beta stimulation may be due to increased concentrations of myocardial norepinephrine. (*Gould, L., and others: Treatment of Cardiac Arrhythmias with Phentolamine—Appraisal and Reappraisal of Cardiac Therapy, Amer. Heart J. 78: 189 and 276 (Aug.) 1969.*)

ABSTRACTER'S COMMENT: Phentolamine, an alpha blocker and beta stimulator, joins the beta blockers, quinidine and various local anesthetics as a drug effective in the treatment of ventricular arrhythmias. This suggests that the therapeutic control of arrhythmias is related to something other than stimulation or depression of the autonomic nervous system.

Respiration

HYPOVENTILATION A condition resembling idiopathic alveolar hypoventilation in seven patients with impaired pulmonary function is described. The patients had chronic cor pulmonale, diminished ventilatory responses to inhaled carbon dioxide, carbon dioxide retention disproportionate to the extent of airflow obstruction, and ability to hyperventilate voluntarily and return Pa_{O_2} and Pa_{CO_2} to normal or near-normal levels. These studies suggest that the clinical spectrum of idiopathic alveolar hypoventilation can be extended to include some patients with obstructive and restrictive pulmonary disease and that respiratory insensitivity to carbon dioxide is not uncommon. (*Rhoads, G. G., and Brody, J. S.: Idiopathic Alveolar Hypoventilation: Clinical Spectrum, Ann. Intern. Med. 71: 271 (Aug.) 1969.*)

VENTILATION When pulmonary blood flow is interrupted, deleterious effects on the lungs can be minimized by maintaining alveolar ventilation. It is possible that structural lung changes following cardiac bypass may be modified by maintaining ventilation during interruption of pulmonary circulation. Ventilation is mechanically assisted for three to four hours postoperatively. A readily controllable respiratory alkalosis replaces a mixed acidosis from poor arterial oxygenation. (*Saperstein, W., and Kohari, J.: Pulmonary Ventilation during Open-heart Surgery, Surgery 66: 555 (Sept.) 1969.*)

TENSION PNEUMOTHORAX A 41-year-old woman was admitted following severe trauma which resulted in a skull fracture, patellar injury, trauma to the thorax at the level of the third rib, and a fractured arm. X-ray showed a slight pneumothorax on the right side. While the wounds were being sutured with the patient under local anesthesia, she suddenly became short of breath and sustained a cardiac arrest. After three minutes of extracorporeal cardiac massage and ventilation, the heart resumed normal function. Spontaneous respiration started after ten minutes and was rapid and shallow. Positive-pressure ventilation was extremely difficult. Reintubation did not help and a second cardiac arrest followed 20 minutes after the first. After several fruitless attempts at resuscitation, the possibility of a pneumothorax was considered. A second x-ray showed a bilateral tension pneumothorax, which was treated by needle aspiration. Within a minute, the heart strated beating again and spontaneous respiration resumed. Paradoxical respiration, due to an anterior thoracic flap, was controlled by artificial ventilation. After resuscitation, a third x-ray revealed multiple fractures of the right ribs. The patient left the hospital a few months later completely recovered. The cause of the first cardiac arrest was obscure. It occurred 30 minutes after the injection of procaine (30 ml of 1 per cent). The rapid resumption of cardiac function excluded an extensive pneumothorax at that time. The second arrest was due to a bilateral tension pneumothorax, which may have been caused by artificial ventilation, but probably resulted from trauma to the lung from rib fractures produced during the external cardiac massage. If there are ventilation problems during external cardiac massage, a pneumothorax should be considered. (*Otteni, J. C., and others: Pneumothorax Bilatéral à soupape après Massage Cardiaque Externe, Anesth. Anal. (Paris) 26: 401, 1969.*)

ATELECTASIS In a study of 56 patients undergoing superficial surgical operations and light anesthesia, 12 developed atelectasis in the postoperative period and six of these also inhaled iodized oil fluid on being asked to

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.*)