HYPOTHERMIA The temperatures of the right atrial blood, cerebral cortex, rectum, subcutaneous tissue, nasopharynx and lower one-third of the oesophagus were measured continuously in sheep during surface cooling. The blood was colder than the brain during cooling and warmer on rewarming. The lower one-third of the oesophagus, its coolest portion, did not differ in temperature from the cerebral cortex by more than 0.2 C. at any time. (*Hercus, V., Cohen, D., and Bowring, A. C.: Temperature Gradients During Hypothermia, Brit. M. J. 1: 1439 (June 6) 1959.*)

METHODS OF COOLING Hypothermia may be induced by extracorporeal cooling (either arteriovenous or venovenous) or surface cooling (immersion in water, use of cooling blankets, packing in ice or ice bags or irrigation of the pleural cavity with cold saline). Four problems arise in clinical hypothermia: (1) drift in body temperature; (2) myocardial irritability; (3) rewarming shock; (4) bleeding tendency. One of the best controls of drift in body temperature is experience with the method one uses. The more rapidly the patient cools, the further will be the drift, once active cooling is stopped. Infants have labile thermoregulating mechanisms and may cool rapidly. If a very heavy individual is cooling rapidly, considerable drift must be expected because of the large body mass. During active cooling esophageal temperature is 1 degree to 4 C. below rectal temperature. The temperature gradient is proportional to the rate of cooling. Reduction of this gradient when active cooling has ended signals that temperature drift is near an end. The desired temperature range is 28 C. to 31 C. After temperature is stabilized, esophageal temperature becomes higher than rectal temperature and remains so during rewarming. Cardiac irritability is increased as temperature falls below 30 C. A higher incidence of ventricular fibrillation which is more often irreversible occurs with the use of citrated rather than as with heparinized blood. The syndrome of rewarming shock is characterized by a progressive decline in blood pH to levels below 7.15. Ultimately an abrupt loss of consciousness occurs associated with respiratory inadequacy, and occasionally associated with a fall in blood pressure and tachycardia. Presumably, the metabolic acidosis is due to accumulation of metabolites caused by intense peripheral vasoconstriction. These are liberated upon vasodilatation during rewarming. Upon correction of the metabolic acidosis with sodium bicarbonate, the clinical recovery is usually dramatic. The use of largactil, phenergan, or ether prevents rewarming acidosis probably by maintaining peripheral circulation. The only consistent blood abnormality associated with generalized oozing is thrombocytopenia. Platelets are decreased an average of 43 per cent during hypothermia being sequestered principally in the portal system and return to normal on rewarming. Heparin, 15 to 20 mg., administered prior to cooling decreases the average platelet drop to 19 per cent. The patient is rewarmed in bed with a heat cradle, hot air and warm water bottles. Shivering is controlled with largactil. (*Bigelow, W. C.: Methods for Inducing Hypothermia and Rewarming, Ann. New York Acad Sc. 80: 522 (Sept. 14) 1959.*)

HYPOTHERMIA Trauma to dogs under ether anesthesia produces a marked increase in pituitary ACTH and adrenal cortical secretion. Induction of hypothermia greatly depresses the output of these hormones, while rewarming restores them. Cooling the adrenal gland either separately or together with the rest of the body causes a marked depression of adrenal sensitivity to exogenous ACTH. Adrenomedullary secretion is likewise reduced in dogs, but not in the one human tested at 30 C. The disappearance rate of exogenously administered hydrocortisone was slower in the hypothermic adrenalectomized animal than the normothermic dog. This may explain why in some studies peripheral corticosteroid levels did not fall with hypothermia. (*Hume, D. M., and Egdahl, R. H.: Effect of Hypothermia and of Cold Exposure on Adrenal Cortical and Medullary Secretion, Ann New York Acad. Sc. 80: 435 (Sept. 14) 1959.*)

RESPIRATION IN HYPOTHERMIA Hypothermia alters virtually every measurable phenomenon involved in respiratory gas trans-

fer. Oxygen and carbon dioxide are more soluble at lower temperatures. Therefore, at a given tension, more oxygen and carbon dioxide will be dissolved in the blood of an hypothermic animal. Both *p*K and *p*H are increased *in vitro*. The oxygen dissociation curve is shifted to the left making less oxygen available to the tissues. However, tissue need for oxygen is reduced and dissolved oxygen may thus supply a large portion of the metabolic needs, particularly if high inspired oxygen concentrations are provided. Spontaneous respiratory exchange is depressed, its magnitude dependent on depth of anesthesia, type and amount of premedication, and individual variation. Anatomic dead space is increased due to bronchodilatation, but distribution is not significantly altered and carbon dioxide is eliminated without difficulty. If *p*H or P_{CO_2} is held constant during artificial respiration, carbon dioxide is retained. The current favoring of hyperventilation is based largely on the incidence of ventricular fibrillation in animals cooled without assisted respiration. However, hyperventilation may deprive tissue of oxygen by: (1) increasing *p*H and thus shifting the oxygen dissociation curve to the left, and (2) reducing cardiac filling. It is more reasonable to propose that normal ventilation is that in which carbon dioxide elimination equals its rate of metabolic production as cooling progresses. In the absence of metabolic acidosis this may be achieved by maintaining alveolar carbon dioxide at the same tension as that of blood cooled *in vitro*. (*Severinghaus, J. W.: Respiration and Hypothermia, Ann. New York Acad. Sc. 80: 384 (Sept. 14) 1959.*)

HYPOTHERMIA Analysis of pressure pulse contours, calculations of cardiac work and efficiency, and measurements of myocardial contractile force in the hypothermic heart indicates that myocardial function is adequate for the work required of it at reduced temperatures. Coronary resistance is decreased in severe hypothermia either by a direct effect on the coronary vessels or possibly by retarding the rate of destruction of a vasodilator substance. Induced increases in heart rate affect myocardial function and coronary blood flow adversely. (*Berne, R. M.: Cardiodynamics and the Coronary Circulation in Hypothermia, Ann. New York Acad. Sc. 80: 365 (Sept. 14) 1959.*)

HYPOTHERMIA AND FIBRILLATION The efficacy of some antihistaminics, local anesthetics, and antimalarials in preventing spontaneous fibrillation during both progressive cooling and ventriculotomy at 26 C. indicate that activity in each test may depend on different pharmacologic effects. Quinidine among the antiarrhythmics, and antazolamine, chloromethapyrilene, methapyrilene and doxylamine among the antihistaminics were effective against spontaneous fibrillation. Many other antihistaminics and several local anesthetics and antimalarials had no effect. Procaine amide increased the incidence of fibrillation at relatively high temperatures. In experimental ventriculotomy with controlled *p*H, quinidine, antazolamine and chloromethapyrilene had definite antifibrillatory effect. Ouabain did not alter the incidence of either spontaneous or surgical hypothermic ventricular fibrillation. Thus digitalization does not appear to be a contraindication to hypothermic surgery. (*Angelakos, E. T.: Influence of Pharmacological Agents on Spontaneous and Surgically Induced Hypothermic Ventricular Fibrillation, Ann New York Acad. Sc. 80: 351 (Sept. 14) 1959.*)