only when more than ten units of blood per hour are required. With severe protracted shock, the tolerance for citrate will be less, and calcium should be administered. With slower rates of transfusion, 2 or 3 ml. of 10 cent calcium chloride appears to provide adequate *in vivo* compensation for the citrate in a unit of ACD blood. An equivalent dose of calcium gluconate is approximately three times larger. In massive transfusion, thrombocytopenia should not be expected until the volume of stored donor blood at least equal to the patient's blood volume has been transfused. Until that amount of blood has been given, Its storage age is of no importance. Once that amount has been exceeded, further blood should have been stored less than 24 hours. Blood of this degree of freshness can be expected to maintain the patient's platelet count above 50,000 per mm.^{3} If the platelet count is lower than that, blood or platelet concentrates collected within 3 to 6 hours will be required to correct the thrombocytopenia. If O negative blood is used for emergency unmatched transfusions in patients who are groups A, B, or AB, this group O blood should be tested and shown to have reasonably low levels of anti-A and anti-B by one or more standard methods. (*Perkins, H. A.: Problems with Massive Transfusions, Questions and Answers Section, Tranfusion 6: 282 (May) 1966.*)

FIBRINOGEN-HEPATITIS RISK The risk of hepatitis following administration of fibrinogen has resulted in attempts to find better ways of preparing fibrinogen. The following steps are thought to have reduced the risk in the present study: (1) the use of volunteer blood donors and the Red Cross Program, (2) two to 8 months storage of plasma prior to fractionation, (3) the use of very large pools containing hepatitis antibody, and (4) the treatment of fibrinogen with ultraviolet light. In one group of patients who received fibrinogen alone (and no blood transfusions), only one hepatitis case resulted. In a much larger group of patients (394) who received an average amount of 7 units of whole blood, 34 cases of hepatitis developed within a 6 month followup period. (*Anderson, H. D., and others: The Clinical Use of Dried Fibrinogen (Human) and the Risk of Transmitting Hepatitis by Its Administration, Transfusion 6: 234 (May) 1966.*)

VASOPRESSORS The effects of intravenous hypertensin and norepinephrin on arterial blood pressure were compared and dose response relations were studied in 18 healthy volunteers. The blood pressure rise is directly proportional to the dose of either drug. Weight for weight, hypertensin is approximately 5 times as potent as norepinephrine in regard to systolic blood pressure and 7 times as potent as to diastolic blood pressure. Consequently the pulse pressure decreases with increasing doses of hypertensin. Both drugs, in equipotent doses, decrease the pulse rate by 16 to 19 per cent. There was practically no change of blood volume and hematocrit after hypertensin. A 7 per cent fall of blood volume and an 8 per cent increase of hematocrit was observed after the injection of norepinephrin. The cause of this difference is attributed to the potent effect of norepinephrin on the venous vascular bed. Serum lactate and pyruvate did not change after injection of therapeutic doses of either drug. There was a mild (0.05 units) shift to the alkaline side of the *p*H with hypertensin; no change was observed after norepinephrine. (*Avenhaus, H., Gerlach, W., and Buchhorn, E.: Comparison of Effects of Vasopressor Substances in Normal Volunteers, Klin. Wschr. 44: 314 (March) 1966.*)

SERUM POTASSIUM The serum potassium level measures the concentration of potassium in the extracellular fluid, not the total body potassium or the potassium of the cells. Total body potassium is 3,000 mEq., only 50 to 70 mEq. normally is present in extracellular fluid. Hyperpotassemia can result from renal failure, tissue trauma, excessive intake, adrenocortical insufficiency, rapid transfusion of bank blood containing high concentrations of extracellular potassium, and respiratory or metabolic acidosis. Hypotassemia can result from gastrointestinal or urinary losses of potassium, decreased intake, transfer of potassium to liver and muscle cells, the action of anabolic

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