volume. Clinical observations in 390 cases of acute myocardial infarction revealed that independent atrial activity leads sometimes to serious hemodynamic consequences due to a fall in blood pressure. Beat-to-beat variations in blood pressure caused by asynchronous atrioventricular contractions sometimes produce a cyclic inequality of the peripheral pulse, which can be detected by palpation, sphygmomanometry or digital plethysmography. (*Valero, A.: Atrial Transport Dysfunction in Acute Myocardial Infarction, Amer. J. Cardiol. 16: 22 (July) 1965.*)

EXPERIMENTAL HEART BLOCK Effects of acute and chronic complete heart block on the mechanical function of the heart were studied in dogs. Surgically-produced heart block resulted in profound bradycardia. There was an immediate decrease in cardiac output, increase in stroke volume, decrease in aortic (and pulmonary artery) diastolic pressure, but no change in aortic systolic pressure. End-diastolic volume increased due to increased filling time. The heart adjusted to the increased diastolic load acutely by dilatation and chronically by hypertrophy. Heart failure ensued when the heart block persisted beyond four months. Each atrial contraction was associated with a forward and backward movement of blood (atrium to ventricle and back to atrium) and closing and opening of the A-V valve. Atrial contraction made no overall contribution to ventricular volume except immediately preceding ventricular tension and fiber length (optimal PQ interval 0.085 to 0.125 sec.). *Brockman, S. K.: Cardiodynamics of Complete Heart Block, Amer. J. Cardiol. 16: 72 (July) 1965.*)

EXPERIMENTAL HEART BLOCK Direct and reflex activation of the cardiac sympathetics and parasympathetics in dogs with complete heart block shows that (1) the ventricle is almost solely under the control of the efferent sympathetic nerves, (2) the vagus nerve has a negligible effect on the ventricle, and (3) severe cardiac depression and ventricular arrest may be mediated through reflex diminution of cardiac sympathetic tone but not through the vagus nerve. Vagal fibers may supply the bundle of His and its branches but the vagus had no direct effect upon the ventricles. The positive inotropic and chronotropic effects of stimulation of the cardiac sympathetics were striking. Activation of the carotid sinus (carotid hypertension) reflexly induced a ventricular negative inotropic and chronotropic effect. The vagus played no role in this effect but it was mediated by reflex inhibition of the cardiac sympathetics. An increase in pressure within the carotid sinus reflexly induced a decrease in ventricular contractility. (*Brockman, S. K.: Reflex Control of the Heart in Complete A-V Block, Amer. J. Cardiol. 16: 84 (July) 1965.*)

LEFT BUNDLE BRANCH BLOCK In a group of 146 patients studied over the past decade who exhibited electrocardiographic findings of a complete left bundle branch block, the life expectancy after diagnosis averaged 36 months. The finding duplicated that of a previous study 15 years ago (*Johnson, R. P., and others: Amer. Heart J. 41: 225, 1951*) and emphasized the fact that invariably there is serious underlying organic disease associated with such an abnormality and that recent advances in the management of heart disease have not influenced the prognosis in this condition. (*Smith, S., and Hayes, W. L.: Prognosis of Complete Left Bundle Branch Block, Amer. Heart J. 70: 157 (Aug.) 1965.*)

PACEMAKER COMPLICATIONS Two problems encountered during implantation of electronic pacemakers for the treatment of heart block are of interest to the anesthetist. One is the hazard of aspiration pneumonitis following seizures preoperatively which may indicate tracheostomy at time of implantation; the other is the block of myocardial response to the electrical impulses by the repeated administration of large doses of succinylcholine. (*Lawrence, G. H., Paine, R. M., and Hughes, M. L.: Management of Complications Associated with the Use of Implantable Electronic Cardiac Pacemakers for the Relief of Complete Heart Block, Amer. J. Surg. 110: 177 (Aug.) 1965.*)

CENTRAL VENOUS PRESSURE A method of cannulating the subclavian vein

between the clavicle and first rib utilizing a 6 inch 15 gauge Rochester needle is described. The plastic catheter is advanced into the innominate vein or superior vena cava over a Seldinger guide wire which is then removed and an infusion begun with a pressure monometer attached for monitoring central venous pressures (CVP). Although isolated absolute CVP measurements are of value only if they are exceptionally high or low, continuous monitoring of CVP for several days, if necessary, and correlation with associated fluid balance and therapy is useful in assessing and treating abnormal circulatory blood volume (ineffective venous return). Complications of the method have an incidence of 4–10 per cent and include pneumothorax, hemothorax, hydrothorax, brachial plexus injury, subclavian artery puncture, thrombophlebitis and breaking of the catheter with embolization. Increasing experience and proper technique minimize these. (*Longerbeam, J. K., and others: Central Venous Pressure Moniotring. A Useful Guide to Fluid Therapy During Shock and Other Forms of Cardiovascular Stress, Amer. J. Surg. 110: 220 (Aug.) 1965.*)

PULMONARY FLOW Pressures were measured in various segments of the pulmonary vascular bed in 20 closed-chest preparations in which airway hypoxia was acutely induced. Total pulmonary blood flow was simultaneously recorded by means of an electromagnetic flowmeter. In every experiment acute hypoxia resulted in increased pulmonary blood flow, pulmonary arterial pressure, and pulmonary arterial wedge pressure while left arterial pressure remained unchanged. A marked rise was noted in postcapillary pulmonary vascular resistance, while precapillary resistance fell. The net result was an insignificant change in total pulmonary vascular resistance. If the pulmonary artery wedge pressure accurately reflects pulmonary venous pressure, the constriction would appear to occur in the pulmonary veins. (*Berry, W. C., and others: Effects of Acute Hypoxia on Pressure, Flow, and Resistance in the Pulmonary Vascular Bed, Surgery 58: 404 (Aug.) 1965.*)

ACCELERATION Six dogs under morphine-pentobarbital anesthesia were exposed to forward accelerations of 2, 4 and 6 G for one minute and 6 G for three minutes while in the horizontal, 15 degree head-up, and 15 degree head-down positions breathing room air. Femoral artery oxygen saturation decreased at 6 G, averaging 11 per cent at the end of 60 seconds with return to normal 30 seconds after exposure. The average increase in pulmonary arterial-venous shunt when air was breathed during 1 G was 17 per cent. The oxygen saturation of mixed venous blood decreased during exposures to 2, 4 and 6 G. Changes in blood oxygen saturation were not systematically affected by the presumed differences in intrathoracic blood volume and position of the diaphragm associated with the 15 degree head-up or head-down positions. (*Banchero, N., and others: Blood Oxygen Changes Induced by Forward (+G) Acceleration, Aerospace Med. 36: 608 (July) 1965.*)

CITRATE TOXICITY Toxic effects of citrate relate only to the production of a reduced serum ionized calcium concentration which causes cardiac arrhythmia, reduced output, arrest and death. There is no evidence that coagulation difficulties result from citrate-induced hypocalcemia. If an animal is supported by cardiac massage or parallel pumping during the brief period of cardiac difficulty, ionized calcium is quickly mobilized and cardiac function restored. Metabolism of citrate appears unimportant as mobilization of calcium occurs much before the citrate is removed. These effects can be prevented by the use of ionized calcium. Open heart surgery with total cardiopulmonary bypass has been performed on 27 patients with acid-citrate blood modified with 2 mg. of heparin and 0.5 g. of calcium chloride per unit. No difficulties have been encountered on this regimen. (*Jennings, E. R., and others: Citrate Toxicity and the Use of Anticoagulant Acid-Citrate Dextrose Blood for Extracorporeal Circulation, Surg. Gynec. Obstet. 120: 999 (May) 1965.*)