tion, J. Thoracic Surg. 36: 88 (July) 1958.)

PULMONARY COMPLIANCE Elastic properties of the lung were measured in 5 patients during Cheyne-Stokes respirations. Compliance was normal in 3 patients and reduced in 2. Thus the periodic breathing is due to periodic fluctuations in the activity of the respiratory center in some patients and to peripheral factors in other patients. Aminophylline abolished periodic breathing in the 3 patients to whom it was given. Oxygen will abolish periodic breathing of central origin and carbon dioxide will usually do the same for that associated with cardiac disease. *(Lyons, H. A., and others: Pulmonary Compliance in Patients with Periodic Breathing, Circulation 17: 1056 (June) 1958.)*

EXTENDED ASYSTOLE Normothermic dog heart begins to show EKG evidence of myocardial injury after 39 to 44 minutes of asystole and 50 to 60 minutes of asystole ended fatally in cardiac by-pass experiments. Thirty-five minutes is a recommended safe limit. *(Milnes, R., and others: Extended Asystole, A. M. A. Arch. Surg. 77: 13 (July) 1958.)*

HYPOTENSION Reduction in cerebral cortical oxygen as measured by the oxygen electrode occurred more readily when dogs given chlorpromazine were subjected to blood-letting hypotension. Greater reduction in blood volume was necessary to produce hypotension in a control group and at the same arterial pressure cortical oxygen tension dropped less in the control group than in the group of dogs receiving sympatholytic drugs. Electroencephalographic abnormalities occurred at higher blood pressure levels when a blocking agent was used than in the controls. *(Bloor, B., and others: Study of Cortical Oxygen Tension During Induced Hypotension, A. M. A. Arch. Surg. 77: 65 (July) 1958.)*

SHOCK Rats raised in germ-free conditions and subjected to hemorrhagic shock under germ-free experimental conditions did not differ from a control group in their response. This evidence must be interpreted carefully according to the authors and does not necessarily eliminate the possibility that bacteremia is of importance as a cause of irreversibility of shock as postulated by Hine. *(Zweifach, B. W., and others: Irreversible Hemorrhagic Shock in Germ-free Rats, J. Exp. Med. 3: 437 (March) 1958.)*

EXTRACORPOREAL CIRCULATION Magnesium sulfate or potassium chloride solution perfused into the coronary arteries separately were not as effective in stopping an isolated cat heart as the combination. Persistence of asystole followed use of potassium alone. Magnesium sulfate caused increase in ectopic beats. Neostigmine was added to induce a slower rate with better coronary perfusion as the heart was recovering. *(Merritt, D., and others: Potassium, Magnesium and Neostigmine for Controlled Cardioplegia, A. M. A. Arch. Surg. 75: 365 (March) 1958.)*

HEMOLYSIS With the plastic-sheet bubble oxygenator, hemolysis is partly due to the jets of oxygen entering the blood at the bottom of the chamber. Other factors include the percentage volume of red cells, cohesion of cells, spheroidicity, changes in membrane strength and heating to 52–58 C. The reticulo-endothelial system probably removes hemoglobin from the blood stream as rapidly as the hemoglobin is released. Another potential hazard of hemolysis is the effect of the red cell stromata which may produce hypotension and shock. Disturbances in the blood clotting mechanisms have also been ascribed to hemolysis. *(Ferbers, E. W.: Studies of Hemolysis with a Plastic-Sheet Bubble Oxygenator, J. Thoracic Surg. 36: 23 (July) 1958.)*

ULTRASONIC DEFIBRILLATION When ventricular fibrillation was induced electrically in the hypothermic rabbit ultrasonic irradiation restored 20 per cent of the rabbit hearts to normal rhythm. This technique failed entirely when dog hypothermic heart was used. *(Haeger, K. H.: Ultrasonic Irradiation of Ventricular Fibrillation in Hypothermic Rabbit and Dog, Acta chir. scandinav. 114: 99 (Feb.) 1958.)*

VENTRICULAR FIBRILLATION Rabbit hearts were perfused through the

aorta with a solution containing a controlled concentration of potassium and electrical stimulation was applied to the ventricle at a high rate to start fibrillation. Lack of glucose in perfusing solution allowed fibrillation to persist but fibrillation could be stopped by the addition of glucose, insulin, mannose or pyruvate. Fibrillation at 37 degrees centigrade was abolished by lowering temperature to 32 degrees possibly due to the fact that oxygen deficiency was less at the lower temperature. Adrenaline promoted fibrillation, possibly by increasing oxygen demand. (*Goodford, P. J.: Metabolic Factors and Ventricular Fibrillation, Brit. J. Pharmacol. 13: 44 (June) 1958.*)

MYOCARDIAL CONTRACTILITY When the lungs of a denervated heart-lung preparation were ventilated with 15 or 30 per cent CO_2, a striking cardiac decompensation resulted. This was manifested by a precipitous reduction or cardiac output against constant outflow resistance, elevated superior vena caval pressure, severe cardiac dilatation, decline in outflow pressure, and elevated left atrial pressure. In contrast was the remarkable tolerance of the intact dog to severe hypercapnea. The nature of the compensating mechanism in the intact animal is not clear; however, the myocardial failure consequent to high CO_2 administration was reversed or prevented by epinephrine, norepinephrine, acetyl strophantidin, or hydrocortisone. Each of these substances, with the possible exception of a digitalis-like derivative, is present, in the intact animal. (*Calvert, H. M.: Some Current Views on Biochemistry and Physiology of Myocardial Contraction, Bull. New York Acad. Med. 34: 445 (July) 1958.*)

CORONARY CIRCULATION The effects of atropine cardioacceleration on the coronary flow, cardiac work rate and cardiac oxygen metabolism was studied in six patients. A 38 per cent increase in rate produced a 38 per cent increase in coronary flow and a 33 per cent increase in oxygen consumption. There was a direct relation between the increased oxygen consumption and the increase in number of seconds of systolic contraction time occurring with tachycardia. The increased coronary flow was mediated by a decrease in vascular resistance which compensated both for the increased flow and the decreased diastolic time. (*Gorlin, R.: Studies on Regulation of Coronary Circulation in Man, Am. J. Med. 25: 37 (July) 1958.*)

CARDIAC ARREST No signs of cerebral or cord damage are noted in moderately hypothermic dogs after ten minutes of vena caval occlusion. Recovery without evidence of neurologic impairment is slower after 15 minutes of vena caval occlusion. Electrical cardiac standstill can be accomplished in the moderately hypothermic dog by coronary perfusion with sodium citrate and resuscitation by coronary perfusion with calcium gluconate or calcium gluconate followed by oxygenated blood. Ventricular fibrillation may occur if complete electrical arrest has not occurred. In such cases, conversion follows continued perfusion with oxygenated blood, electric shock or massage and electric shock. The use of sodium lactate is effective in producing clinical and electrical arrest in the moderately hypothermic dog. (*Riberi, A., and Shumacker, H. B.: Elective Cardiac Arrest Under Moderate Hypothermia, Ann. Surg. 148: 21 (July) 1958.*)

CARDIAC ARREST Elective cardiac arrest in dogs could not be maintained with acetylcholine with or without cardiac hypothermia. Infusion of cold blood into the coronary arteries following potassium arrest gave the lowest incidence of ventricular fibrillation and the best acute recoveries. Coronary perfusion with cold blood alone, cold blood prior to potassium arrest, or potassium arrest followed by coronary perfusion with warm blood showed a high incidence of ventricular fibrillation and/or post-recovery arrests. (*Berne, R. M., and others: Myocardial Hypothermia in Elective Cardiac Arrest, J. App. Physiol. 12: 431 (May) 1958.*)

CEREBROSPINAL FLUID A cerebrospinal fluid sample was obtained by lumbar puncture from 19 patients who either were in congestive heart failure at the time or had been in failure within the prior 14 days. The CSF pressure was elevated in those patients in congestive failure. Although the total protein content was within normal limits in all cases,