Literature Briefs

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Briefs were submitted by Drs. C. M. Ballinger, N. Bergman, R. B. Boettner, A. Boutros, D. R. Buechel, H. F. Cascorbi, R. B. Clark, D. Duncalf, W. H. Mannheimer, F. C. McPartland, D. H. Morrow, R. C. Morton, J. W. Pender, A. D. Randall, L. J. Saidman, and A. D. Sessler. Briefs appearing elsewhere in this issue are part of this column.

Circulation

VENOUS OXYGEN SATURATION Serial measurement of central venous oxygen saturation may be useful for detecting changes in myocardial function in patients with myocardial infarction. Under basal conditions, the changes in central venous oxygen saturation can be used to determine whether cardiac output is changing and, if so, the direction in which it is changing. Thus, the effects of various therapeutic modalities such as cardiotonic agents, diuretics, and analgesic agents, and the effect of cardiac arrhythmias on cardiac output can be easily assessed using this simple measurement. The detection of a central venous oxygen saturation of less than 60 per cent or the decrease of a previously normal level to less than 60 per cent strongly suggests heart failure. Although there are occasional false-positive and false-negative results of this test, it appears more sensitive for detection of heart failure than elevation of central venous pressure. (*Goldman, R. H., and others: The Use of Central Venous Oxygen Saturation Measurements in a Coronary Care Unit, Ann. Int. Med. 68: 1280 (June) 1968.*)

HEMORRHAGIC SHOCK Although it is a homeostatic mechanism in the initial response to hemorrhage, sympathoadrenal hyperactivity when sustained is deleterious to the organism and is the primary factor in the development of irreversible shock. Previous theories that the deterioration and the metabolic acidosis resulted from vasoconstriction and tissue hypoxia must be reassessed. It appears that the course of the deterioration is much more fundamental and is mediated at the cellular level through alpha and beta receptors. Combined alpha- and beta-adrenergic blockade provides significant improvement in the tolerance of the organism to hemorrhagic hypotension. The present study leaves many unanswered problems, but suggests that future experimental and therapeutic approaches to irreversible hemorrhagic shock should consider the problem in terms of alpha- and beta-adrenergic receptor activity rather than in terms of vasoconstriction and tissue perfusion. (*Irving, M. H.: The Sympatho-adrenal Factor in Haemorrhagic Shock, Ann. Roy. Coll. Surg. Eng. 42: 367 (June) 1968.*)

SHOCK TOLERANCE Tolerance to hemorrhagic shock was induced in rats by a series of injections of *E. coli* endotoxin. Tolerance was manifested by: (a) greater bleed-out volume to maintain a mean arterial pressure of 30 mm Hg; (b) delayed uptake in blood; and (c) longer survival time. In contrast to the control rats, the tolerant rats demonstrated: (a) greater fall and delayed rise in hematocrit; (b) less rapid decline in arterial *p*H; (c) slower rise in blood glucose with ultimately greater hyperglycemia. The production of tolerance has been interpreted as due to: (1) less vascular sensitivity to the circulatory epinephrine during prolonged shock; (2) an insulin-like effect of endotoxin on cellular metabolism. (*Drucker, W. R., and others: Metabolic Factors Associated with Endotoxin-induce tolerance for Hemorrhagic Shock, Surgery 64: 75 (July) 1968.*)

SHOCK CHANGES A 40 per cent reduction of oxygen uptake and carbon dioxide production during experimental shock in dogs