

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. Mannheimer, 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-

sign and Use of a Hyperbaric Oxygen Chamber (German), Der Anaesthetist 14: 100 (Apr.) 1965.

(Meyerowitz, B. R.: Present Status of Hyperbaric Oxygenation, Amer. J. Surg. 109: 611 (May) 1965.)

HYPERBARIC OXYGENATION In a controlled study, the effects of hyperbaric oxygenation (100 per cent oxygen at three atmospheres) on hemodynamics and mortality rate were studied in dogs in whom acute myocardial infarction was produced by intracoronary microsphere injection. The 24 hour mortality rate was 30 per cent in the hyperbaric oxygen group as compared with 85 to 90 per cent in the control group, which included animals breathing 100 per cent oxygen at one atmosphere. There was less ventricular fibrillation, atrio-ventricular block, reduction of cardiac output and central aortic pressure in treated than in control animals, although the anatomical extent of the infarction was similar in the two groups. (*Kuhn, L. A., and others: Hemodynamic Effects of Hyperbaric Oxygenation in Experimental Acute Myocardial Infarction, Circulat. Res. 16: 499 (June) 1965.*)

HYPERBARIC OXYGENATION The generally optimistic reports of the efficacy of hyperbaric oxygenation in a variety of conditions is questioned. Doubt is cast upon the actual advantages of hyperbaric oxygen over 100 per cent oxygen at ambient pressure in treating shock from various causes, barbiturate poisoning, hyaline membrane disease and congenital heart disease, particularly in view of the fact that earlier widely quoted clinical studies in these areas involved administration of mask oxygen in an environment of compressed air in such a manner as to suggest oxygen concentrations inhaled were no greater than 40 per cent instead of the anticipated 100 per cent, thereby resulting in final plasma tension little greater than achieved with efficient inhalation of 100 per cent oxygen at ambient pressure. Strikingly good results reported in a relatively small number of cases of carbon monoxide poisoning, of certain anaerobic infections, of selected patients with severe myocardial infarction or ischemia of extremities, and of asphyxia neonatorum not due to hyaline membranes, emphasize the need for further careful studies involving controls wherever possible to clarify the true value of the method.

PULMONARY RESISTANCE Simultaneous measurements of pulmonary compliance and resistance were made in supine, presurgical patients, first awake and then continuously during 45 to 60 minutes of general anesthesia. Halothane was the primary agent, while thiopental was used for induction in 75 per cent. Compliance decreased 25 per cent after induction but changed little during the remainder of the experiment. Resistance increased more than a 100 per cent without an oral airway. It was still significantly elevated after the insertion of an oral airway and returned to control levels only after endotracheal intubation. Thiopental induction was associated with a greater decrease in compliance and tidal exchange and a larger increase in resistance than inhalation induction. Changes in compliance and resistance during anesthesia were unrelated to each other. (*Gold, M. I., and Helrich, M.: Pulmonary Mechanics During General Anesthesia in Normal Man, Fed. Proc. 24: 268 (Mar.-Apr.) 1965.*)

ARTIFICIAL VENTILATION The most common cause of hypoxia during artificial ventilation is shunting of mixed venous blood past underventilated alveoli. The two most common causes of shunting are atelectasis and interstitial pulmonary edema. While little is known about the origin and treatment of the latter, diffuse atelectasis can be prevented by the use of large tidal volumes and is promoted by the use of small tidal volumes. Since constant use of large tidal volumes may be undesirable for several reasons, it is fortunate that diffuse atelectasis also is partially prevented by a ventilation pattern incorporating intermittent use of large tidal volumes as produced by deep inflations several times hourly. (*Pontoppidan, H.: Prolonged Artificial Ventilation, Postgrad. Med. 37: 576 (May) 1965.*)

INDUCED COUGH Production of involuntary cough by means of intermittent instillation of small quantities of saline or mucolytic agents through an indwelling polyethylene tracheal