Literature Briefs

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Briefs were submitted by Drs. C. M. Ballinger, Norman Bergman, Peter P. Bosomworth, M. T. Clarke, H. S. Davis, Deryck Duncalf, J. E. Eckenhoff, Martin Helrich, G. Hohmann (Germany), J. J. Jacoby, F. C. McPartland, Alan Paterson, Alan D. Randall, H. S. Roe, Norman Rosenbaum, P. H. Sechzer, and E. A. Talmage. Abstracts of Japanese and Russian articles were prepared by Excerpta Medica Foundation. Briefs appearing elsewhere in this issue are part of this column.

OXYGEN TOXICITY Animals were exposed to 95–99 per cent oxygen for 240 hours. Labored breathing and lethargy occurred after 15–20 hours in rats, 36–42 hours in dogs, and 72–96 hours in monkeys. Most showed extensive bilateral pleural effusions and pulmonary edema along with emphysema and dilatation of the tracheobronchial tree. If they survived the 240 hours the pleural effusion was less but there was severe organ and tissue damage with edema of the adventitia of pulmonary vessels and tracheobronchial tree. Necrosis of the pulmonary vein and thickening of the pulmonary arterioles were also seen. (*Weir, F. W., and others: Study of Effects of Continuous Inhalation of High Concentrations of Oxygen at Ambient Pressure and Temperature, Aerospace Med. 36: 117 (Feb.) 1965.*)

HYPEROXYGENATION Mice were injected intraperitoneally with a suspension of pneumococci. The times between injection and death were computed until 90 per cent of the mice had died. Some of the animals were exposed to two atmospheres of oxygen shortly after the intraperitoneal injection. In two of eight experiments the difference between treated and control groups was highly significant in that the time from injection to death was prolonged. (*Ross, R. M., and McAllister, T. A.: Protective Action of Hyperbaric Oxygen in Mice with Pneumococcal Septicaemia, Lancet 1: 579 (Mar. 12) 1965.*)

DECOMPRESSION ILLNESS On two occasions attending personnel exhibited decompression illness symptoms and in both instances the individuals were anesthetists with "a rather thick layer of fat" who remained seated on a stool at the patient's head during decompression. Circulation in the layers of fat in the buttocks was so limited that stored nitrogen was not transported away gradually during decompression but formed bubbles after these doctors got up. The complication has not recurred since it has been required that all personnel be on their feet and moving about during decompression. (*Boerema, I.: Editorial: The Use of Hyperbaric Oxygen, Amer. Heart J. 69: 289 (Mar.) 1965.*)

PULMONARY EMBOLISM Experimental pulmonary thromboembolism in dogs supports the clinical observation that severe systemic arterial hypotension and death may result from a relatively small embolus. The weight of the pulmonary embolus, the magnitude of the pulmonary hypertension, and the extent of the decrease in cardiac output could not be consistently related to the severity of postembolic systemic hypotension. Sympathectomy did not appreciably alter the pulmonary hemodynamic changes of pulmonary embolism in dogs but favored systemic arterial hypotension. Bilateral vagotomy produced a marked decrease in the tachypneic and hypotensive responses to pulmonary embolism in dogs and increased the weight of clot embolus required to produce fatal postembolic shock. Systemic arterial hypotension resulting from pulmonary embolism, exclusive of that caused by the massive pulmonary embolus, is initiated by a vasodepressor response mediated by the vagus; and recovery is dependent in part upon the ability of the peripheral vascular bed to react appropriately with reflex vasoconstriction. Value of a pressor agent (metaraminol) in contrast to one that decreases peripheral vascular resistance (isoproterenol) was emphasized.