

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

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

**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

catheter is advocated as a simple, safe and effective means of reducing postoperative pulmonary complications caused by retained secretions. The catheter is inserted through the cricothyroid membrane by means of a 17 gauge needle and left in place for periods of time up to several weeks with no discomfort to the patient. The method is useful in patients who cannot or will not cough voluntarily. However, no data are presented on results in the 200 patients so treated, other than to state that complications due to the method were absent. (Myers, R. N., Shearburn, E. W., and Haupt, G. J.: *Prevention and Management of Pulmonary Complications by Percutaneous Polyethylene Tube Tracheostomy*, *Amer. J. Surg.* 109: 590 (May) 1965.)

**INFANT TRACHEOSTOMY** By comparing the findings in 62 infants receiving tracheostomy in the period from 1936 to 1953 with those of 86 during the period from 1954-1963, a clear-cut trend was away from the operation for acquired inflammatory lesions and toward operation for congenital malformation and neoplasms of the airway, with an overall increase in total incidence in recent years as indications have broadened and skepticism for the procedure lessened. Morbidity associated with the procedure has been lowered through improved surgical techniques, including preoperative establishing of a patent airway by means of a bronchoscope to avoid emergency tracheostomies, and better postoperative management. Complications do still occur and the most common continues to be delayed extubation due to a variety of causes such as granuloma formation, stenosis of trachea or larynx and vocal cord paralysis; acquired inflammatory disease seldom leads to this difficulty. (Holinger, P. H., Brown, W. T., and Maurizi, D. G.: *Tracheostomy in the Newborn*, *Amer. J. Surg.* 109: 771 (June) 1965.)

**INTERSTITIAL FLUID PRESSURE** Pressure-volume curves of interstitial fluid spaces were determined in anesthetized dogs by sampling of pressures in subcutaneously implanted perforated capsules. Pressures within these capsules were recorded by means of inserting into them a needle attached to a Statham pressure transducer. Intracapsule

pressure was assumed to be equal to interstitial pressure. Control interstitial pressures were found to be in the range of minus 4 to minus 9 mm. of mercury in both awake and anesthetized dogs. However, persistent immobilization of the animals led to a gradual pressure rise toward zero of about 1 mm of mercury per hour, which trend reversed rapidly with reinstitution of activity. By means of appropriate fluid administration in both isolated limbs and intact animals, pressure-volume curves were constructed, indicating very low compliance of the interstitial spaces so long as the capsule pressure was in the negative pressure range but a many-fold increase in compliance once ambient pressure was exceeded. These findings explain the "safety factor" which protects against early development of edema, since even in the presence of a low plasma protein oncotic pressure there must be a concomitant large rise in normally negative interstitial pressure (about 7 mm. of mercury increase) before the tissues begin to collect appreciable volumes of fluid. Once this critical point is reached, edema fluid forms rapidly and in large amounts. (Guyton, A. C.: *Interstitial Fluid Pressure: II. Pressure-Volume Curves of Interstitial Space*, *Cir. Res.* 16: 452 (May) 1965.)

**PULMONARY SHUNTING** Large physiologic shunts resulting in hypoxemia were found in all patients breathing air 20 to 24 hours after cardiac surgery. The average shunt on 100 per cent oxygen was 13 per cent of cardiac output. The most important single cause of these shunts was diffuse atelectasis, usually not visible on roentgenograms. Extracorporeal circulation had no apparent effect in increasing physiologic shunting. The increase in shunting on room air, as compared with oxygen, was significant in the group of patients who had mitral valve replacement or open mitral valvuloplasty. This shunting caused by uneven distribution of ventilation in relation to perfusion, was equivalent to a right to left shunt of 12.5 per cent of the cardiac output. The combination of atelectasis and maldistribution resulted in the mean arterial oxygen tension on air of this group being only 53 mm. of mercury. (Hedley-Whyte, J., and others: *Pulmonary Ventilation-Perfusion Relations After Heart*