7. Saidman LJ, Eger EI II, Munson ES, et al: Minimum alveolar concentrations of methoxyflurane, halothane, ether and cyclopropane in man: Correlation with theories of anesthesia. ANESTHESIOLOGY 28:994–1002, 1967
8. Eger EI II, Shargel R: The solubility of methoxyflurane in human blood and tissue homogenates. ANESTHESIOLOGY 24:625–627, 1963
9. Statement regarding the role of methoxyflurane in the production of renal dysfunction. Committee on Anesthesia, National Academy of Sciences–National Research Council. ANESTHESIOLOGY 34:505–509, 1971
10. Bond AM, Murray MM: Kidney function and structure in chronic fluorosis. Br J Exp Pathol 33:168–176, 1952
11. Taylor JM, Scott JK, Maynard EA, et al: Toxic effects of fluoride on the rat kidney. 1. Acute injury from single large doses. Toxicol Appl Pharmacol 3:278–289, 1961
12. Holaday DA, Rudofsky S, Treuhaft PS: The metabolic degradation of methoxyflurane in man. ANESTHESIOLOGY 33:579–593, 1970
13. Taves DR, Fry BW, Freeman RB et al: Toxicity following methoxyflurane anesthesia. II. Fluoride concentration in nephrotoxicity. JAMA 214:91–95, 1970
14. Sawyer DC, Eger EI II, Bahlman SH, et al: Concentration dependence of hepatic halothane metabolism. ANESTHESIOLOGY 34:230–235, 1971
15. Cohen EN: Metabolism of halothane-2¹⁴C in the mouse. ANESTHESIOLOGY 31:560–565, 1969
16. Cirksena WJ, Bastian RC, Malloy JP, et al: Use of mannitol in exogenous and endogenous intoxications. N Engl J Med 270:161–166, 1964
17. Barry KG, Mazze RI, Malloy JP: Therapy of Acute Renal Failure in Man. Mannitol Symposium, Walter Reed Army Institute of Research, Washington, D. C., February 3, 1962, pp 23–24

Respiration

HUMAN ALVEOLAR MACROPHAGE Although the alveolar macrophage has been implicated as the most vital mechanism for protecting the lungs from damage by microorganisms, very few studies of the function of this cell have been published. Human alveolar macrophages were lavaged from surgically resected lungs and also from lungs of normal subjects. Cells that had been purified by adherence to glass were maintained by tissue culture for as long as 54 days. After three to four weeks *in vitro*, transformation into multinucleated giant cells was apparent, at which time the cells had more than 30 times the phagocytic capacity than after one day *in vitro*. Phagocytosis of heat-killed *Candida albicans* was inhibited by: iodoacetate, sodium fluoride, potassium cyanide, and low partial pressures of oxygen; ancillary evidence suggests that these cells need both oxidative and glycolytic energy sources for optimal particle ingestion. Alveolar macrophages killed *Listeria monocytogenes*, as did monocyte-derived macrophages in a similarly efficient manner, but neutrophils were more efficient than either. Clearing of bacteria is probably not dependent upon myeloperoxidase in the monocyte-derived macrophage or in the alveolar macrophage, since histochemical staining for peroxidase was negative. During four hours of observation, *C. albicans* blastospores were killed by neutrophils and monocytes containing myeloperoxidase, but not by human alveolar macrophages. Large cells with supernormal phagocytic capacity were recovered from patients who had acute pneumonia of infectious origin. This suggests that disease alter macrophage function. Human alveolar macrophages are unique phagocytes, in that they depend on an environmental P_{O_2} greater than 25 torr for maximal activity. A P_{CO_2} as high as 70 torr did not alter this function when the pH was constant. These data suggest that patients with chronic bronchitis or atelectasis may have suboptimal macrophage action in areas of the lung where P_{O_2} is abnormally low. Since bronchial clearance of particulate matter is impaired and the lower respiratory tract is frequently infected, defense of the lungs by macrophages assumes an important role in the clinical course of chronic infection. (*Cohen, A. B., and Cline, M. J.: The Human Alveolar Macrophage: Isolation, Cultivation in Vitro, and Studies of Morphologic and Functional Characteristics, J. Clin. Invest. 50:1390, 1971.*)