- Shulman K: Small artery and vein pressures in the subarachnoid space of the dog. J Surg Res 5:56-61, 1965
- 16. Welch K, Friedman V: The cerebrospinal fluid valves. Brain 83:454-469, 1960
- Langfitt TW: Increased intracranial pressure. Clin Neurosurg 16:436–471, 1969
- Rosomoff HL: Effect of hypothermia and hypertonic urea on distribution of intracranial contents. J Neurosurg 18:753-759, 1961
- contents. J Neurosurg 18:753-759, 1961

  19. Langfitt TW, Weinstein JD, Kassell NF, et al.:
  Transmission of increased intracranial pressure. II. Within the supratentorial space.
  J Neurosurg 21:998-1005, 1964
- Cotev S, Cullen D, Severinghaus J: Cerebral ECF acidosis induced by hypoxia at normal and low pCO<sub>2</sub>. Scand J Clin Lab Invest 22, suppl 102:3:E, 1968
- Kaasik AE, Nilsson L, Siesjo BK: Acid-base and lactate-pyruvate changes in brain and CSF in asphyxia and stagnant hypoxia. Scand J Clin Lab Invest 22, suppl 102:3:C, 1968
- 22. Pappius HM: The distribution of water in

- brain tissues swollen in vitro and in vivo, Biology of Neuralgia. Edited by EDP De Robertis, R Carrea. Progr Brain Res 15: 135-154, 1965
- Adolph RJ, Fukusumi H, Fowler NO: Origin of cerebrospinal fluid pulsations. Amer J Physiol 212:840-846, 1967
- Bering EA Jr: Choroid plexus and arterial pulsation of cerebrospinal fluid; demonstration of choroid plexuses as cerebrospinal fluid pump. Arch Neurol Psychiat 73:165– 172, 1955
- Hardung V: Properties of pulse waves in visco-elastic tubings, Handbook of Physiology. Volume I, section 2, Circulation. Washington, D. C., American Physiological Society, 1962, pp 107-135
- 26. Heck AF: Observations on continuous vascular pulsative phenomena in the cerebral cortex of experimental animals with special reference to the effects of closed us. opened calvarium, Cerebral Circulation. Edited by W Luyendijk. Progr Brain Res 30:145— 149, 1968

## Respiration

LUNG ULTRASTRUCTURE Morphology which correlates structure and function of the lung requires fixation techniques which preserve the lung in a condition close to its physiologic state. With standard methods, the fixatives are instilled into the airways. The results are adequate for most studies, provided that conditions are standardized with reference to the toxicity of the instillate (it should be isotonic), and the pressure under which it is injected is controlled. This technique, however, does alter the morphology of the extracellular materials, including the alveolar lining layers, surfactant, and the mucous lining of the bronchial tree. To preserve these factors, the author recommends that the fixative be perfused through the lesser circulation, while airway pressure is kept constant, with the transpulmonary and perfusion pressures under close control. A set of five solutions was administered directly into the pulmonary artery: 1) Ringer's solution containing papaverine sulfate and heparin; 2) 1.5 per cent glutaraldehyde with 1.5 per cent dextran in collidine buffer; 3) Ringer's solution alone; 4) osmium tetroxide with 1.5 per cent dextran in collidine buffer; 5) uranyl acetate in maleate buffer. The dextran adjusts the colloid osmotic pressure to levels similar to blood. With this technique the author was able to visualize clearly the extracellular lining layers and also to identify tubular myelin figures as a major component of the alveolar lining layer. Intravascular perfusion fixation will also preserve other extracellular liquids in the lung, such as the lining laver of airways and intra-alveolar edema. The author suggests that this technique would be of special value in studies of air pollution. (Gil. I.: Ultrastructure of Lung Fixed under Physiologically Defined Conditions, Arch. Intern. Med. 127: 896-902, 1971.)