BURN SHOCK Studies reveal that cardiac output decreases precipitously after burn and is restored with blood volume expansion. After severe burns in patients, pulmonary edema may develop—most likely due to myocardial injury revealed upon pathological examination. The use of digoxin and restoration of blood volume are necessary to increase cardiac output to normal or to prevent its fall after moderate to severe burns. (*Fozzard, H. A.: Myocardial Injury in Burn Shock, Ann. Surg. 154: 113 (July) 1961.*)

BLOOD LOSS Changes in total red cell volume and blood volume were studied by the use of a radioactive chromium technique in 16 patients undergoing radical surgery for cancer. Blood loss at operation was measured by the automatic conductivity bridge technique. Fifteen to 42 per cent of the blood loss at operation was contained on the drapes and surgical gowns. Fifteen of the 16 patients were anemic by blood volume standards the fourteenth postoperative day. The hematocrit was an unreliable guide in assessing changes in the patient's blood volume. After radical operations, the insidious loss of red blood cells in the postoperative period may equal the operative loss. (*Hoye, R. C., Ketcham, A. S., and Berlin, N. I.: Total Red Cell Volume Changes Associated with Extensive Surgery, Surg. Gynec. Obstet. 112: 697 (June) 1961.*)

BLOOD PRESERVATION The three major problems associated with the preservation of whole blood are anticoagulation, surfaces, and storage temperature. Acid-citrate-dextrose is still the most widely used anticoagulant-preservative solution. There appears to be little danger from the use of the citrate ion as an anticoagulant except under unusual circumstances. Silicone coating and plastic containers possibly present less damaging surfaces to whole blood. Newer methods of storage of whole blood include the addition of a nucleotide, adenosine, in order to lengthen the period of erythrocyte survival. In addition, storage at temperatures below −79 C. has permitted almost indefinite survival of red blood cells. (*Ballinger, W. F., and Cohn, H. E.: Preservation of Whole Blood, Int. Abstr. Surg. 112: 411 (May) 1961.*)

BANK BLOOD TOXICITY The depressant effect of banked blood, stored with ACD solution, on the isolated dog heart has been studied. As blood is infused, the depressant effect is evidenced by a decrease in cardiac rate and a negative inotropic effect with final cardiac standstill. This effect is initially seen at *p*H of 7.1 to 7.2 with standstill occurring at *p*H 6.8 to 7.0. Calcium alone will not correct this. Buffering of the blood with THAM to a normal range will result in improvement and, in the presence of a normal calcium level, will restore normal cardiac function. (*Baue, A. E., Hermann, G., and Shaw, R. S.: Study of Bank Blood Toxicity, Surg. Gynec. Obstet. 113: 40 (July) 1961.*)

HYPOTHERMIA Studies of circulation, including ballistocardiography, were performed on 17 subjects before and during induced hypothermia (37 to 30 C.) using either barbiturate or ether or Fluothane anesthesia. Ether and Fluothane produced optimal circulatory conditions. With barbiturates, it was necessary to produce ganglionic blockade in order to achieve a similar status of circulation. Blocking of the muscular component of the thermic counterregulation by curare was insufficient. (*Klensch, H., Goett, U., and Felderhoff, B.: Analysis of Circulation during Induced Hypothermia, Der Anaesthesist 10: 161 (June) 1961.*)

HYPOTHERMIA In the management of critically ill patients, either highly febrile or facing prolonged surgical procedures, hypothermia is used to increase chances for survival. Hypothermia produces numerous effects beneficial to these patients: reduction of oxygen requirements, inhibition of harmful enzymatic activity, reduction in amount of anesthesia required, reduction of cardiac work load, diminution of tissue metabolism, production of hypotension, and inhibition of shock reactions. The surface cooling method of producing hypothermia is used because of its simplicity. Allowing the temperature to go no lower than 32 C. and providing constant electrocardiographic monitoring virtually eliminates the threat of ventricular fibrillation. (*Comar, I., and Farris, J. M.: Hypothermia in*

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 pre-existing 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 extra-ocular 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.)*