Acute Surgical Emergencies, West. J. Surg. 69: 67 (Mar.–Apr.) 1961.)

GLAUCOMA In a review of approximately 25,000 patients, 5 were found who developed acute glaucoma following general surgery. Possible etiological factors include: (1) a preexisting narrow ocular filtration angle; (2) the stress situation of anesthesia or surgery which may produce mydriasis through endogenous catecholamine release; (3) the brief but significant rise in ocular tension caused by succinylcholine during its period of extraocular muscle depolarization; (4) pupillary dilation caused by scopolamine. Of the two commonly used parasympatholytic agents in premedication, atropine and scopolamine, only the latter apparently results in significant pupillary dilation (up to 2 mm. in one series). There are three basic steps to perform in an attempt to avoid acute glaucoma as a complication of anesthesia and surgery: (1) examine the anterior chamber grossly as to depth and inquire as to prior symptoms suggestive of glaucoma, *viz.*, blurred vision, halos around lights, and ocular pain; (2) avoid the use of scopolamine or succinylcholine in "risk" patients if possible; (3) use pilocarpine 2 per cent miotic drops in the eyes before anesthesia is begun in such patients; (4) examine the eyes of every patient during the recovery period for cloudy corneas, pericorneal vascular congestion, or dilated fixed pupil—the signs of acute congestive glaucoma. Any ocular pain should be quickly evaluated and treated. Therapy of acute congestive glaucoma includes the use of a miotic and acetazolamide (Diamox). *(Wang, B. C., and others: Acute Glaucoma After General Surgery, J. A. M. A. 177: 108 (July 15) 1961.)*

ANGIOCARDIOGRAPHY In angiocardiography, a relatively large volume of hypertonic contrast medium is injected rapidly into a vein or into the right side of the heart in patients suffering from cardiac or pulmonary disease. It is not surprising that the mortality of this procedure varies from 0.2 to 4.0 per cent, an extremely high risk for any investigation. The main deleterious actions of contrast media (water-soluble organic iodine compounds) are (1) peripheral vasodilatation and consequent fall of blood pressure; (2) angiotoxic action, *viz.*, increased capillary permeability, congestion, and parenchymal edema; (3) allergic responses such as laryngospasm and bronchospasm, pain and anaphylactoid shock. Contrast media are histamine-releasing agents, and the severity of reaction depends upon the amount of histamine liberated. Patients who suffer from allergic diseases are more liable than normal subjects to have a reaction from contrast media. Pulmonary hypertension is the cardiac condition in which the highest fatality rate occurs. One property of histamine liberators is self-potentiation, by which is meant the increased response to a second injection given a short time after the first. Clinically, there is great danger in repeating an injection of contrast medium without allowing a sufficient interval (at least ten minutes) to elapse from the original injection. There are many reports of patients who tolerated the first injection of contrast medium but suffered a severe and often fatal reaction when it was repeated. *(Lester, E. R., and others: Angiocardiography, Proc. Roy. Soc. Med. 54: 469 (June) 1961.)*

CARBON MONOXIDE POISONING The fatal effects of carbon monoxide even at low concentrations in the inspired air have been attributed to the greater affinity of hemoglobin for this gas than for oxygen. Carboxyhemoglobin alters the dissociation curve of oxyhemoglobin thus impeding oxygen release to tissues. The treatment of carbon monoxide poisoning must be directed to adequate oxygenation of the tissues and to the rapid elimination of carbon monoxide from the body. The administration of oxygen under pressure is a logical form of treatment. Rats, guinea pigs, and dogs were poisoned by breathing 3 per cent carbon monoxide which ordinarily would prove fatal. Oxygen administered at a pressure of 2 atmospheres was successful in preventing the death of the animals. This technique has also been used successfully in persons poisoned with carbon monoxide. *(Lawson, D. D., McAllister, R. A., and Smith, G.: Treatment of Acute Experimental Carbon-Monoxide Poisoning with Oxygen under Pressure, Lancet 1: 800 (Apr. 15) 1961.)*