an impediment for any one molecule by analogy with any other. Most evidence from dye studies has been discredited. (*Dobbing, J.: Blood Brain Barrier, Physiol. Rev. 41: 130 (Jan.) 1961.*)

PULMONARY CIRCULATION In normal pulmonary circulation vasomotor activity is slight when compared to mechanical influences. The adjustment of alveolar perfusion to alveolar ventilation is good when the patient is supine; but in the lateral or standing position, the upper lung becomes hyperventilated with respect to perfuson and the lower lung becomes overperfused. This is manifested by higher respiratory exchange ratios and by lower oxygen uptakes in the upper lobes. (*Fishman, A. P.: Respiratory Gases in Regulation of Pulmonary Circulation, Physiol. Rev. 41: 214 (Jan.) 1961.*)

PULMONARY ANATOMY Three distinct subgross lung types are recognized: type I is represented by the cow, pig, and lamb; type II by the dog, cat, and monkey; type III by the horse and man. Great caution should be exercised in the choice of an experimental animal for pulmonary studies if the results are to be applied to man. Known interspecies anatomical differences, which at times can be severe, and known interspecies differences in susceptibility to disease not only reinforce this concept but could cause the failure of any experiment which neglects them. (*McLaughlin, R. F., and others: Subgross Pulmonary Anatomy in Various Mammals and Man, J. A. M. A. 175: 694 (Feb. 25) 1961.*)

VENTILATION CONTROL A large volume of evidence demonstrates conclusively that carbon dioxide is a powerful respiratory stimulant, and there is indisputable proof that the arterial tension of oxygen and the *p*H of the arterial blood do have some effect on respiration. The role of these three classical stimuli in the control of pulmonary ventilation has been investigated, and it has been found that they are not an adequate explanation either for the hyperpnea of muscular exercise in normal and abnormal subjects, or for the hyperventilation observed in patients suffering from cardiopulmonary disease. They apparently play a minor role, if any, in the control of pulmonary ventilation under normal conditions. Studies demonstrate that the arterial carbon dioxide tension is determined by the activity of the respiratory center. There is no correlation between carbon dioxide tension and pulmonary ventilation at various levels of physical exercise. Patients who hyperventilate have a low carbon dioxide tension, and these subjects are less sensitive to inspired carbon dioxide than is the normal, despite the low tension. Both normal and abnormal subjects show a decreasing sensitivity to carbon dioxide as the exercise stimulus is increased. These observations are not compatible with the hypothesis that carbon dioxide is an effective regulator of the respiratory response to muscular exercise. A possible change in the *p*H of the arterial blood cannot be invoked to explain the inadequacy of carbon dioxide. The tension of arterial oxygen can be dismissed as a factor in normal subjects, and investigation suggests that it is of little importance as a cause of hyperventilation in patients with cardiopulmonary disease. (*Sinnott, J.: Control of Pulmonary Ventilation in Physiological Hyperpnea, Canad. Med. Ass. J. 84: 471 (Mar. 4) 1961.*)

PARADOXICAL RESPIRATION From a discussion of the theory of paradoxical respiration following thoracoplasty ("pendelluft") and from experimental studies of ventilation, P_{CO_2} measurements in both main bronchi and intrapleural pressures, it is demonstrated that this condition does not exist. True "pendelluft" can exist in the presence of open hemithorax and an anesthesia bag distended to a pressure greater than atmospheric in a patient who is breathing spontaneously. The concept of "pendelluft" in the presence of a closed chest should be abandoned. (*Maloney, J. V., and others: Paradoxical Respiration and "Pendelluft," J. Thor. Cardiov. Surg. 41: 291 (Mar.) 1961.*)

NITROGEN NARCOSIS The effect of increased air pressure was studied on trained divers and submarine escape instructors. Electroencephalograms were taken with subjects inside a recompression chamber. The subject was given arithmetic problems to solve which produced alpha blocking. Air pressure was increased up to 10 atmospheres. Alpha blocking was abolished, and there was an increased incidence of mistakes in solving problems. At 10 atmospheres, symptoms of nitrogen narcosis developed. When oxygen and helium were substituted for air, at the same pressures, the electroencephalogram activity was restored, and there was increased mental clarity. Nitrogen is therefore implicated as the agent responsible for intellectual changes at increased atmospheric pressure. (*Bennett, P. B., and Glass, A.: Electroencephalographic and Other Changes Induced by High Partial Pressures of Nitrogen, Electroenceph. Clin. Neurophysiol. 13: 90 (Feb.) 1961.*)

HIGH PRESSURE OPERATING ROOM A high pressure operating room was constructed, 4 by 6 meters in area, permitting pressures of three atmospheres. A pressure of three atmospheres can be achieved in 12 minutes. This room has been used for saturating blood plasma, intercellular fluid and cells with a high level of physically dissolved oxygen. It has been used for prolonging the period of circulatory arrest under hypothermia; for substituting plasma for hemoglobin as a means of oxygen transport; for preventing ventricular fibrillation under deep hypothermia; and for the treatment of certain anaerobic infections. (*Boerema, I.: Operating Room with High Atmospheric Pressure, Surgery 49: 291 (Mar.) 1961.*)

JOSEPH BARCROFT Barcroft's first high altitude expedition to Teneriffe at 11,000 feet in the Canary Islands was designed to investigate the effect of altitude upon the oxygen dissociation curve of man. Early in this century the mechanism of adjustment to high altitude or low oxygen pressure was unsettled. J. S. Haldane firmly believed that acclimatization was associated with active secretion of oxygen by the alveolar epithelium of the lungs. The studies at Teneriffe, followed by those at 15,000 feet at Monte Rosa supplied additional evidence against the oxygen secretion theory. The first World War diverted Barcroft's physiological interests into the study and treatment of poisoning from war gases. On the basis of chamber experiments, attention was directed to the use of oxygen in treatment. (*Editorial: Joseph Barcroft, The Oxygen Physiologist, J. A. M. A. 175: 802 (Mar. 4) 1961.*)

HYPOTHERMIA Thermal gradients were recorded throughout the dogs' heart during the production and reversion of profound hypothermia with extracorporeal perfusion. Temperature variations as much as 10 C. were noted. It is concluded that thermal gradients alone are not responsible for hypothermic ventricular fibrillation. (*Fisher, B., and Fedor, E. J.: Cardiac Temperature Gradients During Profound Hypothermia with Extracorporeal Perfusion, Proc. Soc. Exp. Biol. Med. 106: 275 (Feb.) 1961.*)

HYPOTHERMIA The pressor response to injection of epinephrine and norepinephrine is potentiated in the anesthetized dog at blood temperatures of 27–28 C. The increased response to norepinephrine is greater than to epinephrine. The hypothermic dog retains reflex activity as indicated by the respiratory and circulatory responses to bilateral cardiac occlusion and hypoxic hypoxia. The magnitude of such a response, however, is less than that seen in a dog at normal body temperature. (*Salzano, J., and Hall, F. G.: Effect of Hypothermia on Reflex Activity in the Anesthetized Dog, Proc. Soc. Exp. Biol. Med. 106: 199 (Jan.) 1961.*)

ATROPINE TOXICITY A systemic reaction, primarily involving the central nervous system, is reported in 5 patients who received topical atropine to the eyes. All the cases occurred in patients who were being prepared for refraction studies following the administration of atropine at frequent intervals for a three day period prior to the refraction. Signs and symptoms may appear minutes to hours after the beginning of the use of eye drops. In none of these cases did excessive