Literature Briefs

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Literature Briefs were submitted by Drs. A. Boutros, D. R. Buechel, W. Mannheimer, D. H. Morrow, F. C. McParland, J. W. Pender and H. Roc. Briefs appearing elsewhere in this issue are part of this column.

Circulation

CEREBRAL BLOOD FLOW Cerebral blood flow was measured in baboons anesthetized with pentobarbital. The blood flow increased with hypercapnia and hypoxia and was independent of mean arterial pressures between 60 and 130 mm Hg. Blood flow decreased during sympathetic nerve stimulation and increased after sympathetic nerve section. The blood flow response to hypoxia and hypercapnia was reduced when the carotid, vagus and aortic nerves were cut. Conversely, when these nerves were stimulated, blood flow increased. Stimulation of the seventh cranial nerve caused an increase of blood flow if the vagus nerves were cut. The results indicate that cerebral blood vessels are under reflex control. The data do not elucidate the reflex level at which vasoconstrictor–vasodilator activities interact upon cerebral vessels nor the intrinsic response of cerebral vessels to changes in blood gases. (James, I. M., and others: Observations on the Extrinsic Neural Control of Cerebral Blood Flow in the Baboon. Circ. Res. 25: 77 (July) 1969.)

VENOUS BLOOD GASES Central venous oxygen saturation (CV\textsubscript{O\textsubscript{2}}) in the superior vena cava, innominate vein, or right atrium (RA) and mixed venous oxygen saturation (MV\textsubscript{O\textsubscript{2}}) in the pulmonary artery were measured simultaneously in 24 critically-ill patients. Mean CV\textsubscript{O\textsubscript{2}} was significantly greater than mean MV\textsubscript{O\textsubscript{2}}, whereas RA\textsubscript{O\textsubscript{2}} and MV\textsubscript{O\textsubscript{2}} agreed closely. When the patients were divided into groups depending on the presence or absence of left ventricular failure and/or shock, the results showed that CV\textsubscript{O\textsubscript{2}} was an accurate reflection of MV\textsubscript{O\textsubscript{2}} only in the absence of failure and shock. The reason for the higher CV\textsubscript{O\textsubscript{2}} than MV\textsubscript{O\textsubscript{2}} (normally MV\textsubscript{O\textsubscript{2}} is higher) is that low cardiac output is associated with greater decreases in renal and splanchnic circulations than in cerebral circulation. This leads to decreased inferior caval oxygen saturation and, finally, decreased MV\textsubscript{O\textsubscript{2}}. The close correlation of RA\textsubscript{O\textsubscript{2}} and MV\textsubscript{O\textsubscript{2}} justifies use of RA oxygen content to approximate cardiac output by means of the Fick formula. (Scheinman, M. M., and others: Critical Assessment of Use of Central Venous Oxygen Saturation as a Mirror of Mixed Venous Oxygen in Severely Ill Cardiac Patients. Circulation 40: 165 (Aug.) 1969.)

VENOUS PRESSURE The method is simple, but so much of our thinking, and it is only comparatively recently that the complex, interacting and interdependent factors which affect this easy measurement are becoming widely appreciated, adding greatly to the value of the information derived. The margin between adequate expansion of the vascular column and overload is narrow, especially in the old and in patients who have sepsis or burns, in pregnancy, and in those with heart disease. For such cases, the trial of small transfusions during careful observation of central venous pressure is an essential part of intensive care. Central venous pressure measurement needs a minimum of instrumentation but a modicum of sophistication in interpretation of results. (Beard, J.: Venous Pressure and Other Thoughts. Proc. Roy. Soc. Med. 62: 655 (July) 1969.)

PACKED ERYTHROCYTES Human blood is clearly a valuable therapeutic agent in modern medical practice. The total requirement for blood grows annually, as does the difficulty in obtaining the quantities required. In most circumstances in which transfusion is