tion *in situ*, care being taken not to deform or collapse the lungs in any manner. Alveolar depth-width averages ranged from 38.69 microns in the mouse to 166.11 microns in man. Alveolar sizes ranged in an ascending order from the mouse through the baboon, dog, goat, guinea pig, monkey, rabbit and cat to man. The average volume of alveoli in man was ten times that in the dog. (*Hartcroft, W. S.: Size of Pulmonic Alveoli of Common Laboratory Animals and Man, Amer. Rev. Resp. Dis. 81: 734 (May) 1960.*)

ALVEOLAR VESSELS Pulmonic capillaries are 12 to 14 microns in diameter. Arterioles up to three hundred microns in diameter may have capillary branches arising at right angles as well as terminal branches. An alveolus may be supplied by more than one arteriole; several arterioles may supply one alveolus; and one capillary may traverse several alveoli. Capillary anastomoses occur between arterioles. In these vessels the flow may be linear, negatively pulsatile or positively pulsatile or occluded by increased intratracheal pressures. (*Knisely, W. H.: In Vivo Architecture of Blood Vessels Supplying and Draining Alveoli, Amer. Rev. Resp. Dis. 81: 735 (May) 1960.*)

ALVEOLAR WALL Thin sections of human lung examined by electron microscopy showed the capillary endothelium to be continuous with discrete interlocking cellular junctions. The basement membrane was homogenous and was interposed between endothelium and a continuous cytoplasmic syncytium that lined the capillary on the air side. This layer contained large septal cells which protruded into the air space. (*Baker, R. F., and Loosli, C. G.: Morphology of Alveolar Wall in Human Lung, Amer. Rev. Resp. Dis. 81: 735 (May) 1960.*)

ALVEOLAR PROTEINOSIS Pulmonary alveolar proteinosis emerges as a disease with definite pathophysiological features. Pulmonary function studies show no impairment of ventilatory ability, obstruction to air flow, and moderate decrease in vital capacity. There is a significant loss of functioning lung volume. Blood gas studies indicate pulmonary alveolar block and venous admixture in arterial blood as the causative agents of pulmonary insufficiency and variable degrees of cyanosis. Histologically there is a relative lack of cellular infiltration or fibrosis in interalveolar septa, and capillary architecture appears morphologically and functionally normal. Alveolar septal cells are increased in size and number, and may be cause of impaired gas diffusion. Large groups of alveolar spaces are filled with a proteinaceous material and explain the cause of physiologic shunting. Diagnosis is made by clinical course, pulmonary function findings, and lung biopsy. Clinical improvement of reported cases is noted with expectorant therapy, inhalation of proteolytic enzymes in form of aerosols, and symptomatic treatment. Steroids are at present contraindicated. Mortality is 30 per cent of reported cases. Exacerbations are common and follow-up lung biopsy is desirable. (*Fraimow, W., Cathcart, R. T., and Taylor, R. C.: Physiologic and Clinical Aspects of Pulmonary Alveolar Proteinosis, Ann. Int. Med. 52: 1177 (June) 1960.*)

THAM THAM was found to prevent the hyperventilation and acidosis which normally occurs while breathing 3 per cent carbon dioxide. A change occurs in the respiratory center in alkalosis whereby it responds primarily to an increase in arterial *p*H instead of an elevation of P_{CO_2}. The end result is the prevention of further alkalosis developing from hyperventilation. (*Brinkman, G. L.: The Use of THAM to Prevent Hyperventilation and Acidosis While Breathing Carbon Dioxide, Amer. J. Med. Sc. 239: 72 (June) 1960.*)

PULMONARY FUNCTION Incremental changes in total mechanical work are a more accurate gauge of the respiratory response to carbon dioxide than are increments of ventilation or oxygen consumption. The response of the respiratory center and muscles of patients with emphysema, when measured in terms of total mechanical work done, is lower than that of normal subjects. In emphysematous subjects with carbon dioxide retention at rest the response is less than that of patients who are normocapnic. (*Brodovsky, D., MacDonell, J. A., and Cherniack, R. M.: Respiratory Response to Carbon Dioxide in Health and Em-*