swallow the dye within two or more hours after the anesthetic. It is suggested that these complications are due to laryngeal incompetence and that this incompetence persists well into the postoperative period. Consequently, oral fluids should not be given for several hours after anesthesia, and patients should be encouraged to lie on their sides slightly face down and to spit out secretions that accumulate in the front of the mouth in an effort to reduce the incidence of these pulmonary complications. (*Tomlin, P. J., Howarth, F. H., and Robinson, J. S.: Postoperative Atelectasis and Laryngeal Incompetence, Lancet 1: 1402 (June) 1969.*)

LACTATE RISE DURING HYPOXIA Thirty anesthetized, paralyzed dogs were ventilated before and after beta-adrenergic block with propranolol to make them hypoxic. During 30-minute hypoxic periods, 15 dogs were made hypocapnic (Pa_{CO_2} 20 torr) and the rest hypercapnic (Pa_{CO_2} 75 torr). Slopes of the lines showing the increases in lactate (L) and excess lactate (XL) in relation to the accumulated net O_2 deficits were alike for all four experimental conditions. Both beta block and hypercapnia acted to shift the lines for L and XL to the right on the net O_2 deficit axis, but the combination of hypercapnia and beta block produced the greatest increase in net O_2 deficit intercept before L and XL appeared. The similarity of the effects of hypercapnia and beta block on these relationships was attributed to inhibition of catecholamine calorigenesis. Calorigenic factors accounted for 70 per cent of the difference between L values in hypocapnic hypoxia without beta block and in hypercapnic hypoxia with beta block. The remainder was attributed to direct effects of *p*H on glycolytic rates. Excess lactate was apparently independent of these direct *p*H effects. (*Cain, S. M.: Diminution of Lactate Rise during Hypoxia by P_{CO_2} and Beta-adrenergic Blockade, Amer. J. Physiol. 217: 110 (July) 1969.*)

HYPERBARIC OXYGENATION Oxygen available to tissues is increased by hyperbaric oxygenation. Hazards to patients include oxygen toxicity, pulmonary and aural atelectasis, pain in sinuses or cavities in teeth, nausea or vomiting, and increased work of breathing. Hazards to medical personnel include decompression sickness, avascular necrosis of bone, and inert gas narcosis. Hyperbaric oxygen is indicated in the treatment of coma due to carbon monoxide poisoning and clostridial infections, and as an alternative to compressed air in air embolism and decompression sickness. It is valuable in a variety of other conditions. (*Chew, H. E. R., Hanson, G. C., and Slack, W. K.: Hyperbaric Oxygenation, Brit. J. Dis. Chest 63: 113 (July) 1969*). ABSTRACTER'S COMMENT: This timely review, containing 149 references, covers the history, terminology, physiology, methods of administration, and hazards of hyperbaric oxygen, and also discusses the methods for its administration.

OXYGEN TOXICITY Baboons, Macaca and squirrel monkeys were exposed to pure oxygen at 720 mm Hg pressure for periods as long as 14 days. Deaths occurred from the fourth through the thirteenth day after the start of exposure. The squirrel monkeys were remarkable in their apparent resistance to oxygen. Their pulmonary response was considerably less than those observed in the other species. Both acute exudative and subacute proliferative lesions with fibrosis of the interstitium were seen in all three species, although these changes appeared after different time intervals and severity varied. Complete recovery seemed possible after the acute exudative stage, but marked interestitial fibrosis persisted in the animals that reached the subacute proliferative stage but survived. (*Robinson, F. F., and others: Pathology of Normobaric Oxygen Toxicity in Primates, Aerospace Med. 40: 879 (Aug.) 1969.*)