of fibrinogen to fibrin in hemorrhagic shock strongly suggests that intravascular coagulation takes place in this type of shock. (Hardaway, R. M., and Burns, J.: Mechanism of Action of Fibrinolysin in the Prevention of Irreversible Hemorrhagic Shock, Ann. Surg. 157: 305 (Feb.) 1963.)

HEMORRHAGIC SHOCK In dogs which survived hemorrhagic shock, effective blood volumes during the shock period were found to be 3 to 9 per cent greater than expected; after blood replacement their blood volumes were 5 per cent less than expected. The animals which died of hemorrhagic shock had lower than expected blood volumes at all times. The prompt and progressive entry of extravascular fluid and erythrocytes into the effective circulation appears to be the major factor promoting survival. After re-transfusion, plasma is lost from the circulation. animals were given lethal doses of Escherichia coli endotoxin. Blood volumes were not al-The rise in tered during endotoxin shock. hematocrit was attributed to plasma sequestration and simultaneous entry of erythrocytes into the effective circulation. (Doberneck, R. C., Johnson, D. G., and Hardaway, R. M.: Blood Volume Adjustments to Shock in Dogs, Arch. Surg. 86: 267 (Feb.) 1963.)

HEMOLYTIC REACTION In a Kell-negative recipient without irregular antibodies, Kell-positive erythrocytes from one transfusion reacted with high-titered anti-Kell antibodies present in a unit of blood transfused subsequently. It was possible to demonstrate in vitro sensitization of mixed populations of Kell-positive and Kell-negative red cells exposed to antibody dilutions analogous to those in the patient. (Zettner, A., and Bove, J. R.: Hemolytic Transfusion Reaction Due to Interdonor Incompatibility, Transfusion 3: 48 (Jan.–Feb.) 1963.)

TRANSFUSION REACTIONS Renal shutdown is a serious complication of transfusion reactions. When oliguria is observed, resuscitative procedures, including mannitol infusion, should be applied promptly. Even when oliguria has been present for several hours and signs of renal shutdown are present, mannitol

frequently is capable of restoring sufficient renal function to prevent the complications and mortality associated with oliguric renal failure. When oliguria persists, early transport to a Renal Center is indicated. (Barry, K. G., and Crosby, W. H.: Prevention and Treatment of Renal Failure following Transfusion Reactions, Transfusion 3: 34 (Jan.-Feb.) 1963.)

NITROUS OXIDE TOXICITY tion of nitrous oxide for periods of time varying from 3 to 19 days was effective in reducing white cell formation in acute and chronic myelogenous leukemia. Nitrous oxide may be toxic on chronic administration. The mechanism by which nitrous oxide produces depression of cell formation is not known, but presumably is similar to the depression of the central nervous system associated with analgesia, anesthesia, and unconsciousness following the administration of nitrous oxide. Nitrous oxide may not be of major benefit in the treatment of leukemia, but the studies of the action of the agent on the hematopoietic system may reveal information concerning the mechanisms of narcosis. An unusual benefit from the administration of nitrous oxide for long periods of time was the relief of pain which continued during times when the patients were not receiving the nitrous oxide. The possibility of addiction following repeated administrations was raised. (Eastwood, D. W., and others: Effect of Nitrous Oxide on the White-Cell Count in Leukemia, New Engl. J. Med. 268: 297 (Feb. 7) 1963.)

HYPERBARIC OXYGEN Two patients with arterial disruption in the lower leg were treated with administration of oxygen by face mask in a chamber in which the pressure was two atmospheres. One had no loss of limb and one needed only midtarsal amputation. eral patients with occlusive arterial disease of the legs appear to have done better than might have been expected. Dogs with total circulatory arrest when similarly treated survived 50 per cent longer under hypothermia (21° C.) than dogs treated with hypothermia alone. (Illingworth, C.: Treatment of Arterial Occlusion Under Oxygen at Two Atmospheres Pressure, Brit. Med. J. 2: 5315 (Nov. 17) 1962.)