CHAPTER 33

TRAUMATIC SHOCK

A full account of traumatic shock cannot be presented here. The experiences of a long war have given us better understanding of the physiological problems involved, but our knowledge is still not complete.

The true basis of humoral shock was recognised by W. B. O'Shaughnessy in 1831 in an enlightening communication on the treatment of cholera, but even in recent times peripheral circulatory failure has not always been understood, and has been confused with failure of the heart.

The histamine hypothesis of 1919 was no longer found adequate, and in 1940 was replaced by the views of Blalock and others who have shown that the symptoms and signs of shock may be explained by the loss of circulating fluid into the injured part. The curious special instance of the “crush syndrome” has, however, reminded us again of the possible importance of chemical and reflex phenomena, as well as of more purely physical mechanisms. The clinical features of traumatic shock appear to be well explained by an interplay of compensatory and decompensatory phenomena, as shown by the experience of Zweifach and others in 1945.

Fluid loss, especially blood loss, soon after injury inaugurates a compensatory mechanism which is characterised by arterial vasoconstriction, augmented response to adrenalin, capillary ischaemia, haemo-concentration and increased spontaneous arteriolar contractility. This increase in the normal periodic contraction and relaxation of arterioles affects the local circulation in the capillary bed, and the prolongation of their phase of contractility is protective in nature. It has been shown that the serum from animals in this phase of shock is vasoexcitor. If death occurs at this early stage the post-mortem findings may be predominantly compensatory, the most notable findings being those due to ischaemia. If the patient survives up to this point the phase of decompensation follows, and if loss of blood and that of plasma are combined and severe the stage of compensation may be very brief.

Decompensatory reactions last longer and cause progressive slowing and eventual suspension of the periodic contraction of arterioles and intense congestion of blood in venules and capillaries. Experiments have shown that the serum at this stage has a vasodepressor effect on control animals. The peripheral vascular tone may be relaxed while the arteries are still constricted, but later they too are affected by decompensation.

It will be seen from these considerations that a balance of the processes of compensation and decompensation, depending on a variety of causes, will determine the issue.

In the decompensatory phase, the capillary blood flow may become so slowed that the state becomes irreversible and a fatal result must supervene. It has been postulated that during prolonged hypotension changes may occur in the damaged tissues which may produce a chemical toxemic
factor, and thus a vicious circle. Fluid replacement can be effective only before these tissue changes occur; after that it is valueless. Deprivation of oxygen and nutriment can be tolerated by the body cells for only a limited period. This is not accurately known: for nerve and muscle the period may be only a few hours.

Study of the "crush syndrome" and similar conditions has shown that profound oligaemia may produce serious and peculiar dysfunction of the kidney, due, it would appear, to a vascular spasm which shunts the renal circulation from the cortex. Delayed effects may appear during the first and second weeks after injuries causing ischaemic necrosis of tissue, chiefly hypertension and uraemia, and death may ensue from pulmonary oedema and uraemic cachexia. It is continued action of the causal factors of the oligaemia that is so dangerous, and therefore in states in which this may occur replacement of lost blood may not alone be effective. Such states are the crush syndrome, severe burns, intestinal obstruction, and special types of infection, such as gas gangrene or continued bone sepsis.

Some understanding of the dynamics of traumatic shock is of greater value than a mere listing of its possible causes. The causes are usually stated as being neurogenic, oligaemic, renal, mechanical, toxicaemic and chemical. With the exception of such mechanical causes of circulatory collapse as embolism due to fat or clot, these "causes" are in part at least stages in a continuous, though often fluctuating, set of phenomena. To assess the exact physiological stage of the condition of patients suffering from shock is a problem of the first magnitude. "Resuscitation" has come more and more to mean timely and adequate replacement of body fluids. This knowledge was reinforced by the experience of the 1914-1918 war in which the now standard resuscitation methods were applied with great success by specially trained and skilful teams. A War Office brochure on resuscitation was issued in 1944, and this, with some local amendments, was adopted for the use of the Australian Military Forces.

Traumatic shock was dealt with in one of a series of special articles in *The Medical Journal of Australia* on war medicine and surgery compiled under the direction of the National Health and Medical Research Council of Australia.

In these publications the following important practical points were stressed: that replacement of the blood volume rather than of the red cells was the first essential, that haemo-dilution is a characteristic response to loss of blood, and that true traumatic shock may exist for some time before a fall of blood pressure is detected. The man who has lost blood obviously needs corpuscles as well as plasma, but they are not his prime need. Further, tests revealing haemo-concentration are not necessarily an accurate measure of the degree of shock from which he suffers, and in addition, compensatory vasoconstriction may maintain his blood pressure for some time, even in the presence of shock, and so prove deceptive.

The essential differences must be realised between traumatic shock due to a rapid and heavy loss of blood, and that due to an injury such as severe burns. A "resuscitation officer" in the field needs clinical acumen and
judgment of a high order to decide quickly what a wounded man needs most, whether it be quiet and rest only for the time being or immediate transfusion. *Triage* is an important duty in a forward surgical station.

A patient's initial condition is not always a sure guide: the nature and extent of his wounds must be ascertained if his true state is to be properly assessed. A systolic blood pressure of 70 to 75 millimetres of mercury is usually regarded as the critical level, but this may be a later sign of severe shock. Haemo-concentration, a valuable sign in some injuries like burns, may also be deceptive. The so-called "classical" picture is like all such type descriptions; it is not a phenomenon which can be relied upon to appear when danger threatens. However, the appearance is distinctive if a pallid patient is seen, cold on the surface of the body, especially on the extremities, with increased pulse rate and falling blood pressure, thirsty, often apathetic, and perhaps somewhat dulled of sensation. More ominous is the alert, garrulous man, perhaps euphoric, perhaps anxious; his falling blood pressure and peripheral cyanosis presage a state which is irreversible and ends in death. A dangerous transition stage is the so-called hyperkinetic phase seen late after massive bleeding. In this there is vasodilatation with warm limbs and a bounding rapid pulse, but the picture may soon change to coldness and pallor. In the former stage immediate transfusion may not be without risk of pulmonary oedema, a danger which may be overcome by a slow transfusion of packed red cells. Severe and untreated wounds sometimes call for caution also and a slow initial transfusion rate is occasionally desirable. Fat embolism, pulmonary embolism and pulmonary blast are conditions in which there is a risk of producing pulmonary oedema by the introduction of any considerable bulk of fluid into the circulation. Obviously the aim in treating traumatic shock is to diagnose accurately the pathological condition underlying the patient's condition and to anticipate any phase of deterioration so that the cycle may be broken before it becomes irreversible.

The accessory factors in treatment are also important. The value of warmth was overstressed in the 1914-1918 war because of climatic conditions, and it has been found since that the application of external warmth is not without risk. Following injury a significant difference has been found in the volume of limbs in cold and warm baths, and fluid loss from the circulation of a damaged limb may be reduced by cooling.

Conversely, caution should be observed in raising the temperature of the skin, lest the protective vasoconstriction give place to dilatation. R. D. Wright and John Devine point out that a drop in the internal temperature is not usual in shock, though the mouth temperature may be subnormal the rectal temperature is more reliable, and should be used as a guide to the external application of heat. Heat should not be applied unless facilities for the restoration of fluid volume are to hand. It is recognised now that the conservation of body warmth at its correct level usually requires enough insulating covering to prevent heat loss, and sometimes, under conditions of severe cold, as occurred in the Western Desert at night, additional external heat. Like many other practical applications of physiology, the
application tends to last as a ritual after the physiology has been forgotten and neglected. It should be remembered at least that "shock cages" are usually dangerous, and that severely damaged limbs should never be warmed.

Pain must be relieved if present. Sometimes the clouding of conscious-
ness due to shock mercifully extends to the diencephalon and morphine is not needed. When it is needed it may not be easy to get it into circulation owing to the sluggish capillary flow. Cumulative action of several doses has occasionally caused dangerous degrees of morphinism. Intravenously administered morphine was often found valuable by medical officers. Anxiety of mind may be as bad as pain; rest and quiet if obtain-
able, together with reassurance and provision of some bodily comfort will alone improve the condition of many patients. Many forward surgical units noted how the condition of ill or wounded men, fatigued by an uncom-
fortable journey, improved noticeably after they had been shaved and fed.

Fluids by mouth are almost always an essential. All men who are conscious, except those who have abdominal wounds, should be freely given fluids to drink. Hot sweet tea was usually popular, and in providing an ample supply of this social and medical necessity chaplains and mem-
ers of auxiliary services often did invaluable work. Yet the practical aspects of the oral route for administration of fluids were sometimes overlooked. The value of this anti-shock measure was clearly stated in 1917 in a report by the Medical Research Council, but in 1940 in the first Western Desert campaign it was sometimes shown that memory is short. Other methods of fluid replacement are considered in full in the following section on blood transfusion. The threat or actual occurrence of ileus in abdominal injuries led to the logical development of fluid replacement by the special measure of continuous gastric or duodenal suction, combined with parenteral infusion of fluids. This is mentioned here to draw attention to the need for maintaining a due balance of water and electrolytes in the body fluids in those forms of abdominal trauma which may cause further dehydration.

The signs of dehydration may not be easy to recognise if attention is diverted by the state of severe wounds. The War Office handbook on resuscitation aptly remarks: "If the medical officer will always consider the possibility of dehydration he will have gone far towards prescribing the best treatment".

Oxygen administration is not necessary as a routine measure. Indeed the anoxia of the wounded is not usually anoxic in origin, but when the mechanical factors of a chest injury are present, or when capillary stagna-
tion is in evidence oxygen is indicated. A practical difficulty in a field unit is that attention is necessary by some adequately trained person for its administration.

The decision as to the form of treatment indicated for any given patient in need of resuscitation rested in the field on clinical considerations. Certain precise measurements were of assistance: estimations of pulse rate and blood pressure are of course necessary parts of a clinical examination. If
local facilities permit, haemoglobin estimations are in this category also, and in the later years of the war a simple method of determining the specific gravity of blood and serum was generally available. Where wounded men could quickly reach a relatively stable forward base these tests could readily be applied, with perhaps in addition the haematocrit reading. The copper sulphate method of the falling drop was adopted as standard. Readings of the specific gravity are easily made by this test, as each drop of the standard solution is coated with copper proteinate and remains discrete for as long as 20 seconds. Neither the size of the drop nor temperature impaired the remarkable accuracy of this test, but like many another precise measurement its interpretation was more difficult. In burns, and in crush and blast injuries the specific gravity of the blood was a reliable index of the degree of shock and of the amount of extra fluid needed in the circulation. But in injuries complicated by continued bleeding and in extensive haemorrhage it was easy to be misled if the haemo-concentration index alone was taken as a guide. If no dilution of blood had taken place, this index was most easily determined in any case by a simple estimation of haemoglobin. These observations in no way invalidated the specific gravity test in other conditions, such as continued sepsis or prolonged fevers. Control of intravenous infusions is important, so as to avoid the risks of unrelieved dehydration and overloading of the circulation, with consequent oedema of the lungs and other tissues.

Biochemical methods, such as described above, have been found of value, provided the fallacies are realised, and corrected by close clinical study of individual patients. Majors A. E. F. Shaw and A. Daly Smith published the results of the study of fifty cases, in which they used estimations of haemoglobin, haematocrit readings, the plasma protein level of the plasma, and plasma chloride content. They concluded that the haematocrit and plasma protein findings were of value in indicating the need of intravenous therapy and the type to be used. They found these methods most useful in the less acute varieties of shock, and realised that without other data such as blood volume and the amount of the electrolyte and fluid reserves caution is required, especially in acute states. H. H. Kretchmar has also constructed a useful nomogram which indicates the degree and type of fluid loss and its compensation.

Experience in resuscitation under varied conditions has shown that while standard routines could be adopted with advantage, judgment based on many factors and often on serial observations was most valuable. The choice of fluid was sometimes dependent on opportunity: blood was nearly always available from donors, whereas serum or plasma was not always at hand. Reconstituted plasma was not as valuable as at first it appeared. These matters are dealt with in the next section. Mention may be made also of two subtle and dangerous sequels of injuries treated by fluid replacement. These are anaemia, appearing some days after stabilisation of the circulation has been obtained, and hypoproteinaemia, occurring at a later date. Both of these were often concomitants of infection, and
could seriously retard healing and recovery. Transfusion of whole blood is obviously indicated.

The accepted practice for resuscitation during the war was well stabilised by 1942 and may be given as follows: (1) arrest of bleeding, (2) tilting the patient's head downwards, (3) assuring that he is warm, but avoiding overheating and sweating, (4) relief of pain, (5) administration of fluids by mouth unless specifically contraindicated, (6) early intravenous use of fluids, and (7) use of oxygen if necessary. It will of course be recognised that the distribution of oxygen by the blood must be adequate for the last measure to be effective.

Little mention has been made here of the effects of toxaemia as a cause of continued shock. The importance of this was well understood in the medical services, where bacterial toxins were a causal agent. The special instance of the crush syndrome was not met with, except rarely, in the Australian forces: it is briefly dealt with elsewhere. The action of bacteria in sustaining shock was dealt with on the prophylactic lines which form one of the chief ends of war surgery.

No special clinical research on shock was carried out in the Australian forces, but close touch was maintained with the work done in England and America. In particular the work by Green and others on the depressor substances such as adenosine triphosphate liberated from damaged tissue, such as muscle, was of interest since research on this and similar subjects had proceeded for some years at the Walter and Eliza Hall Institute of Medical Research. References to literature by Kellaway and others may be found in the reports of that Institute. Further information on field researches will be found in the clinical section of the British Medical War History.

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