(2) CO_2 tension indicates the balance between CO_2 production (metabolism) and CO_2 elimination (breathing). It is the "blood urea" of respiratory medicine. (3) CO_2 tension indicates the alveolar oxygen tension (breathing air). (4) CO_2 tension is the respiratory variable in acid-base disturbances and in the evaluation of acid-base state. (5) Clinical experience shows that patients with chronic elevation of CO_2 are liable to develop further CO_2 retention with narcosis if oxygen or sedatives are given ill-advisedly. Conversely, patients with normal CO_2 tensions are unlikely to develop further CO_2 retention in such circumstances. (6) Patients with a high CO_2 tensions are liable to develop fluid retention and edema. (*Campbell, E. J. M.: Neurological Complications of Cardiac Surgery and Respiratory Disorders, Proc. Roy. Soc. Med. 60: 860 (Sept.) 1967.*)

RESPIRATORY DISTRESS Carbonic anhydrase is a zinc metalloenzyme that reversibly catalyzes the hydration of CO_2 to bicarbonate and hydrogen ions. Carbonic anhydrase activity and zinc concentrations were measured in blood from adults, full-term infants, premature infants without the respiratory distress syndrome, and premature infants with the syndrome. Infants exhibiting the respiratory distress syndrome had carbonic anhydrase activity averaging 5 per cent of that measured in adults, whereas normal premature and full-term infants averaged 13 and 25 per cent, respectively. Blood zinc concentrations in premature infants with the respiratory distress syndrome averaged 29 per cent of adult values but were not significantly different from those of normal infants. Thus, the reduced enzymatic activity probably was not related to a zinc deficiency. It was postulated that the carbonic anhydrase activity of diseased infants was reduced to a level where adequate CO_2 exchange in the lungs might not take place. The resultant CO_2 retention could contribute to the vicious circle thought to occur in infants with the respiratory distress syndrome: CO_2 retention > acidosis > increased pulmonary vascular resistance > shunting > decreased pulmonary blood flow > decreased pulmonary surfactant > alveolar instability > atelectasis > CO_2 retention, etc. (*Kleinman, L. I., Petering, H. G., and Sutherland, J. M.: Blood Carbonic Anhydrase Activity and Zinc Concentration in Infants with Respiratory Distress Syndrome, New Eng. J. Med. 277: 1157 (Nov.) 1967.*)

PULMONARY SURFACTANT Progressive decreases in distensibility occurred in excised rat lungs which were ventilated at greater-than-tidal volume at room temperature. The magnitude of change became greater as respiratory rate increased. Pressure-volume characteristics of excised lungs hyperventilated in a similar manner at 37° C. restored prehyperventilation pressure–volume characteristics. Incubation in a cold environment or administration of cyanide prior to excision of the lungs deterred recovery after hyperventilation. Surfactant is modified rapidly by ventilation, especially at high rates, and it is replaced by processes involving cellular metabolism. Lavage of excised lungs with detergent solution altered pressure–volume characteristics by altering surface forces. (*McClenahan, J. B., and Urtnowski, A.: Effect of Ventilation on Surfactant and its Turnover Rate, J. Appl. Physiol. 23: 215 (Aug.) 1967.*)

HYPERBARIC OXYGEN Diffusion of carbon monoxide in the lung was studied in healthy volunteers during breathing of air following breathing of 99 per cent oxygen at different ambient pressures and intervals. Following exposure to oxygen at 1 atmosphere for three hours, neither alveolar volume nor an index of the size of the pulmonary capillary vascular bed changed. Following exposure to oxygen at 3 atmospheres, alveolar volumes did not change significantly in six subjects, but decreased in one subject. Observed changes in diffusion of carbon monoxide were interpreted as indicating an increase in size of the pulmonary vascular bed in response to hyperbaric oxygen, together with slowly-developing pulmonary edema, as duration of exposure increased. (*Rosenberg, E., and MacLean, L. D.: Effect of High Oxygen Tensions on Diffusing Capacity for CO and Krogh's K, J. Appl. Physiol. 23: 11 (July) 1967.*)