tory minute volume, tachycardia, increase in cardiac output and reduction in total peripheral vascular resistance. When the carotid body perfusate was changed from hypoxic recipient blood to oxygenated donor blood, while the recipient animal continued to breathe the low-oxygen mixture, the result was a reduction in respiratory minute volume, further increase in heart rate and cardiac output and decrease in total peripheral resistance. When hypoxic blood perfusion of the carotid body was reestablished these effects were reversed. These results indicated that the cardiovascular effects of systemic hypoxia cannot be attributed to stimulation of chemoreceptors. The mechanism responsible for the cardio-vascular effects observed in systemic hypoxia remain obscure. (*Daly, M. D., and Scott, M. J.: Role of Chemoreceptors in Cardiovascular Responses to Systemic Hypoxia in Dog, J. Physiol. 154: 6P (Nov.) 1960.*)

PULMONARY CAPILLARY VOLUME Hypercarbia produced by inhalation of carbon dioxide-enriched gas mixtures caused an increase in the diffusing capacity of the lung (D_L) of 5 per cent when 10 per cent carbon dioxide was added to the mixture used in determining D_L and of 24 per cent when 7.5 per cent carbon dioxide was breathed for ten minutes before the determination of D_L in normal resting subjects. The increase in D_L was caused by increased pulmonary capillary blood volume during hypercarbia which was probably not dependent on systemic respiratory or circulatory changes. (*Rankin, J., McNeill, R. S., and Forster, R. E.: Influence of Increased Alvolar CO_2 Tension on Pulmonary Diffusing Capacity for CO_2 in Man, J. Appl. Physiol. 15: 543 (July) 1960.*)

CARBON DIOXIDE NARCOSIS Thirty-five emphysematous patients who showed decreased ventilation and increased carbon dioxide retention when breathing 100 per cent oxygen showed a marked increase in ventilation and decreased partial pressure of carbon dioxide when the oxygen was given by intermittent positive pressure. In all but two patients the initially elevated carbon dioxide partial pressures were reduced. Bronchodilators were not used. (*Fraimow, W. F., Cathcart, R. T., and Goodman, E.: Use of Intermittent Positive Pressure Breathing in Prevention of Carbon Dioxide Narcosis Associated with Oxygen Therapy, Amer. Rev. Resp. Dis. 81: 815 (June) 1960.*)

AEROSOL RETENTION The retention in the human lung of aerosols with a mean particle size of 0.2 to 0.5 micron was measured with various types of respiration varying in rate from 7 to 20 per minute and in tidal volume from 600 to 2,000 ml. Retention of particles was higher than 60 per cent in all subjects for all types of respiration. It was lowest with a small tidal air and increased with the tidal air to a retention between 80 and 90 per cent. Breath-holding in inspiration will often cause retention to exceed 90 per cent. There was no spectacular difference between aerosols of different particle sizes. Although it is open to question in which part of the respiratory tract the retention occurred, it is likely that these small particles were retained in the lower part of the tract. (*Herxheimer, H., and Stresemann, E.: Retention of Wet Aerosols in Human Lung, J. Physiol. 154: 9P (Nov.) 1960.*)

AEROSOL RETENTION Retention of inhaled particles in the respiratory tract is influenced by (1) inertial impaction which tends to deposit particles of larger size in the upper portions of the airway and lung, the inertial effect being directly proportional to the density and the square of the diameter, (2) sedimentation which is governed by the same influences and which causes large particles reaching the lungs to gravitate to their deeper portions, and (3) Brownian motion which keeps very small particles in motion and aids in their removal from the alveolar sacs by diffusion. The most effective size for retention in alveoli is from 0.6 to 2.4 microns. Alveolar retention is minimal from 0.4 to 0.6 micron. Particles greater than 7 microns are 90 per cent retained in the lung. Particles smaller than 0.6 micron fail to deposit in terminal bronchi while particles larger than 20 microns fail to reach the respiratory bronchioles and particles larger than 6 microns fail to reach the alveolar ducts. Respiratory rate and depth which affect residence time of par-

ticles in the respiratory tree also markedly affect retention. (*Mitchell, R. I.: Retention of Aerosol Particles in Respiratory Tract: Review, Amer. Rev. Resp. Dis. 82: 627 (Nov.) 1960.*)

DIFFUSING CAPACITY OF LUNG The breath holding diffusing capacity and breath holding lung volume of patients two months after thoracic surgery were decreased proportionally in patients who underwent pneumonectomy while in patients who underwent lobectomy, wedge resection or thoracoplasty there was a greater degree of depression of diffusing capacity with subsequent return toward normal. Probably there would have been an even more disproportionate decrease immediately after operation. This disproportionate functional change is correlated with trauma to the remaining lung during surgery and is due either to altered hemodynamics or to acute changes in the alveolar capillary membrane in the traumatized lung. Similar changes with decreases in diffusing capacity of up to 50 per cent may follow open heart surgery. (*Dietiker, F., Lester, W., and Burrows, B.: Effects of Thoracic Surgery on Pulmonary Diffusing Capacity, Amer. Rev. Resp. Dis. 81: 830 (June) 1960.*)

HELIUM THERAPY Ten patients with severe emphysema while breathing a mixture of 80 per cent helium and 20 per cent oxygen showed a 20 per cent decrease in pulmonary resistance of the value obtained with air. There were no significant decreases in static or dynamic compliance of the lungs, functional residual capacity or static transpulmonary pressures. Though no measures of arterial carbon dioxide tension, oxygen consumption or carbon dioxide production were done, the change in resistance alone is therapeutically significant for short term therapy of such patients. (*Grapé, B., Channin, E., and Tyler, J. H.: Effect of Helium and Oxygen Mixtures on Pulmonary Resistances in Emphysema, Amer. Rev. Resp. Dis. 81: 823 (June) 1960.*)

ALVEOLAR RECRUITMENT The deflation pressure volume curve is different from the inflation pressure volume curve since the former represents only the elastic behavior of the elements which were inflated up to a given pressure or volume while the latter represents the elastic behaviour plus the added pressure necessary to open additional alveoli which have closed. The lung has different populations of alveoli which are recruited at different opening pressures. Thus an increase in inflation produces changes in the shape of the pressure volume curve which are eliminated in the next reinflation to the same point in a subsequent immediate reinflation and subsequent serial reinflations. The extent of alveolar closures during tidal respiration between two large volume inflations depends primarily on the number of tidal inflations rather than the length of the interval. The liability of an alveolus to closure probably depends on its compliance and the conductance of its associated airway. (*Bernstein, L.: Indications of Quantal Behaviour in Inflation and Deflation of Rabbit Lungs, Amer. Rev. Resp. Dis. 81: 744 (May) 1960.*)

SURFACE TENSION OF LUNG A mucoprotein film, probably monomolecular, covers the inner surface of the lung and can lower surface tension below 10 dynes/cm. In effect it increases the coefficient of elasticity of the surface and stabilizes the alveolar structure. The film can be removed and since it reforms at finite speed the minimal volume can be varied experimentally to between 5–50 per cent of the total lung volume. Modifications of this film, lowering surface tension in the alveolar structure or raising surface tension in the distal air passages, may be an important mechanism in the production of air trapping. (*Clements, J. A.: Effects of Intrinsic Surface Active Material on Mechanical Properties of Lungs, with Special Reference to Stability of Alveolar Structure, Amer. Rev. Resp. Dis. 81: 742 (May) 1960.*)

PULMONARY FUNCTION Studies of static pressure-volume characteristics of lungs in normal males indicated a decreasing vital capacity and an increasing residual volume but no change in the slope or position of the pressure-volume curve with advancing age. Older subjects were not able to change transpulmonary pressure between residual volume and total lung volume to the same extent as