OXYGEN TOXICITY Three groups of adult mongrel dogs underwent left thoracotomy. The circumflex branch of the left coronary artery was doubly ligated and transected in each dog. Following closure of the chest, ventilation was maintained with 100 per cent O_2 for six hours in one group, with room air for six hours in a second group and with 100 per cent O_2 at three atmospheres absolute pressure (ATA) for two hours in the third group. Lungs of dogs in the first group (100 per cent O_2) were congested and edematous, with intraalveolar hemorrhage, fluid transudation and perivascular edema. Thirty per cent of dogs in this group survived for one month. In the second group (room air) the lungs were soft, without congestion, and one-month survival rate was 64 per cent. In the third group (O_2 at 3 ATA), pathologic changes in the lungs were similar to those in the first group but more pronounced. Intact dogs in a fourth group were exposed to a six-hour period of ventilation with 100 per cent O_2. These dogs exhibited no ill effects and their lungs were normal at autopsy. Exposure to 100 per cent O_2 at 3 ATA for two hours for five consecutive days produced no ill effects in a fifth group of intact dogs. It was concluded that patients with acute myocardial infarction should receive an inspired oxygen concentration sufficient to ensure a Pa_{O_2} of 100–150 mm Hg, but not more because higher levels may result in oxygen toxicity. (*Pifarré, R., and Hufnagel, C. A.: Pulmonary Lesions Associated with Oxygen Therapy for Acute Myocardial Infarction, J. Thorac. Cardiov. Surg. 56: 203 (Aug.) 1968.*)

BOHR SHIFT A modified Hartridge–Roughton rapid-reaction apparatus with an oxygen electrode was used to measure the rate of the Bohr shift in human erythrocytes. This rate agreed with the hypothesis that the transfer of hydrogen ions across the erythrocyte membrane via CO_2 exchange is the rate-limiting step. When the Bohr off-shift (acidification) was simulated by increasing P_{CO_2}, the process was predominantly limited by the rate of intracellular CO_2 hydration. Deoxygenation appeared a little faster than oxygenation, as might be expected, since the chemical kinetics are generally faster when Hb_{O_2} saturation is higher. (*Forster, R. E., and Steen, J. B.: Rate Limiting Processes in the Bohr Shift in Human Red Cells, J. Physiol. 196: 541 (June) 1968.*) ABSTRACTER'S COMMENT: The Bohr shift plays a vital role in respiratory gas exchange. These investigators used an extremely sophisticated rapid-flow technique for measuring the rate of Bohr shift under a variety of conditions. Their conclusion that the rate of the Bohr shift is normally limited by the catalyzed hydration of CO_2 is fundamental, but some of their other findings also have interesting physiologic implications. For example, the change in cell volume resulting from water movement such as may occur in capillary beds does not appear to change the rate of the Bohr shift.

ALVEOLAR–ARTERIAL OXYGEN GRADIENT Alveolar–arterial gradients for oxygen were measured in human subjects during oxygen breathing at ambient pressure and also at two and three atmospheres absolute. In these healthy subjects, the mean gradients were 17, 10 and 36 mm Hg at 1, 2, and 3 atmospheres respectively. The finding of normal AaO_{O_2} at hyperbaric levels is in disagreement with many previous studies; the source of the discrepancy is probably related to techniques for measuring P_{O_2} in blood. The polarographic oxygen electrode is capable of measuring P_{O_2} at elevated pressures, but several technical problems must be kept in mind when using this electrode. (*McDowall, D. G., Ledingham, I. McA., and Tindal, S.: Alveolar–Arterial Gradients for Oxygen at 1, 2, and 3 Atmospheres Absolute, J. Appl. Physiol. 24: 324 (March) 1968.*)

Erratum

In the Literature Brief, "Respiratory Distress" (ANESTHESIOLOGY 30: 263, 1969), the dosage recommended by Dr. Roberts should have read 1 mEq/lb, *not* 1 mg/lb.