# *Literature Briefs*

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*Briefs were submitted by Drs. C. M. Ballinger, Peter P. Bosomworth, H. S. Davis, Deryck Duncalf, J. E. Eckenhoff, Martin Helrich, G. Hohman, J. J. Jacoby, F. C. McPartland, S. J. Martin, Marvin J. Noble, Alan Paterson, R. E. Ponath, Alan D. Randall, Wallace Ring, H. S. Rottenstein, Philip H. Sechzer, and B. L. Vandemeer. Briefs appearing elsewhere in this issue are a part of this column.*

**ATRIAL ACTIVITY** Atrial contribution to ventricular performance was evaluated during halothane anesthesia. The change from sinoatrial to atrioventricular nodal rhythm was associated with a fall in arterial pressure and a rise in central venous pressure. The heart rate remained unaltered. Changes in systemic and central venous pressures were attributed to failure of atrial systole to contribute to ventricular filling during diastole. Possibly tricuspid regurgitations may occur during nodal rhythm, since synchronous atrioventricular contraction is required for adequate closure of the atrioventricular valve. (*Haver, M. B., and Turndorf, H.: Atrial Activity and Systemic Blood Pressure During Anesthesia in Man, Circulation 28: 63 (July) 1963.*)

**DIRECT PERFUSION** Direct perfusion of oxygen saturated and unsaturated blood to the normal right or left ventricular myocardium, at a rate of 2 ml. per minute, could be tolerated for between 20 and 26 minutes without significant changes in rhythm and pressures. Further perfusion at the same rate led to an irregularity of the rhythm and gradual decrease of the systemic pressure until fibrillation finally occurred. The right ventricle could more easily tolerate the perfusion and the fibrillation was somehow more delayed. Perfusion of saturated blood at the same rate to the ischemic heart was tolerated for a significantly shorter time when compared with the former groups. Perfusion of dextran at a rate of 4 ml. per minute to the ischemic left ventricular myocardium did not alter the heart rate and could restore a normal left atrial pressure and an aortic pressure near normal. (*Petropoulos, P. C.: Effects on Cardiac Function During Direct Perfusion of the Normal or Ischemic Ventricular Myocardium with Saturated or Unsaturated Blood or Colloidal Isotonic Solutions, J. Thor. Cardiov. Surg. 46: 94 (July) 1963.*)

**HYPOCARBIA** Effects of acute hypocarbia during extracorporeal circulation in patients receiving 100 per cent oxygen and in patients receiving 3 per cent carbon dioxide plus 97 per cent oxygen include a rise in blood lactic acid and a fall in phosphorus and a fall in potassium. This compensatory metabolic acidosis contributes to an acute depression of pH in the postperfusion and postoperative period as blood carbon dioxide returns to normal levels. Those receiving 3 per cent carbon dioxide did not get metabolic acidosis and maintained a more nearly normal pH during the immediate postperfusion period. (*Andersen, M., and others: Relationship of Respiratory Alkalosis to Metabolic Acidosis During Extracorporeal Circulation, Surgery 53: 730 (June) 1963.*)

**CEREBRAL ISCHEMIA** Fifty-six patients with occlusive vascular disease tolerated carotid occlusion for periods up to 30 minutes on 66 occasions under general anesthesia without shunt, bypass, or hypothermia. EEG evidence of cerebral ischemia did not appear during occlusion, and no patient suffered any postoperative neurological sequelae. Twenty per cent of patients who had carotid arteries occluded for 30 to 60 seconds without general anesthesia suffered convulsions. Possibly general anesthesia increased the tolerance to cerebral ischemia by decreasing the cerebral metabolic rate for oxygen, increased the cerebral blood flow from hypercarbia, increased arterial oxygen tension, and recruited new routes of collateral circulation. (*Wells, B. A., and others: Increased Tolerance to Cerebral*