hours of operation. Since so little advantage is accrued from the use of tranquilizers in addition to the narcotics, the addition of a third drug is not recommended. Levohyoscyamine is more effective than scopolomine or atropine for suppressing vagal activity. (*Adriani, J., Webb, C., and Steiner, L.: Pre-Anesthetic Medication: 1958 Concepts, South. M. J. 52: 1137 (Sept.) 1959.*)

HYALINE MEMBRANE Contamination of the amniotic fluid with blood at time of incision of the uterus stands out as the chief factor in the formation of hyaline membranes in the air passages of the newborn. In a series of 56 infants delivered by cesarean section at term, microscopic examination of the lungs showed contamination of bronchioles and alveoli with blood in more than 90 per cent. Fetal breathing before incision of the uterus was evidenced by contamination of the air passages by foreign matter in those stillborn infants who had died during labor with ruptured membranes. The highest incidence of hyaline membrane formation was in those infants delivered before labor, rather than in those delivered during labor. (*Snyder, F. F.: Pulmonary Hyaline Membrane, Obst. & Gynec. 14: 267 (Sept.) 1959.*)

PAIN In a series of experiments on frogs and human volunteers it was found that pain producing stimuli caused the release of intracellular potassium which is a pain-producing stimulus. If the potassium releasing process was inhibited or neutralized by calcium or reversed by increasing cellular uptake of potassium by means of a glucose-insulin combination, the response to painful stimuli was decreased. The major effect of a noxious stimulus is not the direct effect on the pain receptor but is indirect due to tissue reaction. (*Benjamin, F. B.: Release of Intracellular Potassium as Physiological Stimulus for Pain, J. Appl. Physiol. 14: 643 (July) 1959.*)

METARAMINOL SLOUGH Tissue necrosis and slough occurred in the leg of a patient with severe atherosclerosis who was subjected to a slow intravenous infusion of metaraminol (Aramine). Concentrations of the agent were not excessive (75 mg. of metaraminol in 2,600 cc. of isotonic dextrose), but subcutaneous extravasation occurred along the course of the vein. Multiple intramuscular injections in the adequately vascularized upper arms and shoulders had been well tolerated, as had been the case with intravenous infusions and one episode of subcutaneous infiltration. However, any potent vasoconstrictor drug, when used in the presence of arterial insufficiency, is a potential cause of tissue necrosis. When metaraminol is used, all precautions against tissue damage, such as polyethylene catheters and phentolamine (Regitine) in the local area after extravasation, should be utilized, particularly in the presence of vascular insufficiency. (*Dippy, W. E., and Dorney, E. R.: Tissue Necrosis and Slough Produced by Metaraminol Bitartrate, J. A. M. A. 170: 1647 (Aug. 1) 1959.*)

SHOCK A very significant elevation in plasma lactic dehydrogenase (LDH) activity occurs in experimental hemorrhagic shock in dogs. An initial lag period is followed by a sharp rise, 15–50 times the original value. Such a rise bears a consistent relationship to rate of spontaneous return of blood from the reservoir to the animal ("taking up") and reversibility to replacement transfusion. Studies suggest that serial plasma LDH determinations may prove to be helpful indicators of the extent of biological deterioration in hemorrhagic shock. (*Vessell, E. S., and others: Plasma Lactic Dehydrogenase Activity in Experimental Hemorrhagic Shock, Proc. Soc. Exp. Biol. & Med. 101: 644 (Aug.–Sept.) 1959.*)

SHOCK Reversible and irreversible shock can be caused by intravascular thrombi in the following manner: (a) reversible shock may be due to a decreased cardiac output secondary to an acute cor pulmonale resulting from blockage of pulmonary capillaries with thrombi and associated serotonin-produced vascular spasm or to a damming of blood in the portal system due to thrombi in the liver and associated vascular spasm. (b) Irreversible shock may be due to a loss of blood and serum into the gastrointestinal tract secondary to hemorrhagic necrosis of the bowel mucosa caused by an episode of intravascular clotting in the bowel mucosa and submucosa. (*Hardaway, R. M.,*