Literature Briefs

JOHN W. PENDER, M.D., Editor

Briefs were submitted by Drs. John Adriani, C. M. Ballinger, Peter P. Bosomworth, H. S. Davis, Deryck Duncalf, J. E. Eckenhoff, Martin Helrich, J. J. Jacoby, F. C. McPartland, S. J. Martin, Marvin J. Noble, A. S. Paterson, R. E. Ponath, Alan Randall, W. H. Ring, H. S. Rottenstein, and P. H. Sechzer. Briefs appearing elsewhere in this issue are a part of this column.

BUFFER Tris buffer was utilized either in the prevention or treatment of profound metabolic acidosis which developed during extended periods of cardiopulmonary bypass. In one group, buffer was added to the 24-hour-old, heparinized, priming blood, prior to initiation of perfusion, to counteract a low *p*H and high lactic acid concentration. All acid-base parameters were stable. In a second group, a severe acidosis developed despite preliminary buffering of the priming blood. In these cases the administration of additional tris buffer toward the end of perfusion successfully restored acid-base equilibrium. An evaluation of renal function following the administration of buffer during bypass disclosed an increased bicarbonate excretion and a rise in urinary *p*H. Osmotic diuresis did not develop although serum osmolarity was transiently elevated. (*Moore, D., and Bernhard, W.: Prevention and Treatment of Acute Metabolic Complications Associated With Prolonged Extracorporeal Circulation, J. Thor. Cardiov. Surg. 45: 565 (May) 1963.*)

ACIDOSIS Influence of respiratory and metabolic acidosis and hypoxia upon the isolated pressor and depressor elements of the epinephrine response was studied in dogs by pharmacologically dissociating the two components by the use of dichloroisoproternol (a beta blocker) and phentolamine (Regitine). Ventricular contractile force, arterial pressure and femoral blood flow were measured to determine the extent of cardiac and vasomotor participation in the responses to epinephrine during acidosis. Both the pressor and depressor actions of epinephrine were appreciably diminished in the presence of acidosis or hypoxemia. This effect was more pronounced with respiratory acidosis than with metabolic acidosis. It would appear that epinephrine requires adequate oxygenation of blood and tissues in order to exert its pressor effect. In shock, where both respiratory and metabolic acidosis is combined with hypoxia, there is a joint depressive action on the cardiovascular response to epinephrnie. (*Wood, W. B., Manley, Jr., E. S., and Woodbury, R. A.: Effects of CO_2 Induced Respiratory Acidosis on the Depressor and Pressor Components of the Dog's Blood Pressure Response to Epinephrine, J. Pharmacol. Exper. Ther. 139: 238 (Feb.) 1963.*)

CARBON DIOXIDE During extracorporeal circulation using the Mayo-Gibbon pump oxygenator combined with moderate hypothermia, the oxygenator was supplied with 3 per cent carbon dioxide. Arterial P_{CO_2} proved to be adequately constant and independent of the degree of metabolic acidosis, the degree of venous P_{CO_2}, the time of exposure of the blood on the oxygenator screens and the thickness of the blood films. (*Gleichmann, U., and others: Carbon Dioxide Exchange in the Vertical-Screen Oxygenator with Addition of 3 Per Cent Carbon Dioxide in Combination with Moderate Hypothermia, J. Thor. Cardiov. Surg. 45: 628 (May) 1963.*)

RESPIRATION CONTROL Actions of carbon dioxide and sodium salicylate on central control of respiration has been studied in cats anesthetized with pentobarbital. Carbon dioxide and salicylate differed in the following respects: (1) Carbon dioxide stimulated the depth of respiration to a proportionately