UNILATERAL HYPOXIA In 11 of 14 subjects with various types of pulmonary disease, ventilating one lung with nitrogen during bronchospirometry caused an average decrease in pulmonary blood flow to that lung of 16 per cent in two minutes and about double that in seven minutes. The response was not altered significantly by atropine. This occurred despite a slight hypoxemia in most patients. Total ventilation increased 22 per cent. (*Lopez-Majano, V., and others: Time Factor in the Shifting of Blood Produced by Unilateral Hypoxia, Amer. Rev. Resp. Dis. 96: 1190 (Dec.) 1967.*)

UNILATERAL HYPOXIA For unilateral hypoxia (*i.e.*, of one lung) pulmonary blood flow rises and falls with the oxygen concentration of the inhaled atmosphere, the relationship being described by an S-shaped curve whose steepest slope is between 8 and 12 per cent oxygen. (*Isawa, T., and others: Effect of Oxygen Concentration in Inspired Gas Upon Pulmonary Arterial Blood Flow, Amer. Rev. Resp. Dis. 96: 1199 (Dec.) 1967.*)

OXYGEN IN EMPHYSEMA Two thirds of 58 subjects with chronic obstructive lung disease had significant increases in the dead-space-to-tidal volume ratio while breathing oxygen. This indicates worsening of the ventilation–perfusion ratio. The changes were not due to changes in ventilation such as hypoventilation during oxygen breathing. Oxygen is a potent pulmonary vasodilator; the observed changes are attributed to vasodilation in areas of lung with poor ventilation. (*Lee, J., and Read, J.: Effect of Oxygen Breathing on Distribution of Pulmonary Blood Flow in Chronic Obstructive Lung Disease, Amer. Rev. Resp. Dis. 96: 1173 (Dec.) 1967.*)

OXYGENATION There are significant increases in tissue P_{O_2} following the use of both hyperbaric oxygen and intra-arterial and retrograde intravenous hydrogen peroxide. The effect of the peroxide is delayed until the oxygen diffuses slowly into these tissues, but the effect is maintained for a longer period after discontinuation of the infusion. Hyperbaric oxygen causes an almost immediate rise in tissue P_{O_2} and, when discontinued, causes an almost immediate fall in the elevated levels. (*Ackerman, N., and Brinkley, F.: Hyperbaric Oxygen and Intravascular Hydrogen Peroxide, Surgery 63: 285 (Feb.) 1968.*)

CELLULAR OXYGEN UPTAKE Oxygen uptake is studied in yeast cells, rat liver slices, and rat liver homogenates by both polarographic and Warburg techniques. The effects that nitrogen, helium and argon, as well as just altering the oxygen partial pressure, have on this uptake are measured. The inert gases depress oxygen uptake except in the homogenized preparation. The difference in depression among the various gases is small and insignificant. Lowering oxygen tension without an inert gas does not cause depression. Since the inert gases fail to suppress oxygen uptake in the homogenate, the cell membrane is postulated as the site of their depressant action. (*Maio, D. A., and Neville, J. R.: Effect of Chemically Inert Gases on Oxygen Consumption in Living Tissues, Aerospace Med. 38: 1049 (Oct.) 1967.*)

ADRENAL EFFECTS OF OXYGEN Rats are exposed to 464 and 700 mm. Hg oxygen tension at ambient plus 7 mm. Hg pressure for one to 15 days. Urinary epinephrine and norepinephrine, serum corticosterone and corticosterone content of the adrenal gland are measured. Serum corticosterone remains normal at the lower oxygen tension, but by the third day rises dramatically in the higher oxygen group. Urinalysis shows significant increases in both compounds, lasting one to two days, with a return to normal levels at the lower oxygen tension. At the higher oxygen tension, epinephrine excretion rises but norepinephrine excretion falls, with a maximum change just prior to death. The data seem to indicate a direct adrenal-gland response in the rat to extreme oxygen environment and that changes in adrenal-gland production are of a nature to augment oxygen toxicity. (*Houlihan, R. T., Zavodni, J. J., and Cross, M. H.: Adaptation to Increased Oxygen Tension at Ambient Pressure, Aerospace Med. 38: 995 (Oct.) 1967.*)