HYPOTHERMIA Hypothermic animals survived cardiotomy and inflow occlusion for 16 to 20 minutes when the coronary system was perfused with small volumes of oxygenated blood. Acetylstrophanthidin before inflow occlusion prevented myocardial failure and diminished the frequency of ventricular fibrillation. (*Lombardo, T. A., Radigan, L. R., and Morrow, A. G.: Myocardial Failure in Experimental Hypothermia, Circulation Res. 5: 22 (Jan.) 1957.*)

HYPOTHERMIA As body temperature was progressively reduced to 27 C. in the dog, mean blood pressure decreased progressively to approximately 75 per cent of the control values. This was associated with a progressive reduction in glomerular filtration rate and renal blood flow without significant alteration in urine or sodium excretion. The reduction in rate of glomerular filtration and in renal blood flow was not improved when the blood pressure was raised to control values with an infusion of norepinephrine. However, when body temperature was again increased to control levels, the mean blood pressure returned to control levels although the glomerular filtration rate and renal blood flow returned to only 75 per cent of the control levels. There was essentially no difference in these responses between dogs and man. (*Moyer, John H., De Bakey, M. E., and Morris, George: Hypothermia: Effect on Renal Hemodynamics and on Excretion of Water and Electrolytes in Dog and Man, Ann. Surg. 145: 26 (Jan.) 1957.*)

REWARMING Dogs lightly anesthetized, curarized, and maintained on artificial respiration were observed to rewarm in a temperature controlled room at 24 C. without evidence of striated muscle activity, indicating that factors other than shivering are a significant part of the rewarming process. (*Werner, A. Y., Dawson, Donald, and Hartenberg, Esther: Spontaneous Rewarming of Hypothermic Curarized Dog, Science 124: 3232 (Dec. 7) 1956.*)

COAGULATION DURING HYPOTHERMIA A decrease in proaccelerin, proconvertin, prothrombin and fibrinogen was demonstrated. This may have been due to intravascular clotting which also predisposed to thromboembolic phenomena. Heparin was used to minimize intravascular clotting. The depletion of coagulation factors in this heparin treated group of dogs was minimized. Also, no postwarming hemorrhage was seen. (*Ellis, P. R., Kleinsasser, L. J., Speer, R. J.: Changes in Coagulation Occurring in Dogs During Hypothermia and Cardiac Surgery, Surgery 41: 198 (Feb.) 1957.*)

BLOOD COAGULATION This is an extensive review of the recent work on the problem of blood coagulation with emphasis on the biological activation of prothrombin and the nature of thromboplastin forming reactions. Of particular interest to the anesthetist is the section on the effects of clotting. The initial, but temporary, arrest of the blood flow is probably achieved by primary factors other than clot formation, and firm clotting is essential to maintain hemostasis when the primary factors have ceased to operate. These primary hemostatic factors probably include vascular contraction and platelet agglutination. (*MacFarlane, R. G.: Blood Coagulation with Particular Reference to Early Stages, Phys. Rev. 36: 479 (Oct.) 1956.*)

CARDIAC RESUSCITATION In dogs 1 to 3 cc. of 15 per cent potassium chloride was used to convert fibrillation to cardiac standstill. After transient standstill, five dogs spontaneously reverted to normal rhythm. In the other 42, 2 to 3 cc. of 10 per cent sodium lactate was injected into the left ventricle while massage was continued. Coordinated contractions were produced by injecting 2 to 3 cc. of 25 × concentrated Ringer's lactate into the left ventricle. The first beats were felt in about twelve seconds, while effective beats required as long as five minutes of assisting contractions. If contractions were sluggish, a slow, intravenous drip of calcium gluconate was used (10 cc. of 10 per cent in 200 cc. 5 per cent dextrose in water). (*Schimert, G., and Cowley, R. A.: Defibrillation, Cardiac Arrest and Resuscitation in Deep Hypothermia by Electrolyte Solutions, Surgery 21: 211 (Feb.) 1957.*)

CARDIAC ARREST A solution of magnesium sulfate 2.47 per cent, potassium

citrate 0.54 per cent, and small quantities of Prostigmin, was compared to a control solution of potassium citrate 1.6 per cent. Both solutions were effective in producing cardiac arrest in hypothermic dogs. The danger of serious arrhythmia during ventriculotomy in the hypothermic animal was almost abolished using controlled cardiac arrest. (*Glenn, W. G., and others: Method for Controlled Cardiac Arrest as Adjunct to Open Heart Surgery, J. Thoracic Surg. 32: 604 (Nov.) 1956.*)

VENTRICULAR FIBRILLATION Dogs were allowed to develop ventricular fibrillation during hypothermia and maintain the fibrillation for thirty minutes. One group of dogs had cyanotic heart disease induced prior to experiments. Fifteen dogs had pulmo-aortic fistula created and a third group were normal dogs. Ninety-two per cent of the controls and cyanotic group were resuscitated, but only 53 per cent of the chronic heart strain group. The implication is that ventricular fibrillation developing during hypothermia in the presence of a diseased heart should be strenuously avoided. (*Riley, P. A., Barila, T. G., and Hughes, C. W.: Ventricular Fibrillation During Hypothermia A. M. A. Arch. Surg. 73: 985 (Dec.) 1956.*)

CHEYNE-STOKES Increased transit time of blood from heart to brain produces periodic breathing in dogs, similar to Cheyne-Stokes respirations in humans. A circulatory delay system was inserted between the heart and brain of thirty dogs to prolong the transit time of blood from lungs to brain. Cheyne-Stokes breathing was produced in each animal and the duration of the cycle increased with increase in volume of the delay system. Variations in per cent oxygen saturation and carbon dioxide concentrations of the blood were directly related to the phases of the Cheyne-Stokes breathing. (*Guyton, A. C., and others: Basic Oscillating Mechanism of Cheyne-Stokes Breathing, Am. J. Physiol. 187: 395 (Nov.) 1956.*)

PULMONARY EDEMA Antifoam compound no. 5507 is more effective in treating epinephrine-induced pulmonary edema in rabbits than is 10 or 20 per cent ethyl alcohol or octyl alcohol. Compound no. 5507 consists of silicone 0.01 per cent, Superinone (a polyhydric alcohol) 0.75 per cent, glycerin 1 per cent and potassium bicarbonate 1 per cent. After negative toxicity studies on animals, the compound was used to treat successfully the pulmonary edema of eight patients. (*Balagot, R. C., Reyes, R. M., and Sadove, M. S.: Antifoam Agents in Pulmonary Edema, J. A. M. A. 163: 630 (Feb. 23) 1957.*)

BLOOD ACTH LEVEL Utilizing a cross circulation technique with rats, it was demonstrated that a severe stress will produce a high secretory rate of ACTH for one to two hours. This was followed by a decline in ACTH release despite the presence of adequate pituitary stores. ACTH almost disappeared from body fluids six hours after a severe stress. After a milder stress a moderate rate of ACTH secretion was maintained during the twelve hour period studied. The fall in ACTH is not due to the pituitary inhibiting effect of adrenal cortical steroids. (*Brodish, A., and Long, C. N.: Changes in Blood ACTH Under Various Experimental Conditions Studied by Means of Cross Circulation Technique, Endocrinology 59: 666 (Dec.) 1956.*)

CARBON DIOXIDE Utilizing the denervated nictitating membrane of the cat as the test body, it was demonstrated that carbon dioxide is a potent stimulus for increasing the circulating sympatho-catechol amines. The threshold for adrenal stimulation was about 15 per cent carbon dioxide in alveolar air. The author points out the fallacy of using the blood pressure response to assess vascular smooth muscle action when carbon dioxide is administered. (*Tenney, S. M.: Sympatho-Adrenal Stimulation by Carbon Dioxide and Inhibitory Effect of Carbonic Acid on Epinephrine Response, Am. J. Physiol. 187: 341 (Nov.) 1956.*)

APNEA Respiratory acidosis is the chief factor responsible for the bradycardia of apnea, based on response of eleven dogs to apnea when breathing 100 per cent oxygen and also room air. Apnea was produced by *d*-tubo curarine. Bradycardia was observed after ninety seconds of apnea. This