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