative respiratory pressures that could be developed by these patients were far lower than normal. The ease with which adequate ventilation could be carried out in these emphysematous patients suggests that irreversible airway obstruction is not the sole cause of respiratory failure. Failure of the inspiratory pump may be an important factor. (Sharp, J. T., and others: The Thorax in Chronic Obstructive Lung Disease, Amer. J. Med. 44: 39 (Jan.) 1968.)

MARFAN'S SYNDROME In Marfan's syndrome there is a defect in one or more connective tissue elements. In the lungs such defects might lead to increased lung compliance and residual volume and increased airway resistance during expiration. In five patients with Marfan's syndrome no such abnormalities were found. (Chisholm, J. C., and others: Results of Pulmonary Function Testing in Five Persons with the Marfan's Syndrome, J. Lab. Clin. Med. 71: 25 (Jan.) 1968.)