Literature Briefs

C. Philip Larson, Jr., M.D., Editor

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

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reverted to normal with reinfusion. This was associated with a 50 per cent increase in minute ventilation. Three times as much air was ventilated to exchange 100 ml of oxygen during shock as during the control period. The mechanics of ventilation were also altered. During the shock period, the ventilatory work increased, due to a combination of increased respiratory rate and increased ventilatory pressure. Although gas exchange returned to normal with reinfusion, the increase in minute ventilation and ventilatory work and the decrease in ventilatory efficiency persisted during the follow-up period. This may be due to an acute congestive process causing postcapillary venous constriction. (*Cook, W. A., and Webb, W. R.: Pulmonary Changes in Hemorrhagic Shock, Surgery 64: 85 (July) 1968.*)

SHOCK Interstitial fluid pressure measured in chronically implanted perforated chambers in dogs was always negative. After a period of hypotensive shock, interstitial fluid pressure remained low and was not restored to normal by replacement of the shed blood alone. The addition of 5 per cent of weight with lactated Ringer's solution restored interstitial fluid pressure to normal. The use of clinical dextran or low-molecular-weight dextran instead of lactated Ringer's solution after a period of hypotensive shock lowered interstitial fluid pressure further. The survival rates of dogs treated by blood replacement plus lactated Ringer's solution or plus dextran were the same, suggesting that increased tissue perfusion and oxygen consumption are more important than replacement of "extracellular fluid losses" in the treatment of hypotensive shock. (*Hopkinson, B. R., and others: Interstitial Fluid Pressure Changes during Hemorrhage and Blood Replacement with and without Hypotension, Surgery 64: 68 (July) 1968.*)

VASOACTIVE AGENTS Vasoactive agents augment, mimic or modify the action of neurohumoral transmitters released at the ganglia or effector cells. Ganglionic blocking agents do not prevent the release of acetylcholine at preganglionic nerve endings, but block the receptor sites and prevent postjunctional nerve action potential. Methyldopa, reserpine and chlorpromazine block storage of norepinephrine and eliminate mobile and fixed stores in sympathetic ganglion cells and adrenal medulla. The strongest alpha-receptor-stimulating agent is angiotensin II, and it also is a powerful stimulant for secretion of aldosterone. Both alpha-blocking and beta-stimulating agent have been advocated for treatment of shock, along with adequate replacement of blood volume. Nearly-pure beta-receptor blocking agents are used to treat cardiac arrhythmias, but these drugs may cause bradycardia and arterial hypotension. (*McQuarrig, D. G., and Humphrey, E. W.: Vasopressors and Vasodilators in Surgery, Surg. Clin. N. Amer 48: 877 (Aug.) 1968.*)

Respiration

OXYGEN AND CORONARY FLOW Studies in open-chest dogs were undertaken to assess the effects of abrupt changes in inspired oxygen tensions on the relationship between isometric systolic tension and coronary flow. Changing the inspired oxygen concentration from 25 to 100 per cent resulted in consistent and equivalent reductions in isometric systolic tension and coronary flow, with alterations in coronary flow preceding those of isometric systolic tension. These changes were not abolished by alpha, beta, or complete sympathetic blockade. Although the alterations in coronary flow and isometric systolic tension were similar in both direction and magnitude, they were not related in a cause-and-effect manner. Experiments in which coronary flow was maintained constant by pump perfusion showed that, when O_2 tension was increased, isometric systolic tension decreased in the same degree as previously. These data support the view that alterations in coronary flow are not entirely dependent on myocardial oxygen demands but that there is some intrinsic component of the coronary vessels sensitive to alterations in O_2 tensions. The observed decrease in isometric systolic tension at high O_2 tensions may be an early manifestation of oxygen toxicity. (*Daniell, H. B., and Bagwell, E. E.: Effects of High Oxygen on Coronary Flow and Heart Force, Amer. J. Physiol. 214: 1454 (June) 1968.*)

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