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