nization by Anti-Rh, Transfusion 10: 142 (May) 1970.)

POLYCYTHEMIA Polycythemia secondary to congenital heart disease may be treated with phlebotomy, but blood volume should be maintained by the simultaneous infusion of albumin or plasma in order to avoid vascular collapse or cerebral vascular accidents during the procedure. In 22 patients reduction of venous hematocrit from 73 to 62 per cent was accompanied by a decrease of systemic vascular resistance from 29 to 17 mm Hg/min/m^2, and by increases in stroke index (7 ml/beat/m^2), systemic blood flow (0.7 l/min/m^2) and oxygen flux (2.8 ml/min/kg). The decline in arterial saturation from 73 to 59 per cent was more than adequately compensated for by the increase in oxygen flux. *(Rosenthal, A., and others: Acute Hemodynamic Effects of Red Cell Volume Reduction in Polycythemia of Cyanotic Congenital Heart Disease, Circulation 42: 297 (Aug.) 1970.)*

FAT EMBOLISM SYNDROME The diagnosis of the fat embolism syndrome still must be made mainly on its classical physical signs. The earliest sign is almost invariably some form of mental disturbance, such as restlessness or apprehension. This should be looked for during the first day or two after any fracture, but especially if the fracture involves the shaft of the femur or the tibia. There are few useful examinations, but chest roentgenogram, ECG and platelet counts are advisable. Blood-gas analysis should be done immediately when the fat embolism syndrome is suspected. This will almost invariably reveal hypoxia associated with a normal or reduced P_{CO_2}. Treatment consists of immediate correction of hypoxia, the means used being determined by the results of frequent blood-gas analyses. Hypoxia, not cerebral fat embolism, is usually the cause of cerebral dysfunction. There is at present no specific treatment for fat embolism. The present fatalistic approach to patients who are in coma as a result of the fat embolism syndrome should be dismissed and, instead, efforts should be made to correct the hypoxia that is the cause of death. *(Ross, A. P. J.: The Fat Embolism Syndrome: With Special Reference to the Importance of Hypoxia in the Syndrome, Ann. Roy. Coll. Surg. Eng. 46: 159 (March) 1970.)*

STORED BLOOD OXYGEN 2,3-diphosphoglycerate (2,3-DPG) concentrations in blood stored in acid citrate dextrose solution were found to decrease by 50 per cent after three days of storage, by 75 per cent after six days, and by 95 per cent after ten days. After that, no further decreases occurred. The 2,3-DPG content of fresh blood was found to be 4,180 ± 415 millimicromoles/ml of erythrocytes. In five patients who received multiple transfusions (9 to 33 units over 14 to 30 hours) of stored blood, there were decreases in 2,3-DPG levels, shifts to the left of the oxyhemoglobin dissociation curves, and decreases in central venous oxygen tensions. Addition of 0.01 molar solutions of inosine, pyruvate and phosphate to stored blood 95 per cent depleted of 2,3-DPG resulted in supranormal levels of this substrate within four hours. *(Sugerman, H. J., and others: The Basis of Defective Oxygen Delivery from Stored Blood, Surg. Gynec. Obstet. 131: 733 (Oct.) 1970.)*

2,3 DPG IN SICKLE-CELL ANEMIA Blood of patients with sickle-cell anemia (SS) had a decreased affinity for oxygen, although the oxygen affinity of hemoglobin S was the same as that of hemoglobin A. SS erythrocytes contained more 2,3 diphosphoglycerate (DPG) than did normal erythrocytes. The oxygen affinity of hemolyzed erythrocytes was decreased by added DPG, and hemolysates prepared from SS erythrocytes did not differ from normal hemolysates in this regard. Reduction of oxygen affinity to the levels found in intact SS erythrocytes required DPG concentrations in excess of those found in most SS patients. The same was true of oxygen affinities of patients with pyruvate kinase deficiencies. Other organic phosphates, as well as inorganic ions, are known to alter the oxygen affinities of dilute solutions of hemoglobin. These substances, the state of aggregation of hemoglobin molecules, and cytoarchitectural factors probably play roles in determining oxygen affinities of both normal and SS erythrocytes. *(Charache, S., and others: Effect of 2,3-Diphosphoglycerate on Oxygen Affinity of Blood in Sickle Cell Anemia, J. Clin. Invest. 49: 806 (April) 1970.)*