

*Ischemia Produced by General Anesthesia During Temporary Carotid Occlusion, Surgery* 54: 216 (July) 1963.)

#### **HOMOLOGOUS-BLOOD SYNDROME**

Extracorporeal circulation introduces large volumes of homologous blood into the patient's circulation and frequently results in a fall in plasma and red cell volumes that continues into the first postoperative day. Subsequently a substantial increase in both compartments occurs without a change in blood balance. When this phenomenon is not taken into account and large amounts of blood are given for post-perfusion hypotension, with associated blood volume deficit, hypervolemia generally ensues. The conclusion is that the deficit represents sequestration of blood with subsequent de-sequestration of blood leading to a rise in blood volume. The implication is given that the lungs are a major site of sequestration. No attempt is made to maintain isotopic normovolemia after operation. Late blood loss is incompletely replaced in anticipation of de-sequestration. (*Litwak, R. S., and others: Homologous-Blood Syndrome During Extracorporeal Circulation in Man. II. Phenomena of Sequestration and De-sequestration, New Engl. J. Med.* 268: 1377 (June 20) 1963.)

**POSTPERFUSION SYNDROME** Hemodilution perfusion techniques lessen the adverse effects of cardiopulmonary bypass upon the lung, as evidenced by lowered minimal surface tension and improved microscopic appearance. The addition of low molecular weight dextran to whole blood reduces pulmonary histological damage but does not increase the survival rate or lower the minimal surface tensions. The blood components were reduced to one-half the usual concentration while the electrolyte concentration was maintained at normal levels. No deleterious effects of profound hemodilution were noted either during the time of perfusion or in the following 24 hours, provided a small amount of pressor agent was added to the perfusate to prevent a serious drop in blood pressure upon initiation of perfusion. (*Hepps, S. A., and others: Amelioration of the Pulmonary Postperfusion Syndrome with Hemodilution and Low Molecular Weight Dextran, Surgery* 54: 232 (July) 1963.)

**CARDIAC ARREST** Effects of peripheral hypoxia on the dog's heart were evaluated at various levels of cardiac oxygenation by the use of two separate extracorporeal circuits. Cardiac hypoxia of 10 minutes duration induced during normal systemic oxygenation did not result in marked bradycardia or arrhythmia. Systemic hypoxia, however, caused moderate to severe bradycardia, nodal rhythm, and sometimes cardiac arrest even when normal myocardial oxygenation was maintained. These effects, which could be abolished by vagotomy, were more profound when systemic unsaturation was suddenly induced and were most deleterious if the heart was hypoxic as well. (*Austen, W. G., and others: Mechanism of Cardiac Arrest in Acute Hypoxia, Surgery* 53: 784 (June) 1963.)

#### **DIRECT CURRENT DEFIBRILLATION**

Alternating current countershock was applied without success following fibrillation after myocardial infarction, whereas direct current electrical countershock was twice successful in this patient. Continued cardiac massage for periods in excess of three hours proved to be useful with restoration of consciousness despite lack of effective heart action. (*Stanzler, R. M., and others: Comparison of Countershock With Direct and Alternating Current in External Cardiac Defibrillation, New Engl. J. Med.* 268: 1289 (June 6) 1963.)

**HYPOXIA** Circulatory effects of breathing low concentrations of oxygen were studied in 10 anesthetized dogs. Simultaneous measurements were made of cardiac output and blood flow to the head, kidney and hind limb. Four experiments were performed with the addition of succinylcholine to inhibit the ventilatory response to hypoxia and maintain  $P_{CO_2}$  constant. Rise in cardiac output and mean arterial pressure occurred which was significantly correlated to the decrease in arterial oxygen saturation. Blood flow tended to increase during hypoxia in the regions studied but the responses were variable and only the change in renal blood flow had a significant correlation to arterial oxygen unsaturation. Systemic and regional vascular resistances during hypoxia varied both in direction and magnitude of change. Effects of hypoxia influence cardiac

output more than peripheral resistance. (*Murray, J. F., and Young, I. M.: Regional Blood Flow and Cardiac Output During Acute Hypoxia in the Anesthetized Dog, Amer. J. Physiol.* 204: 963 (June) 1963.)

**CORONARY VENOUS OXYGEN** Oxygen content of the coronary venous blood of man and dog is about 5 volumes per cent (25 per cent saturation), the lowest of all body tissues. This condition is related primarily to the myocardial contractile activity which augments the demand and curtails the supply of oxygen through the compression of intramural vessels during systole. (*Baeder, H. S.: Physiologic Basis for the Normally Low Oxygen Content of Coronary Venous Blood, Amer. Heart J.* 65: 844 (June) 1963.)

**CAROTID-SINUS STIMULATION** Carotid sinus stimulation resulted in atrial and nodal tachycardia followed by transient ventricular fibrillation which may have been due to a late manifestation of digitalis intoxication. Carotid sinus massage is occasionally hazardous in patients with evidence of digitalis intoxication. However, carotid sinus massage should continue to be used in the differential diagnosis of an arrhythmia that may be digitalis induced. (*Porus, R. L., and Marcus, F. I.: Ventricular Fibrillation During Carotid-Sinus Stimulation, New Engl. J. Med.* 268: 1338 (June 13) 1963.)

**HYPOTHERMIA** Decreased plasma volume and increased arterial hematocrit after hypothermia were confirmed in a series of dogs. There was a delayed mixing of injected, labeled red cells, indicating a slowly moving volume of red cells. The rapidly circulating red cell volume was calculated using compartmental analysis; this volume was found to be greatly reduced after total body cooling. Only 76 per cent of the animal's red cells were circulating rapidly enough to be mixed with the labeled red cells in the usual 10 minute equilibration period. This failure of red cell circulation is similar to that observed in hemorrhagic shock and thermal injury. (*Chang, C. B., and Shoemaker, W. C.: Effect of Hypothermia on Red Cell Volumes, J. Thor. Cardiov. Surg.* 46: 117 (July) 1963.)

**BLOOD TRANSFUSION** Review of the records of patients receiving blood transfusions over a two year period revealed that 409 or 12 per cent of 3,356 transfusions were of pint volume. Of these, 298 had hemoglobin values of 10 g. or more. Of the nonsurgical patients, 49 out of 91 had hemoglobin values under 9 g. and possibly required transfusion. Ninety-three per cent of the transfusions were given either in the operating room or postanesthesia room. Of these, 56 per cent (105 patients) had hemoglobin values of 12 g. or more and 88 per cent (164 patients) had hemoglobin values of 10 g. or more. Surgeons and anesthesiologists are too often inclined to order transfusions before estimating the exact amount of blood loss. (*Mitty, W. F., and Echemendia, E. M.: Misuse of Blood Transfusions, Geriatrics* 18: 368 (May) 1963.)

**SHOCK** Changes that occur in extraction of pyruvate and lactate by the heart in dogs during hemorrhagic shock return to normal if a transfusion is administered within 60 to 75 minutes of onset of shock. Such changes become irreversible to transfusion if the shock period is prolonged to 180 or more minutes. The extraction of oxygen by the heart is greatly elevated in hemorrhagic shock but returns to normal within 60 to 75 minutes after a blood transfusion. The extraction of oxygen is depressed to very low abnormal levels if a transfusion is given after prolonged hemorrhagic shock. Myocardial enzyme systems are depressed after prolonged shock. (*Hackel, D. B., and Breiteneker, R.: Time Factor in Reversibility of Myocardial Metabolic Changes in Hemorrhagic Shock, Proc. Soc. Exp. Biol. Med.* 113: 534 (July) 1963.)

**COMPLIANCE** In 42 patients subjected to a standard posterolateral thoracotomy incision, the force developed by a mechanical retractor was measured electronically. Adaptation of the thoracic cage to rib separation developed as a function of time. Altered only by periods of light anesthesia, bronchial suction, or extrinsic pressure, the maximal degree of compliance was achieved, on the average, in 17 minutes, and was reflected in the 24 pound average drop in thoracic retractor pressure. This compliance, manifested by the