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TREATMENT OF SHOCK IN THE FIELD

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THE TREATMENT OF SHOCK IN THE FIELD

INTRODUCTION -

This paper deals with the treatment of shock in wounded soldiers. It deals with shock occuring in young healthy men as a result of extensive tissue injury and associated haemorrhage. This type of shock has been variously named but can be most clearly defined as shock from trauma and haemorrhage.

From July 1940 to the present time I have been resuscitation officer to the 2nd 8th Australian Field Ambulance and have treated shocked patients under all conditions of desert warfare. Until August 1942 I used the routine army transfusion method but was not satisfied with the results obtained; too many cases did not respond despite apparently adequate transfusion. The day of the standard pint transfusion is long passed, it being generally recognised that the volume of blood given any patient must vary with the severity of shock, however, nowhere in the literature on shock is a method described by which the volume of blood required by a shocked patient can be computed in the field. In recent literature rapid transfusion is advocated; it is realised that the response to transfusion varies with the rate of transfusion but nowhere can a statement be found of the optimum speed of transfusion for all grades of shock. The medical officer when treating a case has only broad principles to guide him, the efficiency of his treatment depending on his own experience. Frequently a shocked patient is treated by transfusion given at drip rate hour after hour, bottle after bottle; the only factors that govern the volume or speed of transfusion being death, a blocked needle or obvious recovery. It is essential that there be some definite standards to govern the volume and the speed of transfusion in any case.

I have attempted in this paper to define these standards.

In August 1942 I evolved a method of classification for the grades of severity of shock, for each grade I estimated the volume of blood required and the speed at which it was to be given. I knew

that within the next few months I would have the opportunity of confirming these standards.

I was fortunate in having six orderlies working with me who were highly trained in transfusion work and in having the services of an accurate clerk, these men were instructed in the new method of treatment and in the method of recording during August 1942.

A record was to be kept of all cases treated and by analysis of these records it was hoped to establish:-

(1) That the method of classification used was accurate and of practical value.

(2) The average (standard) volume of blood and serum required for the treatment of each grade of shock.

(3) The optimum (standard) rate of transfusion for each grade of shock.

(4) The frequency, character and severity of complications of transfusion.

(5) The frequency, character and cause of reactions during transfusion.

(6) The prognosis in traumatic shock.

(7) The cause of death in all cases dying at the Main Dressing Station.

The investigation was carried out from September 1st, 1942 to November 4th, 1942 at the Main Dressing Station 2nd 8th Australian Field Ambulance at El Alamein.

The Main Dressing Station was situated about nine miles from the front line; the roads were poor. The majority of patients were admitted during the Battle of El Alamein. In quiet times most patients were admitted about four hours after wounding; during the battle the majority were in the eight to twelve hour period, but in the later stages when the front moved forward some had been wounded for 18-24 hours. The Casualty Clearing Station was four hours distant over a good road. The policy of the Main Dressing Station was to admit all shocked patients to the resuscitation ward the only exceptions being head wounds uncomplicated by any other injury and not bleeding. Two surgical teams were operating, an attached team from 7th Australian General Hospital and the unit team. The surgical

policy was that all life saving surgery should be done, including all compound fractures and all abdominal wounds. From 1st September 42 to 4th November 42, 1600 battle casualties were treated at the Main Dressing Station. 252 were admitted to the resuscitation ward - 50 patients were transfused.

Very few of the men treated were over the age of 32. Only four men were suffering from a complicating medical disease, one of whom had infective hepatitis and three of whom had clinical dysentery. The men were ideal patients mentally as well as physically. They accepted treatment gratefully, bore pain with great fortitude and were not fearful of the outcome. The weather over the whole period was mild, the days were warm to hot, the nights cold but not freezing. A dust laden wind or a heavy dust cloud was present over the battle field all the time.

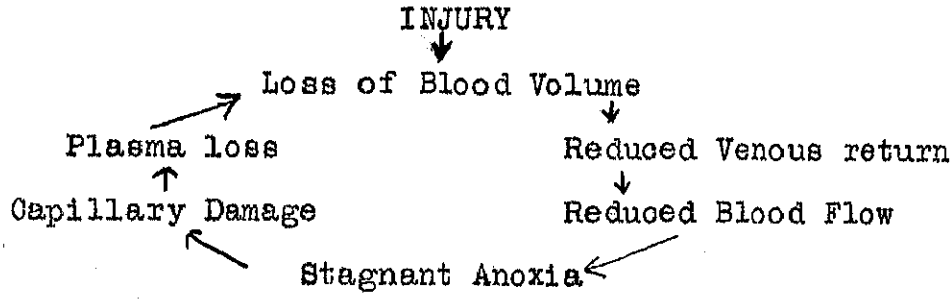
The hypothesis of the cause and mechanism of traumatic shock upon which the method of resuscitation used was based, is a modification of BLALOCK'S (1), (2) theory of shock.

I have modified it to suit the apparent physiological changes occuring in young healthy men.

Traumatic Shock is initiated by an injury. At the site of injury there is a rapid loss of fluid from the injured vessels and tissues. The fluid loss may be due to external haemorrhage, haemorrhage into the injured tissues or to extravasation of plasma from and into the injured tissues. It is usually due to a combination of all these factors. This rapid fluid loss decreases the blood volume and so decreases the venous return. Decreased filling of the Right Auricle results in a reflex general vaso-constriction. Reduction in the vascular field is accompanied by an emptying of all blood storage depots. This is an efficient compensatory mechanism, as the effective blood volume is increased both by decreasing the vascular field and by utilising all the blood available in the body. In a young healthy man this mechanism can compensate for the rapid loss of over 1000 c.cms of blood, without any circulatory failure. If the loss of fluid is stopped at this stage the blood volume returns to normal by an inflow

of extra-vascular tissue fluid. If the loss of blood and plasma continues a progressive fall in blood volume occurs which cannot be compensated and the circulation fails. The volume of blood or plasma necessary to cause a breakdown in the compensatory mechanism varies with rate of loss. If the loss is slow a very large volume can be lost, as it is being continuously made up from the tissues, but a rapid loss of 1500 c.cms of the blood volume cannot be compensated. Decreased blood volume causes a reduction in the rate and the volume of blood flow and a stagnant anoxia occurs in the capillaries. As a result of this the cells of the capillary wall are damaged and lose their function. The capillaries dilate and their permeability is vastly increased, plasma is rapidly lost to the tissues through the damaged capillary walls. This leads to an ever increasing loss of blood volume, a rapidly diminishing venous return, an increasing haemo-concentration, a falling blood pressure and a rapidly failing circulation.

This may be represented diagramatically:-



Many secondary effects occur from the failing circulation (SCHNEDORF and ORR (3)). Stagnant anoxia of the capillaries of the respiratory centre leads to rapid, shallow respirations which are ineffective and an anoxic anoxaemia is added. Anoxaemia decreases the pressor reflexes from the carotid sinus and further lowers the blood pressure.

Unless this cycle is broken the patient will die.

Prolonged anoxaemia causes irreversible damage to the tissues, nervous tissues being damaged most rapidly and heavily. Death occurs in both treated and untreated cases from failure of the respiratory centre.

Various factors play an important part in increasing shock.