

INTRAVASCULAR COAGULATION

Disseminated intravascular coagulation is an intermediary mechanism of disease. Behind every clotting episode lies an etiologic factor. The major categories of etiologic factors are: intravascular hemolysis, release of tissue thromboplastin, bacterial endotoxin, proteolytic enzymes, particulate or colloidal matter, anoxia and anoxemia, endothelial damage and ingestion of certain lipid substances. It is obvious that the best prevention and treatment of intravascular coagulation lies in the prevention and treatment of the underlying disease. Since this is not possible at present, treatment directed at the coagulation and fibrinolytic systems is indicated. (McKay, D. G.: *Pathology, Diagnosis and Therapy of Disseminated Intravascular Coagulation*, Proc. Roy. Soc. Med. 61: 1129 (Nov.) 1968.)

BLOOD VOLUME AND CARDIOPULMONARY BYPASS

Blood volume was determined pre- and postoperatively in 73 patients undergoing open-heart surgery. The purpose of the study was to compare empirically estimated blood-loss values with those obtained by the RISA and/or Volemetron method. Information so obtained appeared particularly valuable in cases of prolonged cardiopulmonary bypass. Most of the perfusions were performed with the Kay-Cross disk oxygenator. The pump was primed with two parts donor blood and one part glucose in 0.25 per cent saline solution. Preoperative blood volumes averaged 82.4 ml/kg in patients with atrial septal defect, 92.7 ml/kg in patients with aortic insufficiency, and 94.7 ml/kg in patients with mitral insufficiency. Control values were 75 ml/kg for men and 70 ml/kg for women. The postoperative blood volume was decreased by more than 20 per cent two hours after perfusion in 50 per cent of the patients; by more than 25 per cent in 35 per cent of the patients; 20 per cent of the patients had volume deficits of more than 30 per cent. Six hours after perfusion the blood volume reflected the success of transfusion. Only 10 per cent of the patients had blood-volume

deficits of 25 per cent or more. After 20 hours, volume deficits were again more pronounced as the result of blood loss during the night, as well as elimination of plasma expanders; 30 per cent of the patients had deficits greater than 20 per cent; 15 per cent deficits greater than 25 per cent; 10 per cent exceeded 30 per cent. Only 20 per cent approached original preoperative values. The large-vessel hematocrit did not reflect accurately changes in blood volume in the first 20 hours. Likewise, blood pressure values did not reveal true changes of blood volume, as hemodynamic adjustments (centralization of the blood volume with peripheral vasoconstriction and increase of pulse rate) compensated for, and masked, even major blood-volume deficits. Rise of diastolic blood pressure and pulse rate were more sensitive indicators of deficits in circulating blood volume. Central venous pressure was helpful in avoiding overtransfusion, though a low CVP did not necessarily indicate a volume deficit. Critical circulatory failure which does not cause changes in conventional circulatory parameters may result from major deficits in blood volume, the correction of which is imperative for patient survival. (Krumhaar, D., Storch, H. H., and Schmitz, W.: *Comparative Studies of Blood Volume, Blood Loss and Replacement after Cardiopulmonary Bypass for Heart Surgery*, Thoraxchirurgie 16: 507 (Dec.) 1968.)

COARCTATION OF AORTA

Urinary catecholamines were measured before and after surgery for coarctation of the aorta (experimental group) or miscellaneous thoracic surgical procedures (control group). Preoperative values in the two groups were similar. Postoperative levels of catecholamines were elevated in both groups but were much higher in the coarctation patients and remained elevated for longer periods. The mechanism for the increased catechol excretion is related to the baroreceptors, which, following resection of the coarctation, are subjected to decreased