taneous respiration. Inhalation of 8 per cent oxygen resulted in arterial saturation of 47 per cent; 5 per cent oxygen produced arterial saturation of 20 per cent. Cardiac output rose; left atrial pressure was unchanged; total pulmonary vascular resistance was unchanged. Mean pulmonary artery pressure increased with 5 per cent oxygen, but was unchanged with 8 per cent oxygen inhalation. (Lancaster, J. R., and others: Effect of Hypoxia on Pulmonary Vasculature of the Dog, Arch. Surg. 87: 485 (Sept.) 1963.)

OXYGEN The results of breathing air or pure oxygen were compared in 44 human subjects utilizing electrocardiographic data. In normal subjects, oxygen induced a small but significant reduction in atrial rate per minute (72 to 66; P = 0.001) and no change in A-V conduction. In diseased hearts (digitalized with normal A-V conduction, prolonged A-V conduction, incomplete A-V block, and atrial fibrillation) there was a similar significant small slowing of heart rate with oxygen, and some significant prolongation of P-R interval in those hearts exhibiting initially either prolonged A-V conduction time (0.24 to 0.26; P = 0.025) or incomplete A-V block (0.22) to 0.24). These effects were brought about by a mild vagal stimulation, presumably via chemoreceptor activity. (Daly, W. J., and Cline, D.: Effects of Breathing Oxygen on Atrioventricular Conduction, Amer. Heart J. 66: 321 (Sept.) 1963.)

BLOOD STORAGE When the blood bank is located some distance from the operating room, blood cross-matched for elective surgery may be released from the blood bank and stored in a commercial refrigerator in the operating room if provided with a temperature recorder and an alarm system. If the refrigerator is not provided with a circulating fan, blood should not be stored near or just beneath the freezing unit. Removal of such blood should be the responsibility of trained and authorized personnel only. The blood bank can stock the refrigerator in the morning and remove unused blood following the surgical schedule. Keeping a stock of Group O Rh negative blood in the operating suite for use when typed blood is being secured

from the blood bank should be discouraged. (Lesses, M. F.: Storage in the O.R., Transfusion 3: 297—Questions and Answers (July-Aug.) 1963.)

SHOCK It is basic to proper therapy to determine whether traumatic shock is hypovolemic or normovolemic. Full blown hypovolemic shock develops when 30 per cent or more of normal blood volume is lost. Blood loss is commonly underestimated. The hematocrit can be used for calculating whole blood volume if the plasma or red cell volume has been measured with RISA or Cr⁵¹-labelled red cells. Normovolemic shock is most frequently caused by toxins, usually bacterial toxins, and the most effective therapy is prompt application of the most effective antibiotic in the most effective manner. There are good reasons for doubting that the use of vasopressors in the long run is advantageous because the pressor effects may be more than counterbalanced by the reduced flow in critical areas such as spleen, liver and intestine. (Fine, J.: Traumatic Shock, Surg. Clin. N. Amer. 43: 597 (June) 1963.)

SHOCK Measurements of blood flow and oxygen consumption were made in animals subjected to hemorrhagic shock. Studies were carried out for limb, liver, and gastrointestinal tract. Reduction of blood flow during shock is proportional to the reduced cardiac output. Oxygen consumption of the gastrointestinal tract remained 40 per cent of control in those animals which failed to respond to transfusion: in those that survived it returned to control values. Hepatic oxygen consumption returned to normal after treatment in the dogs which died; it remained low in those which survived. The hepatic oxygen consumption appears to depend on the amount offered; inadequate utilization by the intestine results in a high portal vein oxygen content and an increase in hepatic consumption. Irreversible shock appears to be related to failure of intestinal tissues to take up oxygen after a critical period of time at a low blood flow. (Bounous, G., Hampson, L. G., and Gurd, F. N.: Regional Blood Flow and Oxygen Consumption in Experimental Hemorrhagic Shock, Arch. Surg. 87: 340 (Aug.) 1963.)