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-