

Literature Briefs

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Briefs were submitted by Drs. Peter P. Bosomworth, M. T. Clarke, H. S. Davis, Deryck Duncalf, Martin Helrich, F. C. McPartland, W. H. Mannheim, R. E. Ponath, Alan D. Randall, and H. S. Roe. Briefs appearing elsewhere in this issue are a part of this column. Abstracts of Russian and Japanese literature were obtained from *Excerpta Medica Foundation*.

HYPERBARIC OXYGEN Eight newborn infants with progressive hypoxia were placed in oxygen at pressures exceeding atmospheric for periods of three to 24 hours. The clinical diagnosis in these infants was hyaline membrane disease. All died. In six of the eight children, hyaline membranes were found at autopsy and all eight demonstrated severe atelectasis. Although a very temporary improvement was observed in arterial oxygen and color of the infants, the use of this tool under present circumstances seemed dubious and could not be advantageously utilized in these infants. (Cochran, W. D., and others: *Clinical Trial of High Oxygen Pressure for the Respiratory-Distress Syndrome*, *New Engl. J. Med.* 272: 347 (Feb.) 1965.)

HYPERBARIC OXYGEN Exposure to hyperbaric oxygen results in several types of tissue damage, some of which are considered to be irreversible. Acute oxygen poisoning of the nervous system is well documented. An equally significant hazard is the technique of intermittent compression, decompression and recompression, whereby patients are exposed to the hazards of decompression illness and cerebral air embolism. (Gillen, H. W.: *Neurologic Hazards of Hyperbaric Oxygen Exposure*, *Dis. Chest*, 47: 369 (Apr.) 1965.)

HYPERBARIC OXYGEN Rats exposed to oxygen at six atmospheres pressure for periods of 30 minutes were protected against both gross

lung damage and convulsions by intraperitoneal doses of gamma-aminobutyric acid. Glucose did not afford this protective effect. Other authors have reported similar protection by tris buffer and by vitamin E. A relation between occurrence of seizures and lung damage is suggested. Probably there are two types of pathologic conditions in the lung. The slow developing type occurs at low oxygen pressures and is not related to seizures. The fast developing type develops when high oxygen pressures are used and is associated with seizures, possibly the result of muscular contractions or release of a compound such as norepinephrine. (Wood, J. D., Stacey, N. E., and Wilson, W. J.: *Pulmonary and Central Nervous System Damage in Rats Exposed to Hyperbaric Oxygen and Protection Therefrom by Gamma-Aminobutyric Acid*, *Canad. J. Physiol.* 43: 405 (May) 1965.)

HYPERBARIC OXYGEN Arterial hypoxia can be relieved by increasing the concentration of inspired oxygen under normal pressure. However, hypoxemia caused by venous admixture of more than 40 per cent to pulmonary capillary blood cannot be corrected by administration of pure oxygen under atmospheric pressure as seen in congenital deformities with marked right-to-left shunt. Mean capillary and venous oxygen pressure also falls with decreasing perfusion and increasing extraction of oxygen. Stagnation hypoxia develops which is frequently seen following cardiac surgery. A small portable high pressure chamber, designed to hold one patient only, was developed for treatment of such conditions. Electrocardiogram, electroencephalogram, body temperature, blood pressure and urine output can be monitored. Arterial and venous blood samples can be taken and infusions administered. Concentration of gases, humidity and temperature inside the chamber can be measured. (Rodewald, G., Harms, H., and Dönhardt, A.: *De-*