thesia, probably because of alveolar collapse, but is improved by an occasional deep breath. Except in very light anesthesia, spontaneous signs do not occur during general anesthesia. This state of partial alveolar collapse continues into the postoperative period, and is aggravated by the rapid, shallow respirations associated with abdominal pain. Morphine, even in doses which do not grossly depress ventilation and gas exchange, may depress the reflex urge to take occasional deep breaths. In addition to an attempt to reduce morphine dosage during the postoperative period, vigorous encouragement may help patients take a deep breath or cough. Forcible expansion of the lungs with a bag and mask, and regional anesthesia to block pain locally are additional measures to reduce pulmonary complications. Normal respiratory function should not be assumed merely on the basis of normal minute volume or carbon dioxide tension. The presence of periodic deep breaths, active or passive, is a component of normal respiratory function. (*Egbert, L. D., and Bendixen, H. H.: Effect of Morphine on Breathing Pattern, J.A.M.A. 188: 485 (May 11) 1964.*)

A-a OXYGEN DIFFERENCE Since the alveolar-arterial oxygen difference may be due to venous shunting, unequal distribution of alveolar ventilation, or diffusion limitations, measurements to determine the significance of each were carried out utilizing alveolar-arterial oxygen difference measurements during breathing of various concentrations of oxygen. Utilizing 14, 21, and 100 per cent oxygen inhalation, venous shunting was 1 to 5 per cent of the systemic blood flow and unequal distribution of alveolar ventilation contributed a shunt of an additional 3.4 per cent of the systemic blood flow. On breathing room air, the diffusion component of the alveolar-arterial oxygen was insignificant. (*Ayres, M .S., and others: Components of Alveolar-Arterial O_2 Difference in Normal Man, J. Appl. Physiol. 19: 43 (Jan.) 1964.*)

RESPIRATORY INERTANCE Total respiratory compliance, respiratory system natural frequency, and total respiratory inertance were measured in 8 normal and 14 excessively obese subjects. The mean total inertance in the normals was 0.0098 cm. of water per liter per second^2. In the obese subjects it was 0.0253 cm. of water per liter per second^2. This is a highly significant difference. There were significant inverse correlations between total respiratory compliance and body weight and between natural frequency and body weight. There was also a significant positive correlation between total respiratory inertance and body weight. A method for estimating separately the gas and tissue components of total respiratory inertance was applied to three normal and three obese subjects. In the normal subjects, tissue inertance averaged 16 per cent of the total inertance. In the excessively obese subjects, tissue inertance averaged 68 per cent of the total inertance. (*Sharp, J. T.: Total Respiratory Inertance and Its Gas and Tissue Components in Normal and Obese Men, J. Clin. Invest. 43: 503 (Mar.) 1964.*)

PULMONARY METASTASES Pulmonary compliance is reduced in all patients with extensive lymphatic and hematogenous spread. Multiple hematogenous metastases cause hypoxemia because of a shunt-like mechanism while diffuse lymphatic metastases reduce oxygenation by their effect on the gas-exchange surface of the lung. (*Emirgil, E., Zsoldos, S., and Heinemann, H. O.: Pulmonary Metastases and Pulmonary Function, Amer. J. Med. 36: 382 (Mar.) 1964.*)

TRACHEOSTOMY Effects of reduction of dead space with tracheostomy were noted in four subjects with lower airway obstruction and in two subjects with upper airway obstruction. The two groups were distinctly different in that the patients with lower airway obstruction demonstrated decrease in minute ventilation after tracheostomy. Also, alveolar ventilation decreased except when alveolar hypoventilation was present to start with. There was either no change or an increase in the arterial P_{CO_2} in subjects with lower airway obstruction. Small increases occurred in the arterial oxygen saturation with tracheostomy. (*Froeb, H. F., and others: Tracheostomy and Respiratory Dead Space in Emphysema, J. Appl. Physiol. 19: 92 (Jan.) 1964.*)