EMPHYSEMA After intravenous injection of 250 mg. aminophylline over a three-minute period into 32 subjects with chronic obstructive pulmonary disease, arterial oxygen tension fell 4 mm. Hg or more or oxygen saturation was reduced 2 per cent or more in 20 subjects. This was thought to be due to worsening of ventilation-perfusion relationships in areas of underventilation of the lung where compensatory vasoconstriction was lessened by the aminophylline. (*Pain, M. C. F., and others: Effect of Intravenous Aminophylline on Distribution of Pulmonary Blood Flow in Obstructive Lung Disease, Amer. Rev. Resp. Dis. 95: 1005 (June) 1967.*)

HYPOXEMIA In five patients with nonspecific chronic pulmonary disease characterized in part by increased airway resistance, breathing 30 per cent oxygen in nitrogen significantly relieved the resistance. Part of the increased resistance in patients with chronic pulmonary disease and hypoxemia appears to be due to the hypoxemia. (*Astin, T. W., and others: Airway Obstruction Due to Hypoxemia in Patients with Chronic Lung Disease, Amer. Rev. Resp. Dis. 95: 567 (April) 1967.*)

PULMONARY FUNCTION In 33 patients studied soon after myocardial infarction, reduction in arterial oxygen tension breathing air, without abnormality in carbon dioxide tension, was invariably found to be due to abnormal venoarterial shunting, and in 24 cases it was due to concomitant ventilation-perfusion imbalances. Diuretics improved arterial hypoxemia by improving ventilation-perfusion relationships, but did not alter the degree of shunt. Oxygen therapy corrected the hypoxemia. (*Pain, M. C. F., and others: Disturbances in Pulmonary Function after Acute Myocardial Infarction, Brit. Med. J. 1: 591 (June) 1967.*)

GAS TENSION Pressure changes arising from counter-diffusion of gases through a sealed silicone rubber tube may be used to measure tension of gases in a mixture. When the tube initially is filled with one of the bases, and the mixture surrounds it, the pressure rise is related to the tension of the remaining gas. (*Winsey, H. S., and Folkman, J.: Silicone Rubber: Oxygen, Carbon Dioxide, and Nitrous Oxide Measurement in Gas Mixtures, Science 157: 203, 1967.*)

OXYGEN UPTAKE On-line oxygen consumption in man can be measured with a polarographic oxygen sensor and a mass gas-flow transducer. The oxygen sensor generates current directly proportional to exhaled P_{O_2} values. The flowmeter's voltage varies linearly with mass flow of exhaled air. The electrical signals are amplified, multiplied and integrated electronically, with the output representing consumed oxygen. Results obtained compare well with older methods involving collection in bags and analysis of expired air. Advantages are continuous on-line results and no restrictions on movement. (*Kissen, A. T., and McGuire, D. W.: New Approach for On-Line, Continuous Determination of Oxygen Consumption in Human Subjects, Aerospace Med. 38: 686 (July) 1967.*)

OXYGEN TOXICITY Prolonged inhalation of high concentrations of oxygen may cause closure of bronchioles or small bronchi, collapse of alveolar units and suppression of critical enzyme processes in the lung, especially those involving sulfhydryl groups. Experimental evidence suggests that continuous prolonged exposure to oxygen tensions below 250 mm. Hg (equivalent to 30 per cent at normal barometric pressure) is safe. Exposure to tensions above 400 mm. Hg (56 per cent) can produce pulmonary damage if prolonged for several days. In caring for critically-ill patients, concentration of inspired oxygen must be sufficiently high to maintain arterial oxygen tensions close to normal. However, that part of the arterial hypoxemia which is due to alveolar hypoventilation (as manifest by carbon dioxide retention) should be corrected if possible by increasing total ventilation. High-oxygen therapy should not be used as a substitute for physiotherapy, suction and bronchodilators directed at improving ventilation. Frequent blood gas determinations must be made to avoid delivering a higher oxygen concentration than that necessary to maintain normal arterial oxygen tensions. (*Editorial: Oxygen Therapy and Pulmonary Lesions, Canad. Med. Ass. J. 96: 1480 (June) 1967.*)