

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 pH 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 pH. 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. Cardio. 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 dichloroisoproterenol (a beta blocker) and phentolamine (Regitine). Ventricular contractile force, arterial pressure and femoral blood flow were measured to de-

termine 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 epinephrine. (Wood, W. B., Manley, Jr., E. S., and Woodbury, R. A.: *Effects of CO₂ 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_{CO₂} proved to be adequately constant and independent of the degree of metabolic acidosis, the degree of venous P_{CO₂}, 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. Cardio. 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

greater extent than rate, whereas salicylate produced the reverse effect. (2) After vagotomy carbon dioxide reduced the rate of breathing although it continued to augment depth, whereas salicylate still effected its characteristic stimulation of rate and depth. (3) Both carbon dioxide and salicylate caused an elevation in threshold for sustained inspiration, but they differed in their effects upon threshold for baseline shift and upon maximum inspiration. Carbon dioxide and salicylate have independent actions in their respective influences upon central control of respiration. (Rosenstein, R., and Borison, H. L.: *Actions of Carbon Dioxide and Sodium Salicylate on Central Control of Respiration in Cats*, *J. Pharmacol. Exper. Ther.* 139: 361 (Mar.) 1963.)

BUBBLE OXYGENATION Hypotension, atelectasis, and renal failure have followed prolonged cardiopulmonary bypass using the bubble oxygenator. Experiments show that serotonin and histamine are released from dog platelets and leukocytes that have been destroyed. Bronchoconstriction, hypotension, and hepatic passive congestion can be produced by these agents. Red cell hemolysis with liberation of adenosine compounds, increase in acid metabolites, and denaturation of protein may also be implicated. (Hollenberg, M., and others: *Vasoactive Substances Liberated by Prolonged Bubble Oxygenation*, *J. Thor. Cardio. Surg.* 45: 402 (Mar.) 1963.)

COUNTERPULSATION Diastolic augmentation or counterpulsation is a form of synchronous assistance during bypass. With properly synchronized counterpulsation, a reduction in mean systolic pressure, tension-time index, left ventricular work, and myocardial oxygen consumption is obtained. The reduction in myocardial oxygen consumption, however, did not parallel the fall in tension-time index. Mechanical efficiency fell and the oxygen cost of pressure generation by the left ventricle rose. Premature triggering of the pump systole resulted in an actual increase in myocardial oxygen consumption despite a fall in tension-time index. (Soroff, H. S., and others: *Effects of Counterpulsation on Left Ventricular Oxygen Consumption and Hemodynamics*, *Circulation* 27: 722 (Apr.) 1963.)

BYPASS ACIDOSIS Diffuse cellular hypoxia and severe metabolic acidosis may develop during extended periods of cardiopulmonary bypass. Since the liver can not metabolize anabolic substrates at low temperatures, metabolites (lactate) must be handled during the immediate postperfusion period. If the repair is inadequate or the heart fails, further hypoxia results. Respiratory acidosis due to perfusion-induced pulmonary damage is equally dangerous. Neutralization of the increased hydrogen-ion concentration present in the heparinized priming blood sufficed to preserve the patient's intrinsic buffers for use in compensation of the lactic acidemia noted at the termination of perfusion. When the arterial pH and plasma bicarbonate content became markedly reduced prior to the end of bypass, the administration of an additional quantity of buffer to the perfusion system was sufficient to restore acid-base equilibrium. (Moore, D., and Bernhard, W. F.: *Method for the Control of Severe Alterations in Acid-Base Equilibrium*, *Circulation* 27: 665 (Apr.) 1963.)

BLOOD AGGLUTINATION Low molecular weight dextran in the priming mixture of a pump oxygenator for clinical extracorporeal circulation almost uniformly increased the electric charge of red blood cells. When 5 per cent dextrose in water was used there was no change in electric charge. The increase in red blood cell electronegativity, associated with the presence of low molecular weight dextran, appeared to decrease aggregation and permit improved tissue perfusion. (Bernstein, E. F., and others: *Effect of Low Molecular Weight Dextran on Red Blood Cell Charge During Clinical Extracorporeal Circulation*, *Circulation* 27: 816 (Apr.) 1963.)

MANNITOL Mannitol infusion has significantly decreased plasma hemoglobin increase during mitral and aortic valvular replacement. The mechanism by which mannitol decreases the build-up rate of plasma hemoglobin is unknown, but seems to be other than by renal excretion of hemoglobin. (Porter, G. A., and others: *Prevention of Excess Hemolysis During Cardiopulmonary Bypass by the Use of Mannitol*, *Circulation* 27: 824 (Apr.) 1963.)