lobeline and a slightly diminished response to carbon dioxide. A paradoxical increase in minute ventilation followed the administration of oxygen which was explained by a supposed sensitivity of the respiratory center to carbon dioxide with improved oxygenation. (*Rodman, T., and Close, H. P.: The Primary Hypoventilation Syndrome, Am. J. Med. 26: 808 (May) 1959.*)

HYPOVENTILATION Arterial blood gas tensions, blood volumes and oxygen cost of breathing were studied in a series of obese subjects. For the most part there was no evidence of gross lung disease as revealed by clinical history and ventilatory function measurements. Twelve of eighteen subjects who had measurements of arterial blood gas tensions had hypoxia. Four had associated hypercapnia which apparently was due to reduced tidal volume. Red cell mass per square meter of body surface was increased in both male and female subjects. Plasma volume was increased only in the female subjects. The oxygen cost of breathing was increased in all the obese subjects. It is suggested that this was due to an increase in elastic resistance of the thorax. There appeared to be a relationship between the oxygen cost of breathing and the arterial carbon dioxide tension in obese subjects. This is in accordance with the hypothesis that respiratory acidosis is an adaptive mechanism sparing oxygen for nonventilatory work, a rise in carbon dioxide tension being tolerated when the work of breathing is increased. The data also indicate that, in the obese individual, further increments in ventilation could result in a disproportionate increase in metabolic work of breathing which would be exaggerated if he developed bronchitis or other lung disease. Conversely, individuals with chronic lung disease and increased work of breathing would get into further difficulty with the development of obesity. (*Kaufman, B. J., Ferguson, M. H., and Cherniack, R. M.: Hypoventilation in Obesity, J. Clin. Invest. 38: 500 (March) 1959.*)

OXYGEN CONSUMPTION The oxygen consumption of the respiratory muscles was measured in normal and emphysematous subjects. The oxygen cost of increased ventilation was considerably higher in the emphysematous subjects and rose even further with slight increases in ventilation. Efficiency of the respiratory muscles was considerably lower in patients with emphysema than in normal subjects. The total mechanical work performed on the lung and thorax tends to be less in the emphysematous than in the normal individual at low ventilation. This might be expected since about 63 per cent of the work of breathing is performed in over-coming elastic resistance and a substantial loss of lung elasticity occurs in emphysema. The oxygen cost of breathing is four to five times greater because of the marked reduced efficiency of the respiratory muscles. Increases in ventilation result in a disproportionate increase in oxygen consumption of the respiratory muscles in emphysema. This may explain the disability present in pulmonary emphysema and the inability of the severely emphysematous patient to meet the increased energy demands of exercise and infection. (*Cherniack, R. M.: The Oxygen Consumption and Efficiency of the Respiratory Muscles in Health and Emphysema, J. Clin. Invest. 38: 494 (March) 1959.*)

PULMONARY EDEMA In this review article outlining a rational approach to the treatment of all types of pulmonary edema, emphasis is placed on relief of anoxia. Some caution regarding continuous inhalation of 60 to 100 per cent oxygen for several hours is emphasized, but the danger of pulmonary edema from hyperoxyia is remote in the treatment of patients with pulmonary edema. In addition to the conventional means of reducing circulating blood volume the use of ganglion blocking drugs is suggested. While atropine is most useful in the treatment of poisoning by cholinergic and anticholinesterase agents, it is not very useful in the treatment of pulmonary edema due to congestive heart disease. A new synthetic compound (#45–50) has been used in the treatment of edema associated with burns of the respiratory tract. (*Aviado, D. M., Jr., and Schmidt, C. F.: Physiologic Basis for the Treatment of Pulmonary Edema, J. Chronic Dis. 9: 495 (May) 1959.*)

PULMONARY EDEMA Experimental toxic lung edema in white mice was induced