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.*)