organ already damaged is open to other onslaughts. One can only speculate on the mechanism or mechanisms which are activated by inhaled inorganic dusts such as silica and asbestos that allows malignant transformation to occur. Does the disturbed architecture of the lung caused by fibrosis allow carcinogens to be sequestered in the bronchial tree? Is the alveolar macrophage in some way disturbed? Is the ciliary transport mechanism interrupted? Does the inorganic material absorb carcinogens and carry them into the lung? Are immune defense mechanisms altered? And on and on the speculation might go. It would be more rewarding to focus study on the host rather than the agent asking, why do not all people with equal exposures develop anthracosilicosis or asbestosis or upon that a malignancy. The elucidation of predisposing host factors seems to be a most appropriate and promising endeavor. To paraphrase Shakespeare, “the fault lies not in the stars but in thee.”

Richard A. Delcomte, M.D.
Rochester, Minnesota

Pulmonary Disease Due To Endogenous Aspiration

Development of pulmonary edema, bronchiolitis, bronchitis and pneumonia following general anesthesia was attributed first by Mendelson (Am J Obst Gynec 52:191, 1948) to aspiration of gastric contents. Since then others published confirmatory clinical and experimental observations and the entity has become known as Mendelson's syndrome. Blitt et al (Anesth Analg 49:707, 1970) observed silent gastric regurgitation in 7.8 percent of 900 surgical patients under general anesthesia. Of this group 8.6 percent had aspirated gastric contents. The assumption has been that pulmonary pathologic changes and consequent clinical manifestations, such as dyspnea, cyanosis, tachycardia and shock resulted from depression of the protective laryngeal reflex. It is known that the lung is endowed with considerable resorptive and absorptive capabilities. Experimental studies demonstrated absorption of 5 percent glucose solution from the lung with subsequent rise in the blood sugar. Also, 50 percent of water introduced into the lungs of guinea pigs disappeared in 9 minutes and more than 50 percent of hypertonic sodium chloride solution in 17 minutes. In Mendelson's syndrome also there is a failure of the defense mechanism of the lung, due to general anesthesia. In addition to cough this protective potential is comprised of normal respiratory and circulatory functions, adequate alveolar-capillary gas diffusion, normally functioning alveolar macrophages, sustained escalator-like motion of the bronchial mucous blanket, and bronchial peristalsis. Impairment of some of these functions may occur in shock. In such instances and after major surgical intervention, Kim et al (Surg Gynec Obstet 133:017, 1971) observed that there was an early increase in pulmonary vascular resistance, associated with pulmonary dysfunction. Pronounced reduction in arterial Po2 might not be corrected by inhalation of 100 percent oxygen in these cases because of increased venoarterial shunting, perfusion of unventilated areas and insufficient perfusion of ventilated alveoli. In marked postoperative hypoxemia generalized metabolic decompensation may develop. It is apropos to refer to the work of Winnie et al (Anesth Analg 50:1043, 1971). They showed that postoperative hypoxemia could be effectively reversed by inducing “pharmacologic sigh” (hypocapnic hyperventilation) with doxapram, a respiratory and central nervous system stimulant, administered postoperatively. Too, impairment of laryngeal reflexes and of the defense mechanism of the lung may be the sequel of alcohol intoxication, heavy doses of barbiturates, central nervous system depressants, sedatives and narcotic analgesics. In such instances aspiration of pathogens from the nasopharynx and sinuses may be the source of pulmonary disease. There is need for optimal finesse in adjusting anesthetics, analgesic, hypnotic and tranquilizing drugs so as to maintain their efficacy without jeopardizing homeostatic potentials of the air passages and the lung.

Andrew L. Banyai, M.D.