fifth loaded breath. This effect was greater with larger loads and was greater in the conscious subjects. The end-inspiratory muscle tension was increased in the first loaded breath in most subjects. The end-expiratory lung volume decreased during the loading, particularly with the higher loads, but the decrease did not exceed 200 ml. The explanation favored was a reflex action, probably a somatic reflex not necessarily involving afferents from the lungs. (*Campbell, E. J. M., Dinnick, O. P., and Howell, J. B. L.: Immediate Effects of Elastic Loads on Breathing of Man, J. Physiol. 156: 260 (Apr.) 1961.*)

**RESPIRATORY ACIDOSIS** Balance studies in dogs exposed to 11 to 13 per cent carbon dioxide for periods of 6 to 15 days produced a consistent pattern. There was a sharp rise in plasma bicarbonate during the first day and a subsequent slower rise over the next 5 or 6 days. When approximately one-half of the total rise in plasma bicarbonate occurred, there was little or no increase in renal acid excretion, indicating that tissue buffers played a major role in the initial defense of the extracellular *p*H. Subsequently, the increment in net acid excretion was far in excess of the amount required to account for the estimated increase in extracellular bicarbonate. Plasma chloride concentration varied in a virtually reciprocal fashion with the plasma bicarbonate. On the first day in the carbon dioxide atmosphere, there was a marked increase in the excretion of potassium and phosphorus and a variable sodium diuresis. Subsequently, both sodium and potassium excretion returned toward or to the control level. At the end of the study there remained a slight deficit of sodium, but after correction of changes in nitrogen balance, there was no evidence of potassium deficiency. (*Polak, A., and others: Effects of Chronic Hypercapnia on Electrolyte and Acid-Base Equilibrium. I. Adaptation, J. Clin. Invest. 40: 1223 (July) 1961.*)

**PULMONARY FUNCTION** Total lung diffusing capacity, diffusing capacity of the pulmonary membrane, and pulmonary capillary blood volume were studied in 34 patients, mostly children with congenital heart disease. Those with increased pulmonary blood flow or increased pulmonary wedge pressure, or both, tended to have a significant increase in diffusing capacities and pulmonary capillary blood volume. Patients with normal or decreased pulmonary blood flow had normal or slightly decreased diffusing capacities and capillary blood volume. When the patients with increased pulmonary blood flow had surgical correction of their malformation, the diffusing capacities and capillary volumes usually returned to normal. In one patient with a left to right shunt and a high pulmonary capillary blood volume, exercise studies suggested that these capillaries may increase in volume still further. (*Bacci, G., and Cook, C. D.: Studies of Respiratory Physiology in Children. VI. Lung Diffusing Capacity, Diffusing Capacity of Pulmonary Membrane and Pulmonary Capillary Blood Volume in Congenital Heart Disease, Clin. Invest. 40: 1431 (Aug.) 1961.*)

**AEROSOL DEPOSITION** Aerosols of 0.5 micron diameter have the least tendency to deposit in the respiratory tract, deposition increasing with increasing and decreasing size. About 10 per cent of 0.5 micron particles is deposited, although they may be retained for a number of respirations. Particles of this size tend to follow air currents and their motion is little affected by molecular bombardment. Gas molecules within alveoli have a mean free path more than enough to carry them to the respiratory surface while submicron particles tend not to cross the virtual interface between tidal volume and functional residual air. The presence of this interface across which the concentration gradient of aerosols falls abruptly can be demonstrated experimentally. (*Nelson, N.: Intrapulmonary Movement of Aerosols, Amer. Rev. Resp. Dis. 83: 415 (Apr.) 1961.*)

**SMOKING** Mild bronchoconstriction, insufficient to cause symptoms, occurs immediately in most individuals after inhalation of cigarette smoke, lasts 10 to 80 minutes, and reoccurs with a second cigarette. In young, healthy males after a one hour period of abstinence from smoking, there was no significant difference in airway conductance/thoracic gas volume ratio between non-smokers and heavy smokers. This may not be true, however, for individuals who have smoked for 20 years or

longer. Diminished airway conductance following the inhalation of cigarette smoke most probably represents reflexely mediated bronchoconstriction initiated by submicroscopic particulate matter in the smoke. It is not related to nicotine or volatile substances in the smoke. (*Nadel, J. A., and Comroe, J. H.: Acute Effects of Inhalation of Cigarette Smoke on Airway Conductance, J. Appl. Physiol. 16: 713 (July) 1961.*)

**NITROUS OXIDE EXCRETION** The excretion of nitrous oxide from the body follows an exponential curve which can be resolved into three exponential components. After two minutes of air breathing end-expiratory nitrous oxide concentration fell from 74 to 25 per cent and at 10 minutes it reached 10 per cent. Values for the rate of nitrous oxide elimination agreed closely with values for the rate of nitrous oxide uptake calculated from other data. (*Frumin, M. J., Salanitre, E., and Rackow, H.: Excretion of Nitrous Oxide in Anesthetized Man, J. Appl. Physiol. 16: 720 (July) 1961.*)

**NITROUS OXIDE EXCRETION** Arterial hypoxemia which may occur during the transition from breathing nitrous oxide-oxygen mixtures to air breathing has been designated "diffusion anoxia." The mechanism of this hypoxemia was postulated to be dilution of alveolar oxygen by nitrous oxide entering the alveoli from the blood. The present study demonstrates that during air breathing following nitrous oxide anesthesia dilution of alveolar carbon dioxide also occurs. The resultant lower alveolar and arterial carbon dioxide tensions, acting on the respiratory center, produce a diminished alveolar ventilation. This may further contribute to the hypoxemia occurring under these conditions. (*Rackow, H. Salanitre, E., and Frumin, M. J.: Dilution of Alveolar Gases during Nitrous Oxide Excretion in Man, J. Appl. Physiol. 16: 723 (July) 1961.*)

**GAS FLOW** Pulsatile gas flow in lobar and segmental bronchi synchronous with the heart beat was measured during bronchoscopy in human subjects. Pulsatile gas flow occurred during inspiration and exhalation. Volume flow rates up to 2.5 liters per minute and displaced volumes up to 5 ml. were recorded. This phenomenon is probably caused by both alterations of intrathoracic pressure with changing heart size and mechanical compression of various parts of the lung by the beating heart. Pulsatile gas flow synchronous with the cardiac cycle facilitates mixing of dead space gas with alveolar gas and may explain the maintenance of adequate arterial oxygenation during gross hypoventilation with oxygen-enriched inspired mixtures. (*West, J. B., and Hugh-Jones, P.: Pulsatile Gas Flow in Bronchi Caused by Heart Beat, J. Appl. Physiol. 16: 697 (July) 1961.*)

**PULMONARY HEMODYNAMICS** During positive pressure mechanical artificial ventilation, cardiac output fell significantly from levels measured during spontaneous breathing in anesthetized dogs. Absolute pulmonary arterial and venous pressure rose during positive pressure breathing but the respective transmural pressures did not change. Pulmonary vascular resistance did not differ, therefore, between the two types of respiration. The cause of decreased cardiac output during positive pressure breathing in closed-chest dogs was diminished venous return which was related to increased intrathoracic pressure. (*Linde, L. M., Simmons, D. H., and Ellman, E. L.: Pulmonary Hemodynamics during Positive Pressure Breathing, J. Appl. Physiol. 16: 644 (July) 1961.*)

**PULMONARY EMBOLISM** Pulmonary embolism was present in 20.3 per cent of 468 necropsies after injury and in 5.5 per cent of 163 after burns. All patients with pulmonary embolism had deep vein thrombosis. The highest frequency of embolism at necropsy (46–60 per cent) was in patients dying after a fractured femur or tibia. This is probably because these were elderly patients who survived long enough to develop deep venous thrombosis. Nearly half of the cases of pulmonary embolism occurred within two weeks of injury. The risk of fatal embolism with diagnosable deep venous thrombosis was about 25 per cent, but about 10 per cent in patients with silent thrombosis. Half the fatal cases of pulmonary embolism were preceeded by no clinical warning signs. Prophylaxis with anti-