Literature Briefs

John W. Pender, M.D., Editor

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.

(Parmley, L. F., Jr., North, R. L., and Pickens, G. E.: Pulmonary Embolism as a Cause of Systemic Hypotension and Shock, Amer. J. Cardiol. 15: 333 (Mar.) 1965.)

HYPERBARIC OXYGENATION Hyperbaric oxygenation was used in the treatment of purpura gangrenosa occurring in a 19 month old Negro child. Within three days, eight five-hour exposures were given at two atmospheres absolute pressure. The child was placed in a pediatric high humidity oxygen tent, and this was in turn placed inside the treatment compartment of the hyperbaric chamber. During pressurization the oxygen tension of inspired air exceeded 1,400 mm. of mercury. The tent was usually opened during the midpoint of each five-hour period for 15 to 20 minutes for medication, nursing care, and to interrupt pulmonary exposure to extreme hyperoxia. Respiration of oxygen at increased atmospheric pressures for prolonged periods is associated with a specific, progressive picture of pulmonary insufficiency terminating in death. However, patients have not demonstrated recognizable symptoms of pulmonary toxicity when breathing oxygen at two atmospheres pressure. *(Waddell, W. B., and others: Purpura Gangrenosa Treated with Hyperbaric Oxygenation, J.A.M.A. 191: 971 (Mar. 22) 1965.)*

PULMONARY EMBOLISM The right heart of dogs was bypassed using a reservoir-pump system. Following experimental pulmonary embolization with glass microspheres or autologous clots, pulmonary artery pressure and pulmonary vascular resistance increased while arterial oxygen saturation decreased. Calculated venous admixture increased to 30 per cent of cardiac output. Imposition of 20 cm. of water resistance to exhalation restored saturation and admixture to pre-embolization values. Decrease in hemoglobin saturation in pulmonary embolism is caused by perfusion of poorly or nonventilated areas of lung caused by collapse of respiratory units due to presence of edema fluid. Pulmonary hypertension following pulmonary embolization may be due to vasoconstriction in addition to mechanical blocking of pulmonary vessels. *(Caldini, P.: Pulmonary Hemodynamics and Arterial Oxygen Saturation in Pulmonary Embolism, J. Appl. Physiol. 20: 184 (Mar.) 1965.)*

EMBOLI During extracorporeal circulation a wide variation in tolerance to air emboli was found in dogs. Air passed rapidly through the coronary capillaries in some while forming a permanent obstruction in others. Carbon dioxide emboli, though causing much less injury than air, were not completely innocuous. The most effective method for prevention of air emboli is the use of induced ventricular fibrillation in combination with decompression of the left ventricle. If emboli have occurred, increasing the perfusion pressure and manually massaging the heart is usually effective. *(Spencer, F. C., and others: Significance of Air Embolism During Cardiopulmonary Bypass, J. Thor. Cardiov. Surg. 49: 615 (Apr.) 1965.)*

PULMONARY EMBOLI Transient pulmonary hypertension lasting up to an hour can be produced in dogs by the introduction of air into the pulmonary artery. This hypertension is due to mechanical increase in pulmonary vascular resistance by the surface tensions of the many bubbles in small vessels, which also cause temporary decrease in compliance and decrease in pulmonary function. A major portion of the vascular bed needs to be obstructed by air emboli before any pressure change is apparent. Pulmonary air embolism during open heart surgery, although tolerated in a person with normal lungs, may result in significant increase in pulmonary hypertension at the critical time of bypass termination in patients with preoperative pulmonary vascular changes. *(Anderson, R. M., and others: Pulmonary Air Emboli During Cardiac Surgery, J. Thor. Cardiov. Surg. 49: 440 (Mar.) 1965.)*

HYPOXIA In chronic hypoxia at high altitudes cardiac output is not increased because of a compensatory increase in oxygen-carrying capacity of blood. Increased volume of ventilation was achieved entirely by greater tidal volume; however, the number of moles of air ventilated actually decreased. Basal oxygen uptake increased slightly. With exercise, at any given level of oxygen uptake, the volume of air ventilated was greater at high altitude.