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. (*Cheek, 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.*)