of insulin. The first measurements were also made under conditions of zero and 5 per cent $CO_2$ in air. Experimental results indicate that the $O_2$ consumption of isolated plasma-perfused lung tissue is about 0.048 ml/min/g dry weight. The lung can utilize carbohydrate almost exclusively for its energy. The lung produces lactic acid even when the lactic acid concentration in the perfusate reaches values greater than 100 mg/100 ml. Under hyperglycemic conditions, a significantly elevated R.Q. indicates a probable conversion of carbohydrate to fat within the lung. The total energy utilization of the canine lung is about 1 per cent of the total energy utilization of the entire body at rest. (*Weber, K. C., and Visscher, M. B.: Metabolism of the Isolated Canine Lung, Amer. J. Physiol. 217: 1044 (Oct.) 1969.*)

**HYPOCAPNIC VERTEBRAL ARTERY PERFUSION** Transient perfusions of hypocapnic and hypercapnic blood were made into the vertebral arteries of anesthetized dogs. During hypocapnic perfusion while the dogs were breathing air there was a 5 per cent reduction in ventilation. With perfusions of hypocapnic blood while the dogs breathed $CO_2$ a greater reduction in ventilation occurred. This was 9 per cent at 40 sec, 14 per cent at 70 sec, and 33 per cent at two minutes. After two minutes of hypercapnic perfusion, rates of recovery of ventilation were twice as fast as those observed after 8 to 10 minutes of $CO_2$ breathing. These rates were still not as rapid as those previously observed during recovery from hypercapnic perfusion of carotid bodies. These results suggest: 1) that CSF and brain tissue act as a reservoir for $CO_2$; 2) that $CO_2$ diffuses slowly out of the brain chemoreceptive tissue following stepwise decreases of $P_{CO_2}$ in the cerebral capillaries; 3) that during transient states, the $P_{CO_2}$ of brain chemoreceptive tissue is dominated by the $P_{CO_2}$ of CSF and adjacent brain tissue rather than by the $P_{CO_2}$ of cerebral capillaries. (*Dutton, R. E., and others: Respiration during Transient Perfusion of Vertebral Arteries with Hypocapnic Blood, Amer. J. Physiol. 217: 1178 (Oct.) 1969.*)

**DIAPHRAGMATIC ACTIVITY IN OBESITY** Minute ventilation, diaphragmatic activity and total chest compliance were measured in eight adults each weighing at least 300 pounds. Four had normal and four had abnormal arterial blood gas values. The diaphragmatic electromyogram was recorded with an esophageal bipolar lead. There was no significant difference between chest compliance in the two groups, but there were marked differences in diaphragmatic activity/mm change in $Pa_{CO_2}$. The increase in integrated diaphragmatic electrical activity averaged 66 units in the obese normal subjects and 17 units in the obese hypoventilation subjects. Results suggest that an incapacity to increase the activity in the respiratory muscles to levels necessary to overcome the load imposed by obesity plays a major role in the genesis of respiratory failure in obese subjects. (*Lourenco, R. V.: Diaphragm Activity in Obesity, J. Clin. Invest. 48: 1609 (Sept.) 1969.*)

**EMPHYSEMATOUS LUNGS** At autopsy, bronchograms of emphysematous lungs were made, using fine particulate lead. Distending pressures of 0, 5, 10 and 20 cm $H_2O$ were used while roentgenograms were taken. The volumes of individual centrilobular emphysematous spaces were calculated at each distending pressure from measurements made from the bronchograms and from pressure–volume curves constructed for each space. Normal lungs and lungs with centrilobular emphysema were compared by determining the volume of air that could be expressed from them at each distending pressure as a percentage of the volume contained at 20 cm $H_2O$. Centrilobular emphysematous spaces have a high residual volume, are less compliant than normal lung tissue, and are much less compliant than the emphysematous lungs which contain them. These spaces undergo little volume change within the tidal breathing range and probably add a relatively nondistensible series deadspace to the surrounding lung parenchyma. (*Hogg, J. C., and others: Elastic Properties of the Centrilobular Emphysematous Space, J. Clin. Invest. 48: 1306 (July) 1969.*)

**CHEST THERAPY** Randomly selected "poor-risk" patients were treated preoperatively and postoperatively with cessation of smoking, bronchodilator drugs, antibiotics, inhalation of humidified gases, segmental pos-

tural drainage, and physio-therapy of the chest. When compared with nontreated poor-risk patients, the treated patients had marked reductions in postoperative morbidity and mortality due to pulmonary complications. Although the incidence and the severity of pulmonary complications were somewhat greater in the treated patients than in a group of "good-risk" patients who were considerably younger, the differences were not significant. (*Stein, M., and Cassara, E. L.: Preoperative Pulmonary Evaluation and Therapy for Surgery Patients, J.A.M.A. 211: 787 (Feb.) 1970.*)

**HYPOXIA AND MALNUTRITION** Male newborn rats were exposed to 12 per cent oxygen for one to seven days, and brain weights, DNA and protein or RNA content were studied at 7, 21 or 35 days. Tissues analyzed included cerebrum, cerebellum, liver, muscle, carcass fat, and skeletal collagen. The resulting values were compared with those of tissues from normal rats and from rats malnourished for one to seven days. Hypoxia prevented brain DNA and protein content from increasing. At 35 days, hypoxic rats had reductions in total body, cerebellar and liver weights, muscle mass, numbers of muscle cells, and skeletal collagen. There was an increase in carcass fat. Cerebellar DNA and protein contents were reduced while RNA contents in the cerebrum, liver and muscle were very low. During hypoxia, cell multiplication is prevented, but subsequently there is interference with RNA production and protein synthesis, with retardation of growth. In the brain the cerebellum is damaged more than the cerebrum. The effects of hypoxia cannot be ascribed entirely to restricted food intake. (*Check, D. B., Graystone, J. E., and Rowe, R. D.: Hypoxia and Malnutrition in Newborn Rats: Effects on RNA, DNA, and Protein in Tissues, Amer. J. Physiol. 217: 642 (Sept.) 1969.*)

**CIRCULATORY RESPONSES TO HYPOXIA** The role of beta-adrenergic receptor stimulation in the circulatory responses to hypoxia was studied in dogs. Inhalation of 7 per cent oxygen increased heart rate, cardiac output and mean arterial blood pressure in unanesthetized dogs. The tachycardia and elevated cardiac output were abolished by propranolol. The circulatory responses to hypoxia could not be reproduced by intravenous infusions of isoproterenol, epinephrine, or a mixture of isoproterenol and norepinephrine. Bilateral adrenalectomy did not modify the response to hypoxia in anesthetized dogs. In dogs with cardiac denervation following cardiac autotransplantation, hypoxia produced increases in heart rate and cardiac output which were markedly reduced by propranolol, but not modified by bilateral adrenalectomy. Autotransplanted dogs following cardiac reinnervation responded to hypoxia before and after propranolol like the unanesthetized dogs. Results suggest that stimulation of cardiac beta-adrenergic receptors is a major factor in the production of tachycardia and increased cardiac output during hypoxia. In normal dogs this is the result of increased activity of cardiac sympathetic nerves, rather than circulating catecholamines. In denervated dogs, circulating catecholamines are responsible for cardiac beta-adrenergic receptor stimulation. This difference between normal and denervated dogs may be due to the absence of reflex control of the heart and to the hypersensitivity of the denervated heart to catecholamines. (*Kontos, H. A., and Lower, R. R.: Rate of Beta-adrenergic Receptors in the Circulatory Response to Hypoxia, Amer. J. Physiol. 217: 756 (Sept.) 1969.*)

**INSPIRED OXYGEN** During ventilation with pressure-cycled ventilators, it is possible to predict the inspired oxygen concentration from a standard graph, when the ventilators are driven by compressed air and oxygen is added to the inspiratory line. No significant difference between the predicted and observed inspired oxygen concentrations was found when: 1) expired minute ventilation was measured accurately; 2) the flowmeter for oxygen delivery had a scale from 0 to 6 l/min and was calibrated and used with a constant-pressure delivery system; 3) there was no leakage of oxygen from the line; and 4) the mixing volume of the inspiratory line was large. (*Lewinsohn, G. E., and others: Control of Inspired Oxygen Concentration in Pressure-cycled Ventilators, J.A.M.A. 211: 961 (Feb.) 1970.*)