

ment central nervous system injury. (*Greenberg, H. B.: Cardiac Arrest in 20 Infants and Children: Causes and Results of Resuscitation, Dis. Chest 47: 42 (Jan.) 1965.*)

**CARDIAC CONDUCTION** Intracoronary injection of potassium chloride was done in mongrel dogs whose vagal tone was accentuated with morphine. Perfusion of the atrioventricular conduction system with potassium consistently inhibited vagally induced atrioventricular block. Similar perfusion of the sinoatrial node had no effect on vagally-induced sinus bradycardia or sinus arrhythmia. Moderate hyperkalemia and acetylcholine are antagonistic with respect to A-V conduction. Failure of potassium to alter the vagal influence on the sinus pacemaker demonstrates that the interrelationship of potassium and the vagus is not uniform for all cardiac tissue and for all electrophysiologic events. (*Feigenbaum, H., Wunsch, C. M., and Fisch, C.: Interrelationship of Potassium and Vagal Action on the Sinoatrial Pacemaker and on Atrioventricular Conduction, J. Clin. Invest. 44: 399 (Mar.) 1965.*)

**CARDIAC METABOLISM** When the heart is incubated anaerobically without glucose, it subsequently exhibits an impaired capacity to utilize glucose when incubated aerobically or anaerobically at a later time. However, when glucose is present during anaerobic conditions, there is no impairment of metabolism when aerobic conditions recur. By increasing the glucose load, glucose utilization can be increased to a maximum, and there is a marked protective effect on the anoxic myocardium, as indicated by ventricular function and contractile force data. (*Austen, W. G., and others: Myocardial Function and Contractile Force Affected by Glucose Loading of the Heart During Anoxia, Surgery 57: 839 (June) 1965.*)

**POLYPEPTIDES—CARDIAC EFFECTS** Besides their effect on the peripheral circulation the polypeptides bradykinin, angiotensin and vasopressin are known to influence cardiac action as well. In order to exclude reflex changes which would affect hearts *in situ* the effects of these polypeptides was studied on

isolated perfused guinea pig hearts, isolated auricles and papillary muscles. Bradykinin produced an increase of coronary flow and had a positive inotropic effect on the isolated heart as well as on the isolated auricle without influencing the heart rate. There was no effect on the isolated papillary muscle. Angiotensin produced a decrease of coronary blood flow associated with a negative inotropic effect without a change in heart rate. In the isolated auricle and papillary muscle, angiotensin had a weak positive inotropic effect. Vasopressin acted qualitatively like angiotensin. Its coronary constricting effect, however, was much stronger. (*Heeg, E., and Meng, K.: Effects of Bradykinin, Angiotensin and Vasopressin on the Auricle, Papillary Muscle and Isolated Perfused Heart Preparations of the Guinea Pig (German), Naunyn-Schmiedeberg Arch. Exp. Path. 250: 35 (Feb.) 1965.*)

**ARRHYTHMIAS** In dogs anesthetized with halothane or methoxyflurane, cardiac arrhythmias were induced by small amounts of epinephrine given intravenously. A Rauwolfia-alkaloid Ajmaline was effective in preventing and/or terminating epinephrine-induced auricular flutter and ventricular tachycardia. Pre-treatment with Ajmaline prevented arrhythmias by epinephrine even with 1.5 times the previous challenging dose. No toxic side effects were observed. (*Petter, A., and Schlag, G.: Treatment of Epinephrine Induced Cardiac Arrhythmias during Anesthesia with Halogenated Inhalation Anesthetics (German), Der Anaesthetist 14: 68 (Mar.) 1965.*)

**ARRHYTHMIA** Sixteen patients with acute myocardial infarction were studied with continuous cardiac monitoring for one to three weeks. Arrhythmias occurred with a much higher frequency than previously reported. They may occur in asymptomatic patients and go undetected by conventional absorption. (*Kurland, G. S., and Pressman, D.: Incidence of Arrhythmias in Acute Myocardial Infarction Studied with a Constant Monitoring System, Circulation 31: 834 (June) 1965.*)

**FIBRILLATION** Ventricular fibrillation appeared to be related to intramyocardial temperature gradients during induced hypothermia.