Literature Briefs

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Literature briefs were submitted by Drs. L. Bachman, D. R. Buechel, R. B. Clark, F. C. McPartland, D. Morrow, R. C. Morton, E. S. Munson, J. W. Pender, H. Roe, L. J. Saidman, A. D. Sessler, and C. J. Wilkinson. Briefs appearing elsewhere in this issue are part of this section.

Circulation

VAGAL INFLUENCE Stimulation of the left or right vagus nerves created equal slowing of pulse rates in anesthetized dogs. Left vagal section accelerated the pulse insignificantly; right vagal section resulted in a significant increase in pulse rate. These data suggest that the right vagus normally contributes more than the left to vagal restraint. Vagal stimulation caused equal prolongation of A-V conduction time; stimulation of the left vagus caused second-degree A-V block. Two physiologically-separate areas may exist in the A-V node; one determining whether or not an impulse crosses the system at all, and a second determining the rate at which it traverses the node. The first areas is affected preferentially by left vagal efferent activity; the second area is affected equally by both vagi. (*Hamlin, R. L., and Smith, C. R.: Effects of Vagal Stimulation on S-A and A-V Nodes, Amer. J. Physiol. 215: 560 (Sept.) 1968.*)

ABSTRACTER'S COMMENT: These dogs were anesthetized with fentanyl and droperidol, followed by an intravenous injection of pentobarbital. The influence of these agents on SA and AV nodal activity can only be assumed to have been insignificant.

ALCOHOL AND THE HEART Although ethyl alcohol has been used (by prescription and otherwise) for angina pectoris since Heberden first described the condition in 1786, recent studies indicate 0.5 g/kg (3–4 jiggers) significantly depresses myocardial function. Stroke volume, cardiac output, tension time index, and the ability to respond to an afterload are all depressed. Exercise after alcohol produces ST and T wave changes not seen prior to inbibation. Hypertonic sucrose in amount producing equal changes in plasma osmolality have a similar effect on myocardial function, indicating that the acute depression noted with alcohol may be due to osmotic changes. (*Conway, N.: Hemodynamic Effects of Ethyl Alcohol in Coronary Artery Disease, Amer. Heart J. 76: 581 (Oct.) 1968.*)

LACTATE Lactated Ringer's solution (LRS), four times the volume of shed blood, administered one hour after bleeding, resuscitated 100 per cent of dogs bled 30 per cent of their blood volumes. If given immediately following hemorrhage, LRS resuscitated 80 per cent of dogs bled 50 per cent of their blood volumes and 60 per cent of animals bled 65 per cent of blood volumes. Even when volume replacement with LRS was delayed an hour, there was an increase in survival and a decrease in the level of circulating lactate. The exogenous lactate in LRS is cleared rapidly after blood volume replacement in animals in hemorrhagic shock. (*Trinkle, J. K., and others: Metabolism of Lactate Following Major Blood Loss, Surgery 63: 782 (May) 1968.*)

pH STABILITY *p*H was studied in dogs exposed to a hypobaric and hypercarbic atmosphere. Two groups of six dogs each were exposed continuously for nine days to a total pressure of 260 mm Hg, comprised of oxygen with 60 or 90 mm Hg CO_2. Arterial samples were collected daily. Exposure to a P_{CO_2} of 60 mm Hg resulted in a reduction in arterial *p*H from 7.42 to 7.32, followed by recovery in four to five days to between 7.36 and 7.39. Exposure to 90 mm Hg P_{CO_2} resulted in a reduction in *p*H to 7.21, with recovery to 7.30.

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Arterial standard bicarbonate levels increased from 23 to 28 mm/l during exposure to 60 mm Hg C_{O_2} and to near 31 mm/l during exposure to 90 mm Hg CO_2. These data indicate at least as good, and perhaps better, *p*H stability in an hypobaric atmosphere as has been found at sea-level pressures. (*Popelko, W. E.: Adaptation of Dogs to 60 and 90 mm Hg CO_2 at a Total Pressure of 260 mm Hg, Aerospace Med. 39: 1294 (Dec.) 1968.*)

POSTVAGAL TACHYCARDIA Vagal stimulation causes sinus arrest or atrioventricular block and fall in blood pressure. After the stimulation, the sinus rate increases to above the control value. This postvagal tachycardia could result from many mechanisms, but a direct release of catecholamines or similar substances by the vagus has been suggested, since vagal stimulation or administration of acetylcholine in atropinized preparations produces tachycardia and liberates an epinephrine-like substance. In anesthetized dogs, the right vagus was crushed in the neck and the peripheral end stimulated. Termination of stimulation consistently was followed by transient sinus tachycardia, which persisted despite maintenance of normal blood pressure, thereby eliminating reflex hypotensive mechanisms. To stimulate the vagus reflexly, the carotid sinus was compressed. Termination of this reflex vagal stimulation was followed by tachycardia. Bilateral vagotomy abolished this response. Tetraethylammonium applied to the sinus node directly blocked the vagus induced tachycardia. Postvagal tachycardia was also abolished by prior administration of reserpine and restored after norepinephrine administration. Postvagal tachycardia results from the excitation of cholinergic parasympathetic fibers, leading to liberation of catecholamines, possibly from chromaffin cells. (*Copen, D. L., Cirillo, D. P., and Vassalle, M.: Tachycardia following Vagal Stimulation, Amer. J. Physiol. 215: 696 (Sept.) 1968.*)

EEG AND ALKALOSIS The EEG was monitored continuously during all cardiac procedures, and blood gases and *p*H were examined at frequent intervals. In a retrotspective study of more than 1,600 cases, the EEG did not prove to be as sensitive an indicator of alkalosis as has been suggested in the literature. However, an interesting case was reported in detail: a sudden drop in P_{CO_2} from 36 to 10 mm. Hg with simultaneous appearance of "spikes" in the EEG tracing. Although alkalosis was corrected promptly, the EEG changes persisted for several days postoperatively. These findings are similar to those reported by A. Harden and B. M. Ashton (EEG Clin. Neurophysiol. 22: 128 (Feb.) 1967.) (*Passelecq, J., Arfel, G., and Dubost, C.: EEG Manifestations of Alkalosis, Anaesth. Analg. 24: 535 (Sept.) 1967.*)

MONITORING A system of measurement and display was developed to care for patients acutely ill with cardiac disease or following open-heart surgery. It is based on the use of sensing systems designed for minimal disturbances of the patient or his nurses. Information such as vascular pressures, EKG, respiration and ventilatory settings is processed by digital computer and displayed as numerical or graphic data at the bedside. Therapeutic management is eased and improved. The ventilatory measurements provide constant assurance that the patient is receiving the intended tidal and minute volumes. (*Osborn, J. J., and others: Patient Monitoring of Acutely Ill Patients by Digital Computer, Surgery 64: 1057 (Dec.) 1968.*)

HALOTHANE Cardiovascular responses to induction of anesthesia with halothane–oxygen, as well as to changes in alveolar concentration from one steady-state level to another, were studied in eight subjects. Heart rate, arterial and right atrial pressures, stroke volume, cardiac output, left ventricular minute work, and total peripheral resistance were recorded beat-to-beat. The last four variables were calculated by a ballistocardiograph–analogue computer system. Induction of anesthesia increased significantly all variables except total peripheral resistance and arterial and right atrial pressures. The last two are the variables conventionally measured during anesthesia and surgery. The response to changing concentration was delayed significantly, and decreased in magnitude late, rather than