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

by adrenaline or ammonium chloride. All derivatives of phenothiazine arrest progress of edema; however, their effectiveness differs somewhat. (*Raevskii, K. S.: Influence of Some Derivatives of Phenothiazine on Experimental Toxic Lung Oedema, Byull, Eksper. Biol. i Med. 43: 33 1957.*)

PULMONARY EMBOLISM In the patient with suspected pulmonary embolism, simultaneous measurement of arterial and end-tidal carbon dioxide tensions may assist in diagnosis. In the absence of pulmonary infarction, a difference of less than 5 mm. of mercury implies either no embolism or an embolus occluding less than the equivalent of a lobar branch of the pulmonary artery. In the absence of emphysema, a large difference indicates pulmonary vascular occlusion. (*Robin, E. D., and others: A Physiologic Approach to the Diagnosis of Acute Pulmonary Embolism, New England J. Med., 260: 586 (March 19) 1959.*)

HIGH ALTITUDE The concentration of lactic acid, sodium, potassium and calcium was measured in four structures of the brain 3 hours after death in anesthetized and unanesthetized dogs which had been subjected to simulated altitude of 30,000 feet. There was elevation of: (a) the sodium concentration in the corona radiata but not in the cerebral cortex or caudate nucleus; (b) the calcium concentration in cortex, corona radiata and caudate nucleus; (c) the lactic acid concentration in all the tissues. No potassium changes were seen. Effect of pentobarbital anesthesia was very slight. (*Van Fossan, D. D., and Biddulph, C.: Effects of Altitude and of Anesthesia on Brain Electrolytes and Lactic Acid, Am. J. Physiol. 196: 1063 (May) 1959.*)

HYPOTHERMIA Neither prednisolone nor hypothermia when used alone significantly decreased mortality from cerebral damage in rabbits. However, hypothermia and prednisolone used in combination decreased the mortality from the cerebral injury and increased the survival time of those animals dying. (*Raimondi, A. J., and others: The Effect of Hypothermia and Steroid Therapy on Experimental Cerebral Injury, Surg. Gynec. & Obst. 108: 333 (March) 1959.*)

GASTRIC HYPOTHERMIA Gastric digestion is retarded considerably by local gastric cooling. Inhibition of peptic activity is the primary cause of this suppression of gastric digestion. Local gastric cooling in man can be obtained by circulating a coolant (equal parts ethyl alcohol and ice-cold water) through a system of tubes and balloons in the stomach. This technique has been applied in treating nineteen patients who had unrelenting hemorrhages from the duodenum, stomach, and esophagus and who, in spite of massive replacement therapy, were in a condition too precarious for emergency operation. Prompt cessation of hemorrhage occurred in all patients, thus accomplishing either adequate treatment or a period in which restoration to normal blood volume and optimal condition for any necessary definitive surgical therapy could occur. Local gastric hypothermia is rational for situations in which the digestive activity of gastric juice may be responsible for continued erosion and hemorrhage. It should not be relied on in situations complicated by blood dyscrasias, carcinoma, and other miscellaneous disorders. (*Wangensteen, O., and others: Depressant Action of Local Gastric Hypothermia on Gastric Digestion, J. A. M. A. 169: 1601 (April 4) 1959.*)

NEW NEUROPHYSIOLOGY Based on current work of the past few years, the four basic tenets or basic concepts of neuron doctrine which arose from electrophysiological studies of the 1920's and 1930's are slowly changing: (1) that all or none impulse with after potentials as the only true form of nervous activity is changing to the idea that some neurons have two axons and can deliver two non-identical nerve impulses simultaneously in two different directions; (2) that neuron excitation spreads to all parts of the neuron as a propagated nerve impulse is changing to the idea that neuronal excitation impulses do not spread directly but prepare the neuron for firing at some given spot within the cell, *e.g.*, at base of axon; (3) that dendrites propagate impulses toward a cell body is changing to the idea that impulses may spread and exert influence on other neurons by electrotonic spread; (4) that the synapse is the only point of selection, evaluation, fatigue and facilitation