

compared to whole blood. For this reason alone, we would emphasize that in the rational management of patients in shock from trauma or burns, gelatin and plasma solutions should be reserved for purely emergency use. They can never be considered as true substitutes for whole blood."

A. W. F.

The Circulation in Traumatic Shock.
Bull. U. S. Army Med. Dept. No. 87
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"Important studies on the circulation in traumatic shock in man were summarized in a recent Harvey Lecture by Dr. D. W. Richards, Jr. The basic dynamic feature, failure of return of blood to the heart with diminished blood flow and tissue anoxia, long recognized from experimental evidence, can now be said to have been proved in human cases of shock. Direct measurements of the pressure of blood in the right auricle and of cardiac output were achieved by means of a long ureteral catheter introduced into a median basilic vein and thence passed along axillary and subclavian veins into the right auricle. Comprehensive studies were made on 92 patients admitted to Bellevue Hospital. . . .

"Evidence was summarized for a strongly selective vasoconstrictor mechanism in shock, shutting off almost completely large organ systems or regions of the body not immediately necessary for survival. In shock, while the total blood flow decreases to half the normal value, the blood flow through the kidney may decrease to one-tenth or one-twentieth, perhaps even less in extreme cases. One patient in deep shock for many hours developed acute renal insufficiency similar to the crush syndrome studied by Bywaters. Spontaneous and abrupt failure of this selective vasoconstriction may precipitate fatal collapse.

"The Trendelenburg, or foot-up, po-

sition increased the cardiac output in patients with moderate reduction of blood volume but was ineffectual when there was marked reduction of blood volume. Cardiac output was not increased by the administration of pressor amines. The effects of alcoholism superimposed on those of shock were most unfavorable. Fat embolism was not observed in the series. . . .

"Persistent shock was characterized by the accumulating effects of tissue anoxia. The brain appeared to fail first. Pulmonary edema was a frequent and difficult complication. Nitrogen retention and oliguria were also observed. The status of oxygen therapy needs further definition.

"When whole blood has been lost in large amounts, replacement by plasma alone will produce an acute anemia. This may actually limit the quantity of plasma that can be given safely. Failure of sustained improvement after the administration of 1,000 to 1,500 cc. of whole blood usually indicated continued bleeding. The author emphasizes the need for further studies on the subsequent maintenance of the patient after he has been resuscitated from shock."

A. W. F.

Special Shock Studies. Bull. U. S. Army Med. Dept. No. 87 (April) 1945.

"Special shock studies have been conducted by Majors D. Ebert and Charles P. Emerson on detached service with the auxiliary surgical group working in field hospitals. Their observations have been summarized in a recent report as follows: All patients with arterial pressure readings below 85 mm. of Hg, excluding cases with cerebral and cord injuries, were found to have an oligemia, the deficiency averaging 40 per cent of the expected normal total blood volume. A significant reduction in blood volume, i.e.,

between 20 per cent and 40 per cent, was not invariably accompanied by hypotension. Hemoconcentration played no role in the pathogenesis of shock, as observed in this series of patients; on the contrary, spontaneous hemodilution of some degree was an almost invariable accompaniment of the condition. The coincidence of anemia and hypotension in these casualties proved to be a reliable index of marked oligemia, adequate treatment of which involved the transfusion of at least 2,000 cc. of whole blood. The pulse rate was found to be useless as an index of the degree of oligemia. Rapid blood transfusion in the treatment of oligemic shock did not appear to precipitate, or materially to enhance, the rate of bleeding in cases with chest or abdominal wounds. The rapid infusion of large volumes of crystalloid solutions—e.g., Alsever's solution employed as the blood diluent—produced but a small and transient increase in the blood volume. True irreversible shock was observed in but two instances; in each of these the total red cell volume on admission was less than 600 cc. Blood volume measurements were made pre- and postoperatively in a number of cases; the blood loss incident to various surgical procedures ranged from 500 cc. to over 3,000 cc.

"As observed by these investigators, complete restoration of the formed elements of the blood to normal levels in all casualties presenting anemia associated with hypotension requires large transfusions of at least 2,000 cc. of whole blood. However, attention should be directed to the fact that a return to 70 or 80 per cent of normal is sufficient to render the casualty safe for initial surgery and evacuation. In view of this fact and since cross-matching is necessary after every 1,000 cc. transfusion of whole blood, it would seem most practical and safe for forward installations such as field and

evacuation hospitals to administer only enough whole blood to satisfy the immediate clinical demands. General hospitals of the base section, on the other hand, have blood available in greater quantity and should assume responsibility for effecting a complete return to normal of hematocrit and hemoglobin values."

A. W. F.

PHEMISTER, D. B., AND LAESTAR, C. H.: *Local Fluid Loss, Nerve Stimuli and Toxins in the Causation of Shock*. Ann. Surg. 121: 803-820 (June) 1945.

"Experiments were conducted on dogs in an endeavor to determine the relative roles of nerve impulses and local fluid loss in the production of shock due to limb trauma. . . . No evidence was obtained from these limb trauma experiments that either a flow of nociceptive stimuli from the injured field or toxin formation is an important contributing factor in the initiation of any circulatory impairment or shock which followed. . . . The animals in which the trauma was applied soon after the administration of a spinal anesthetic were protected from shock principally by the blockage of the vasomotor and (less importantly) motor nerves, which greatly lowered the blood pressure and limited the hemorrhage to an amount that was too small to produce shock, instead of by the blockage of afferent impulses. The maintenance of such a low blood pressure by spinal anesthesia for the prevention of shock during an operation on man is contraindicated as the amount of anesthetic required would be too toxic. . . . In all of the experiments where shock developed the local blood loss was large and constituted the outstanding causative factor. . . . There appears to be no indication for the renewal of efforts to prevent shock by the blockage of afferent nerve im-