THE RESUSCITATION OF BATTLE CASUALTIES

BY

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Progress in surgery has been so rapid that it is difficult for the busy surgeon to keep up with it. The management of shock is an example and this paper reviews some of the recent developments in resuscitation and shock prevention. During the 1914-1918 war the pathology of traumatic shock was seriously studied for the first time and sound principles for its treatment were established. The inter-war years were relatively unproductive, despite some experimental work on both sides of the Atlantic, with sharp differences of opinion over the relative importance of the various factors involved in shock production—neurogenic, oligemic and possibly toxemic. There were few clinical studies of the condition during this time. The 1939-1945 war again brought shock to the forefront of surgical thought; existing knowledge was summarized and extensive clinical studies made from battle casualties. Meanwhile large-scale arrangements were made for the collection, storage, delivery and giving of blood wherever it was needed. The bombing of British cities brought the crush syndrome and again raised the question of a toxemic factor in some types of post-traumatic shock.

By the end of the war there was general agreement on the factors which may produce, increase or modify surgical shock and the principles of treatment were well established, although practical difficulties had prevented them from being followed to their logical conclusions. This had to await the decade since the war when, with the vast amount of war-time experience collected, sifted and...
assimilated, and with important technical advances, the opportunity for trials of improved resuscitation methods came with the localized wars in Korea and Indo-China. The results have been of much interest and are likely to have a profound influence on our ideas about the management of shocked casualties in future.

AETIOLOGICAL FACTORS IN TRAUMATIC SHOCK

Surgical shock is not a single entity but a series of syndromes which, although sometimes seen in almost pure form, more commonly overlap and coalesce. There appear to be three well-defined factors concerned in the production of shock following trauma:

(a) A neurogenic element resulting from excessive nerve stimulation.
(b) An oligæmic element resulting from blood or plasma loss.
(c) A toxæmic element resulting from muscle destruction.

Occasionally one factor predominates so as to produce a distinct type of shock, but the more usual picture is the result of their combination in varying proportions and recognition of this is important in management. Additional factors may complicate the overall picture such as dehydration, salt and electrolyte depletion, and bacterial wound infection. These troublesome and often dangerous complications can usually be prevented by appropriate early measures, if anticipated.

The neurogenic factor in shock results mainly from painful stimulation, although mental apprehension is not unimportant. It is seen in the pure form as the vasovagal syndrome, i.e. syncope after an injury, often slight, though occasionally it may be fatal by itself. Generally with battle casualties, this is the first stage of surgical shock and, although in minor wounds it is transitory, more serious cases with blood loss pass on to secondary shock with or without a period of recovery from the primary neurogenic phase. In its initial state neurogenic shock is not usually serious and responds rapidly to treatment. If it continues, however, or if it is associated with blood loss, it can be dangerous. The importance of neurogenic factors in wound shock has been stressed by Slome & O’Shaughnessy (1938), Wilson (1944), and others.

The oligæmic factor results from loss of circulating blood from hæmorrhage, or of serum, and is well known. It is important to realize that this loss need not be external and that extensive loss may occur into body cavities or tissue spaces, and Blalock (1934), from his experiments, concluded that all the manifestations of surgical shock could be accounted for on this basis alone.

The toxæmic factor, long suspected from strong clinical evidence in certain types of wound or injury, had to wait for biochemical confirmation by Green & Bielschowsky (1943) at Sheffield during the last war. Following intensive work stimulated by the crush syndrome in air raid victims, they finally isolated adenosine triphosphate, a product of muscle disintegration, which can be demonstrated in the blood after extensive muscle damage. The exact role of this and related
substances in the shock syndrome remains uncertain. There is some doubt
whether it can produce serious shock by itself, although the rapidity of onset of
profound shock following the return of circulation to a crushed or damaged
limb is suggestive. However, there is little doubt that toxæmic factors can
be a powerful aggravating influence on the shock syndrome where muscle
damage is extensive (Green & Stoner, 1950). In addition to the above,
surgical shock may be complicated and intensified by such factors as water and
electrolyte loss. Although either of these states can produce a syndrome
of its own with shock-like features (Marriott, 1950), their importance in battle
casualty shock is as a complicating factor under certain circumstances,
recognition of which is necessary. In hot weather or under tropical conditions,
because of excessive sweating and little opportunity to drink during an actual
engagement, a man may be already depleted of both salt and water when wounded.
If, subsequently, water is withheld, for example because of actual or suspected
abdominal penetration, or if, through misguided zeal, the casualty is covered
with blankets despite the climate, or even given hot water bottles, following
common but erroneous practice, both water and salt loss may become so serious,
as a result of excessive sweating, as to deprive the patient of his chance of
survival.

It is therefore necessary to decide, not only which is the predominating
shock-producing element in any casualty, but also to know, in the oligæmic
type, which component of the circulation has been lost. Is it solely blood, is
it largely plasma, or is dehydration the important factor? Usually this is fairly
obvious from the nature and circumstances of the injury—e.g. blood loss from
hæmorrhage, serum loss from burns, a combination of concealed blood loss
and intra-tissue plasma transudation in many soft tissue wounds and crush
injuries, or water-electrolyte deficiency when circumstances have made this
probable. However, in most cases multiple factors are operating and all require
whole blood, although it is important to know how much this should be supple­
mented by other fluids. When facilities exist for quick hæmoglobin and blood
protein estimation, the following table may be helpful:

<table>
<thead>
<tr>
<th>Type of loss from circulating blood</th>
<th>Hæmoglobin</th>
<th>Serum protein</th>
<th>Urinary output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood ... ... ... ... ...</td>
<td>Falls</td>
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<td>Falls</td>
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<tr>
<td>Plasma ... ... ... ... ...</td>
<td>Rises</td>
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<td>Falls</td>
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<td>Water and electrolyte ... ... ...</td>
<td>Rises</td>
<td>Rises</td>
<td>Falls</td>
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Before considering the principles of resuscitation, there are certain important
physiological facts to be remembered. These are:

(a) The total vascular capacity, which for an average adult male is ap­
proximately 5 litres of actual circulating blood together with a potential
capillary capacity of a further 15 litres.

(b) The renal circulation, which at rest is approximately one-fifth of the
cardiac output.
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(c) The compensatory rise in cardiac output as blood hæmoglobin falls. With normal hæmoglobin (14.8 grams per 100 ml.) the cardiac output is approximately 5 litres per minute, but if the hæmoglobin falls to 6 grams per 100 ml., the cardiac output increases threefold to 15 litres per minute.

(d) The compensatory mechanism by which efforts are made to restore the falling blood pressure. The vascular picture in oligemic shock is one of widespread vasoconstriction, involving both arterioles and venules, and the predominance of sympathetic tone is in sharp distinction to primary neurogenic shock where parasympathetic tone predominates. The latter condition is a physiological response to trauma rather than a pathological state resulting from it as occurs in secondary shock.

(e) The fact that urinary secretion (i.e. glomerular filtration) ceases when the blood pressure falls below 80 mm. of mercury.

The points of practical importance in shock management are the efficiency of the compensatory mechanism in maintaining blood pressure and circulation in the face of a falling blood volume; the critical level of 80 mm. of mercury where urinary secretion ceases, and the vast potential capillary reservoir, three times the actual circulating blood, into which blood can be lost when general anoxia leads to capillary dilatation. In the healthy young adult the powers of compensation are considerable and with moderate degrees of blood loss, pressure may be fully maintained; subsequent hæmoglobin estimation alone indicates the loss which has occurred. The corollary is that when the blood pressure does begin to fall and a neurogenic cause can be excluded, the extensive powers of compensation have been fully exploited and are failing. Should the fall be below 100 mm. of mercury the condition is urgent, and if below 80 mm. critical, constituting a surgical emergency requiring priority treatment.

THE TYPICAL SHOCK SYNDROMES

There are four well-defined shock syndromes, recognition of which is a necessary preliminary to appropriate treatment. These are:

(a) Neurogenic shock, which is characterized by low blood pressure, a slow pulse and cold, pale extremities and lips. Unless complicated by hæmorrhage, it is a transitory condition responding rapidly to rest and analgesia.

(b) Compensated oligemic shock, where there is a rapid pulse, cold, pale extremities and lips, but a normal blood pressure (over 100 mm.). Here physiological readjustments have been able to maintain an adequate circulation, but the case needs blood replacement before operation or further evacuation. Two pints (or litres) should be sufficient.

(c) Uncompensated oligemic shock, where there is a rapid pulse, cold, pale extremities and lips, but a low blood pressure. This condition is dangerous and requires urgent treatment.

(d) Severe hæmorrhage, which shows, in addition to a racing pulse and falling
blood pressure, characteristic features of ghastly paleness, intense thirst, dyspnœa, restlessness, mental alertness and such extreme vasoconstriction as to make it very difficult to get a needle into any of the superficial veins because of the spasm.

Needless to say, the latter two syndromes call for immediate, rapid blood transfusion in large amounts. Although severe hæmorrhage presents a fairly characteristic picture, the other three syndromes resemble each other because in each the patient is pale, cold and collapsed. The blood pressure and pulse findings, however, quickly distinguish between them.

THE DANGERS OF SURGICAL SHOCK

The dangers of surgical shock are three: early fatal syncope, irreversibility and late renal failure (traumatic anuria).

Fatal syncope may occur when intense neurogenic stimulation is present, and death may result if attempts are made to move casualties with serious fractures, particularly of the femur, without adequate splinting. It may also occur after extensive burns where many nerve endings are exposed and stimulated. The following case illustrates the danger of continued neurogenic stimulation:

A soldier sustained a compound fracture of the femur when his vehicle left the road. He had to be extricated, carried up the mountain side and taken by truck to a casualty clearing station, where he arrived in less than an hour. The thigh was grossly angulated with the shaft of the femur protruding. He was profoundly shocked although there had been little external bleeding. He died in Reception before transfusion could be started. Autopsy showed no other injury nor evidence of serious blood loss.

Irreversibility occurs when, as a result of failing circulation, tissue anoxia reaches an extent when capillaries lose the power of retaining circulating fluid and transudation into tissue spaces occurs. Although true irreversibility can and does occur in older subjects, it appears to be rare in the young adult and most so-called cases in the past had merely received insufficient blood. Aird (1949) says that he never saw a case during the 1939-45 war, and Allen (1943b, c) was unable to produce irreversibility in his experimental animals. Massive transfusion in Korea produced recovery in some moribund cases who would have been accepted as in irreversible shock by any previous standard (Prentice et al. 1954; Crosby, 1953; Crosby & Howard, 1954).

Post-traumatic renal failure (traumatic anuria) is a comparatively uncommon complication which typically develops several days after injury and carries a high mortality. It occurs when, as a result of loss of circulating fluid, there has been a very low blood pressure of some duration and particularly if this is associated with muscle damage. It is closely related to the crush syndrome, first recognized as a clinical entity during the air raids on Britain in 1940, and will be considered in more detail later.
THE PRINCIPLES OF SHOCK MANAGEMENT

Having discussed the causes and pathology of shock and its dangers, it is possible to lay down rational principles for shock management. These are:

Principle I: To eliminate any neurogenic factors which may be operating and to prevent their recurrence.

Principle II: To restore the volume of circulating blood as quickly as possible and to a level compatible with early operative surgery.

Principle III: To prevent toxæmia from damaged muscle and to protect the kidneys from its effects.

Principle IV: To take such measures as are possible to lower the metabolic requirements of the body until normal circulation can be restored.

It is to new and better ways of giving effect to these principles that progress in shock management must look.

ELIMINATION OF NEUROGENIC FACTORS

The initial vasovagal response (primary shock) is normally a transitory state which, providing the sensory stimulation has not been so intense as to produce fatal syncope, soon passes off, to be followed by a phase of physiological reaction. Much more dangerous is a continuous flow of painful stimuli received centrally from the injured part, and the first principle of shock prevention is to eliminate these completely. This comes second in priority only after the arrest of active hæmorrhage and is achieved by rapid analgesia, followed by effective immobilisation of the injured part wherever anatomically or physiologically feasible.

It is now possible, by combining morphine with a morphine antagonist, to administer it safely in dosage adequate to give almost complete analgesia and to maintain this for an indefinite period. This important new pharmacological discovery provides the means of virtually eliminating neurogenic factors in traumatic shock. Of several known morphine antagonists, the most satisfactory appears to be diaminophenylthiazole hydrobromide (D.A.P.T.), which has little effect on the analgesic properties of morphine but exerts a marked and prolonged antagonism to its respiratory depressant effect, and enables large doses of morphine (up to 2 grains) to be given with safety. The resulting analgesia lasts from six to eight hours, after which the dose can be repeated, and there are said to be practically no side effects (Shaw & Shulman, 1955). So far this drug combination appears to have been used only for the intractible pain of late malignancy, but its application to battle casualties may well prove to be an advance of the same order as the introduction of anaesthesia.

The other factor of major importance in eliminating the neurogenic element from wound shock is effective immobilization of the injured part. This means splinting of major limb wounds whether or not bone has been involved. Although of vital importance in the presence of fractured bone, splinting should not be omitted if soft tissue only is involved because these wounds, too, require
maximum rest and protection. For wounds of the shoulder, arm and elbow the thoraco-brachial plaster offers an effective and comfortable method of immobilization. For major lower limb wounds above the ankle there is no better practical way of obtaining immobilization than the Tobruk splint. With these plasters, casualties can be evacuated safely and comfortably for long distances. Wounds elsewhere should be similarly immobilized whenever possible, and the use of a well-padded temporary plaster shell will give the necessary support and protection. Plaster bandages can, of course, be effectively combined with other forms of splinting to ensure maximum fixation.

Plaster used in this way for temporary immobilization should be applied at advanced dressing station or even at regimental aid post level and arrangements made for its provision there. Recently developed polymer reinforced plaster of Paris can give a strong light cast with half the number of bandages ordinarily used.

RESTORATION OF CIRCULATING FLUID

Urgent replacement of fluid lost to the circulation is fundamental for the prevention and treatment of true surgical shock, *i.e.*, oligemic shock. Of transfusion fluids or blood volume expanders, whole blood is by far the most important. Despite the advances made in transfusion organization and technique during the 1939-45 war, post-war developments have been such that our previous ideas on this subject require reorientation.

Transfusion therapy can be studied under four headings: the types and choice of fluids available; the quantity required and how and when it should be given; the hazards of transfusion; and the methods of storing and supplying transfusion fluids.

THE TYPES AND CHOICE OF TRANSFUSION FLUID

Blood volume expanders now available fall into five groups:

(a) Electrolyte solutions, *e.g.* normal saline or normal glucose;
(b) Synthetic plasma substitutes such as dextran;
(c) Blood plasma and blood serum;
(d) Serum albumin;
(e) Whole blood.

The principle governing transfusion is that, in general, replacement should correspond to the component of the circulation which has been lost. The usual practice with battle casualties is to start off with plasma or serum, or with one of the plasma substitutes, while blood is being checked for compatibility by direct cross match, and then to switch to blood as soon as this is ready. In FARELF we usually start with saline sufficient only to ensure that the set is working correctly, then quickly give one or two bottles of plasma or dextran, by which time the blood is ready.

_Electrolyte solutions_ are retained in the circulation for so short a time as to be of little value except as a very temporary emergency expedient. Their real value comes in maintaining water and electrolyte balance after the blood volume
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has been restored, particularly in abdominal wounds where gastric suction has been necessary or when blood loss is complicated, in hot weather, by dehydration and salt loss which is revealed by the fall or absence of urinary chlorides. In these cases, however, the fluid is better given rectally or by mouth.

Synthetic plasma substitutes have the advantage that they can be supplied in large amounts, they keep indefinitely, and can be given rapidly. At the same time they are retained in the circulation for a considerable time and are therefore effective in expanding and maintaining the blood volume. They are free from all risk of serum jaundice. They are composed of large inert foreign molecules which in due course must be removed from the blood and if possible from the body.

Dextran is the best plasma substitute at present available. It is a polymerized sugar molecule which is metabolized and not stored in the body. All but 7 per cent. is metabolized within ten days, the remainder within the next few weeks. The other plasma substitute in use is polyvinyl pyrrolidone (P.V.P.) which, although equally good for resuscitation, is not metabolized and is excreted only with difficulty. About one-third is excreted quickly, but 25 to 40 per cent. is stored in the tissues. Dextran has in consequence largely replaced it as the compound of choice for clinical use. The amount given is usually restricted to two pints, after which blood is given, and used this way it is a valuable addition to transfusion therapy.

Blood plasma or serum come second only to whole blood as blood volume expanders. Serum is ready for use from the bottle, but plasma is stored dry and must be made up with sterile distilled water or normal glucose immediately before use. Plasma or serum have the great value of providing a highly accessible source of protein which, because of the katabolic phase which follows trauma, is required by the body in large amounts. They have the disadvantage of being a potential source of homologous serum jaundice, and for this reason plasma in particular has been rather under a cloud, although a statistical analysis by Lehan et al. (1949) has shown that, since the use of large-pool plasma was discontinued in Great Britain in June, 1945, the incidence of jaundice with small pool plasma (1.3 per cent.) has been so little greater than that with whole blood transfusion as to be not statistically significant. British-made plasma is now prepared in batches each from the blood of no more than ten donors. It is unfortunate that the method of making plasma, by quick freezing and drying, is also ideal for the long preservation of a virus. This is in contrast to serum where the virus tends to die out fairly soon after storage in solution. Shaw (1955) says that, in Australia, where both plasma and serum are used in the different States, the incidence of homologous serum jaundice varies with the amount of plasma used. In Victoria, where serum only is used, it is almost non-existent. Attempts to destroy the virus in plasma have so far been disappointing.

Although it has been thought that dried plasma can be kept almost indefinitely, experience in Malaya has been that under certain conditions slow deterioration
occurs and reactions, sometimes severe, follow its use. Storage at room temperature in a tropical climate appears greatly to accelerate this deterioration, and the incidence of reactions was found to be proportional to the length of time the plasma had been kept at room temperature. The reactions were sufficiently severe to produce a considerable loss of confidence in plasma among surgeons and transfusion officers in FARELF at the time. When only plasma which had been in continuous refrigeration since arrival from the United Kingdom was used, few reactions were reported (Clyne, 1954).

**Serum albumin** is a product of blood fractionation and has the advantage that it can be effectively sterilized to eliminate jaundice virus. Blood fractionation is popular in America and Australia, as a valuable source of other blood ingredients, but a serious disadvantage to the general adoption of serum albumin in preference to plasma in war time is that considerably less of the former can be prepared from any given number of donors.

*Whole blood* is by far the best transfusion fluid and it supplies all the components lost. Its only disadvantage is the limited period it can be kept before use, and careful storage technique is necessary to retard the progressive haemolysis of red cells which occurs when whole blood is stored.

**THE TIME FOR TRANSFUSION AND AMOUNT REQUIRED**

Having decided on the blood volume expander best suited for the particular case, which will depend on the nature of his injury—simple haemorrhage, extensive soft tissue laceration with blood loss, crush injury, or thermal burns—the question arises as to when transfusion should be given and how much.

On the question of when there can be only one answer—as soon as possible, and this should be sooner than has been customary in the past. Surgical shock is a serious progressive condition which, if severe and unchecked, proceeds to a stage from which no recovery is possible, because a vicious circle develops which cannot be broken, and it becomes irreversible shock. The fact that in healthy young adults true irreversibility is now known to be much longer delayed than was formerly thought does not eliminate its danger. Also, quite apart from irreversibility, the time which renal tissue can tolerate anoxia without suffering irreversible change, leading to traumatic anuria, is limited. Shock is easier to prevent than to treat, and the further it develops the more difficult becomes resuscitation. Blood replacement, therefore, is needed at the earliest possible time, which in war means at field ambulance level, at the advanced dressing station. Although, in Korea, favourable conditions during the static phase enabled the Americans to give blood at battalion level, these circumstances were exceptional. The guiding principle for the management of battle casualties in future should be that no case is evacuated from an advanced dressing station while still shocked. This will make resuscitation one of the most important tasks of the field ambulance. Provided blood loss can be minimized by prompt and effective first aid, and that already lost is replaced without delay, while physio-
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logical compensation is still able to maintain an effective blood pressure, the development of severe degrees of shock can be prevented in most cases, which much simplifies subsequent management. Shock prevention lies in prompt hæmorrhage, effective immobilization and early blood replacement, and the incidence of severe shock among the wounded of a division in action can be taken as a measure of the quality of first aid training among fighting ranks and of the ability of the medical services to provide effective splinting, quick evacuation and prompt blood replacement.

In a division trained in first aid and supported by an efficient field ambulance, organized for blood transfusion when required, casualties suffering from severe shock should be limited to gross multiple wounds, uncontrolled internal bleeding, and extensive soft tissue damage, and making such cases fit for further evacuation will be the main task of resuscitation teams. It has been taught in the past that it is better not to give blood at field ambulance level, but to delay transfusions until arrival at the casualty clearing station, because if a shocked soldier is resuscitated and again relapses, his condition is grave indeed and subsequent resuscitation is difficult. This may be true enough, but to make it a reason against early resuscitation is a dangerous fallacy revealing ignorance of the mechanism of shock production and of the principles of its treatment. One wonders how many lives have been sacrificed in the past to this teaching.

Such cases require early replacement and stabilization followed, if necessary, by a continued slow drip during evacuation, in which case a second serious fall in blood pressure should not occur. If it does under these circumstances, it indicates continued concealed blood loss and the casualty would probably have already died had the early transfusion been withheld. Continuous blood replacement and early surgery offer their only hope. Drip transfusion during evacuation is, in practice, not easy to give and for such cases an attendant experienced with drip technique is almost essential.

In the field, blood loss must be assessed clinically from experience and certain rules of thumb. Blood volume estimation is not practicable at field ambulance level, nor is it necessary, and reference to Table 1 will show how unreliable blood hæmoglobin can be except for cases of pure hæmorrhage. The blood pressure gives little indication of the actual amount lost except that, when it falls below 100 mm. and neurogenic causes can be excluded, it means that loss of the order of about a third of the body blood (i.e. 2 litres) has already occurred and the considerable powers of physiological compensation are failing. One must not be misled in this by lack of evidence of this amount of external bleeding, because plasma exudation and blood extravasation into damaged tissues can account for an extensive loss of circulating fluid.

The amount of blood needed has often been seriously underestimated in the past. In general, a shocked casualty requires sufficient to restore and maintain his systolic blood pressure at about 110 mm. of mercury and, if compensation has failed, this may take a large amount of blood. The usual practice is to run in two bottles of plasma or dextran as quickly as possible while the blood is got ready, and then to carry on with blood. Experience in FARELF has been
that few medical officers, unless with previous experience of battle casualties, have any real idea of the extent of blood loss in serious wounds, and as a working rule I advise the newly arrived to decide carefully, from the size of the wound, the time since wounding, and from evidence of external bleeding on the dressing, etc., how much blood they think has been lost. Doubling this figure will then be nearer to the truth. This has proved quite a useful rule.

With the assessment of blood loss presenting such difficulty to the inexperienced, it is necessary to have some rule of thumb to work from. I have found the following a useful rough guide:

(a) After hemorrhage, compensation fails and the blood pressure falls below 100 mm. when 2 litres (3½ pints) have been lost, but the true picture is seen only after neurogenic hypotension has been eliminated;

(b) Loss of blood and plasma into damaged tissue can be roughly calculated by estimating the size of the wound or wounds in terms of clenched fist volumes and allowing one pint (0.7 litre) for each fist unit of damaged tissue.

Provided compensation has not failed completely (i.e. the blood pressure is still 80 mm. of mercury or over), rapid transfusion of blood estimated as above should restore it, although even this amount may be an underestimation because of the difficulty in deciding how much bleeding has occurred.

If such blood replacement fails fully to restore or maintain the blood pressure, it suggests concealed bleeding and continuous blood replacement is necessary pending operation. But if compensation has already failed and the blood pressure has fallen to 70 mm. or below, the quantity of blood replacement calculated as above is quite inadequate and may have to be at least doubled if resuscitation is to be achieved. Sometimes even doubling is insufficient and in extreme instances massive transfusion has been necessary. The quicker the blood can be given, the less will be required.

The resuscitation of badly shocked battle casualties by massive blood transfusion is a new development which came with the Korean war. Here, the static conditions of the front line during the latter stages and the large amounts of blood available, enabled forward transfusion to be used by the Americans on a quite unprecedented scale, and amounts up to 30 pints of blood were given in some instances (Crosby, 1953; Prentice et al. 1954; Crosby & Howard, 1954). The result was the recovery of moribund cases who would otherwise certainly have died. Surprisingly, there were no ill effects reported from such massive transfusions despite the fact that the amount given was several times the total normal body blood. The question arises as to what happens to this blood and how does it, eventually, produce its effect. It used to be thought that generalized increased capillary permeability resulting from anoxia was the basic mechanism of severe shock (McDowall, 1940; Moon, 1944), but this is now doubtful. Should it happen, shock is truly irreversible, but this state, if it occurs at all in the young adult, is now known to be rare. What does occur is extensive capillary dilatation and blood drains into this great pool (15 litres). Added to this is the considerable
loss of blood and plasma into the damaged tissues themselves, and Prentice et al. (1954) believed that continued oozing in this way during transfusion and operation accounted for much of the large amounts transfused in their series. In order to get blood into active circulation, it is necessary first to fill the dilated capillaries. The quicker blood can be given, the more is available before it drains into the capillary pool and, by supplying much needed oxygen, it prevents their further dilatation. As the circulation improves and capillary tone is slowly restored, sufficient blood returns to active circulation to compensate for the loss which is continuing into damaged tissues, but apparently never so fast as to threaten to embarrass the circulation. The bulk of the transfused blood remains in the viscera and skeletal muscles until it is actively broken down, as part of the phenomenon of post-traumatic katabolism, to help supply protein components needed for repair. The Surgical Research Team of the U.S. Army in Korea, using red cells tagged with radio-active chromium, found that the liver, lungs and skeletal muscles were important regions where the excess blood was concentrated (Crosby, 1953). The Korean experience with massive transfusion was that heart failure and pulmonary oedema did not occur, although a few patients developed cyanosis and râles for a short time. This ability to withstand massive transfusion was attributed to the physical fitness of young soldiers of that age group (Crosby, 1953). Blood volume estimation following massive transfusion showed no increase above normal despite the large amount given (Prentice et al. 1954).

It will be seen that, when compensation has failed, the speed with which blood is given is important because it is necessary here to break a vicious circle. The falling blood pressure, by reducing venous return, leads to a reduced cardiac output which intensifies the capillary anoxia and consequent dilatation, and so further reduces venous return. It is essential to supply rapidly a head of pressure for the heart to pump against. Rapid blood administration has in the past not always been easy to achieve, despite the use of positive pressure and two veins simultaneously, and in the most urgent cases venous spasm may be troublesome. If necessary, a vein should be cut down on and a cannula tied in. Unless given under positive pressure, blood runs in too slowly for the urgent needs of the severely shocked or exsanguinated, and even the faster-running dextran or plasma are not quick enough. Positive pressure transfusion is essential here both to overcome venous spasm and for speedy restoration of circulating fluid. In the past, this has been achieved by the use of a Higginson syringe, but this is potentially dangerous, with a real risk of fatal air embolism unless the utmost vigilance is observed. A momentary distraction may prove disastrous, and even with it the rate of giving is often still too slow. Recently a satisfactory mechanical transfusion pump has appeared on the market.* Blood is forced along the tube by rollers on turning a handle. This enables it to be given rapidly and safely under considerable pressure, a pint in under two minutes, and overcomes minor blockages and local spasm. Such an instrument is an essential addition to transfusion equipment and may well obviate the necessity for using the intra-arterial route which was developed to save these urgent cases.

* The Martin transfusion pump supplied by Messrs. Allen and Hanbury Ltd.
Intra-arterial transfusion has proved life-saving in desperate cases, for which it may in future challenge the intravenous route as the method of choice. The entrance of transfused blood direct to the arterial side immediately raises arterial pressure and, by giving the heart something to pump against, at once improves cardiac output. Intra-arterial transfusion was used by the Russians for reviving shocked casualties during the 1939-45 war and subsequently has been receiving increasing attention. Bingham (1952), reporting over 100 cases, considers it to be far superior to intravenous transfusion. He gives the blood by cannula into the radial or dorsalis pedis artery and advises using heparin as a safeguard against clotting. Wilson, Wallace & Whiting (1952) give similar enthusiastic reports from America; and Brown (1953), using the radial artery, reports 165 cases treated at Edinburgh with better results than obtainable by the intravenous route. Intra-arterial transfusion is more effective in restoring cardiac output than intravenous, and appears to be the method of choice for patients with profound shock or in extremis from massive haemorrhage. Here speed is vital if the patient is to survive, and this outweighs in importance the risk, which is real, of producing arterial vasospasm distally. This may be sufficient to produce gangrene of distal extremities and therefore the left wrist should be chosen. It is considered dangerous to transfuse fluids other than blood in this way. For circulatory collapse during laparotomy, transfusion direct into the aorta or common iliac artery has been life-saving. The use of a transfusion pump with intra-arterial transfusion enables blood to be given rapidly at adequate pressure (120 mm. Hg) without risk of air embolism (Melrose & Wilson, 1953).

In cases with compensated oligemic shock (i.e. blood pressure still over 100 mm.) it is essential to give blood before attempting any surgery, because they may be in the last stages of compensation and further sudden blood loss can be disastrous. The following example illustrates this danger:

A soldier was shot through the elbow, fracturing the lower end of the humerus. He reached hospital six hours later, apparently in good condition, with a blood pressure just over 100 mm. The dressing was soaked in blood but he was not apparently actively bleeding. He was considered fit for surgery and taken to the theatre without preliminary transfusion. At operation, venous bleeding was encountered which welled up from the depths of the wound and could only be controlled by packing. At this stage the patient’s condition deteriorated and he went into deep shock. Only then was blood transfusion started and he was returned to the ward with a drip running. He died two hours later, still profoundly shocked.

In this case, the deceptive powers of compensation of a young adult had disguised the gravity of the blood loss. He should have received 2-3 pints of blood before any surgery was attempted and this should have been continued as a slow drip during operation. When he collapsed, massive blood replacement given rapidly, might have saved his life. The surgeon in this case had had no previous experience of battle casualties.

HAZARDS OF TRANSFUSION

The main complications of blood transfusion are incompatibility, rigors, air embolism, overtransfusion and homologous serum jaundice. Incompatibility
may be either a major mismatch or an intergroup incompatibility. Under field conditions, only Group O blood (universal donor) is supplied for casualty resuscitation and this eliminates the possibility of any major mismatch occurring. Although direct cross-matching is usually done as an extra precaution, it can be omitted in emergencies. Out of the vast amount of blood supplied by the Army Transfusion Service during the 1939-45 war, disasters from blood incompatibility were almost unknown although approximately 10 per cent. of all wounded required to be transfused. This was achieved by meticulous preliminary checking of the agglutinin and agglutinogen content of all Group O blood before release (Whitby, 1953). At base hospitals, where facilities for accurate typing and cross-matching exist, type specific blood, if possible fresh, is preferred, particularly when repeated transfusions must be given.

During the Korean war the Americans made an extensive study of blood transfusion, particularly with regard to possible dangers from the large quantities of blood sometimes given. In the U.S. 8th Army, only Group O blood was used. As all Group O blood contains some anti-A and anti-B antibodies, preliminary screening was always done for these agglutinins and it was divided into two sub-groups: high titre Group O (greater than 1:200) and low titre Group O. Most blood is of the latter type and is universal donor blood, safe to use for any person, regardless of group, and without any preliminary cross-matching. The use of such blood in forward dressing stations eliminates the delay of cross-matching and the need for skilled technicians to do it. High titre Group O blood is safe to use only with Group O recipients, and this sub-group constitute "dangerous universal donors," because such blood may produce with other groups a haemolytic transfusion reaction of the major incompatibility type (Steer, 1953; Crosby, 1953).

Intergroup incompatibility has now reached such complexity that only skilled and time-consuming cross-matching can eliminate its possibility. Of the various possible intergroup factors, the Rh factor is the most important, but in Korea this was disregarded and accepted as a calculated risk in the forward resuscitation of battle casualties. Fortunately, only about 15 per cent. of subjects are Rh negative and of these even fewer are capable of developing Rh antibodies and then only after multiple transfusions over an appreciable interval of time. It was, however, disregarded only in the field and not when patients reached base hospitals (Steer, 1953).

Rigors are common during the administration of both blood and plasma, particularly when given rapidly. The reaction, although alarming to the uninitiated, is innocuous and not an indication for stopping the transfusion, and the rigor nearly always stops when the speed of flow is reduced. If severe, it may be controlled with morphine injection.

Air embolism is a real risk when positive pressure transfusion is used and requires unremitting attention to the apparatus if the risk is to be avoided. Pressure should be released when three-quarters of the bottle have been given. The rotary pump avoids this danger.

Overtransfusion appears not to be a serious risk in acute casualty resuscitation.
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in young adults. Cases of compensated oligemic shock require a prophylactic transfusion of 2-3 pints (1-1½ litres) and no more. Cases of uncompensated shock require rapid continuous transfusion until their blood pressure rises over 100 mm., and until this happens there is no possibility of their being overtransfused, because of the capillary dilatation and exudation into damaged tissues. This is why such large amounts of blood can and must be sometimes given. This, however, is not the case after stabilization has occurred and if transfusion is repeated several days later, in order to correct the post traumatic anæmia which occurs as part of the phenomenon of repair katabolism (Cuthbertson, 1942; 1950). Then the blood must be given very slowly to avoid embarrassing the heart. The same precaution applies to burns transfused after the first forty-eight hours, when serum exudation has ceased. The danger of overtransfusion then is real. Subsidiary electrolyte solutions, if needed, are better given rectally or intra-fascially, e.g. subpectorally, if they cannot be taken by mouth. If gastric suction demands a continuous intravenous drip, it must be a slow one.

Homologous serum jaundice has already been mentioned. It can never be completely eliminated as a risk in whole blood transfusion, although, if donors with a recent history of jaundice are excluded, the risk is relatively small, less than 1 per cent. in England (Lehane et al. 1949). The other potential source is plasma and present methods of preparation tend to preserve the virus, but improved techniques may eventually eliminate this source.

STORAGE AND DISTRIBUTION OF TRANSFUSION FLUIDS
The supply and distribution of plasma, serum and the synthetic plasma substitutes under field conditions present no special difficulties. Blood, on the other hand, has a relatively short life and any way of increasing the period during which it can be usefully and safely administered is important when blood is needed in large amounts. The changes which occur in bank blood during its storage and transportation and any harmful effects which may result from transfusion of such blood in large amounts was the subject of investigation by the Surgical Research Team of the U.S. Army in Korea (Crosby, 1953; Crosby & Howard, 1954).

It is known that, during refrigeration, potassium gradually leaves the red cell and that stored red cells are abnormally fragile when suspended in hypotonic saline. Both these changes have, however, been shown to be reversible. When blood is chilled to 4°C, potassium diffuses out as a result of the much reduced cell metabolism, but if the blood is subsequently warmed and glucose is available, the cell takes up potassium again from the plasma. High plasma potassium in stored blood is thus not necessarily evidence of its deterioration. The increased fragility of stored red cells has been shown to be, in part, a result of this potassium shift and, like it, reversible. After transfusion, the cell regains its normal shape and fragility. On the other hand, plasma hæmoglobin concentration in bank blood appears to be an accurate index of the extent of hæmolysis which has occurred during storage, and plasma hæmoglobin tests show that blood is not
seriously damaged by storage and transport up to three to four weeks, provided adequate refrigeration (4°-10° C.) is maintained, and studies in cell survival show that such blood bank cells survive for at least twenty-four hours after transfusion without being haemolysed. They are viable and capable of function (Crosby, 1953). British experience in North-West Europe was that blood survived well and could be used up to twenty-eight days from time of collection, but that, unless adequate time were allowed for cells to settle, it was impossible, from inspection, to tell whether or not haemolysis had occurred and much blood was needlessly discarded as haemolysed because of its appearance (Conway, 1953).

During transport of blood, any sudden movement or agitation favours haemolysis. It has been found that this can be much reduced and the life of the blood appreciably increased by filling bottles right up to the stopper instead of leaving the usual small air space.

For rapid transfusion under field conditions, the use of expendable plastic giving sets is a marked advance which should eliminate most of the troubles that have been the bane of blood transfusion during the Malayan operations—viz. perished rubber, the difficulty of effectively cleaning sets and tubing after use, unexplained reactions and the like. Giving sets in war should be used once only, then discarded.

THE PROTECTION OF THE KIDNEYS

Toxic changes in the kidneys leading to a lower nephron degeneration are likely to follow continued low blood pressure, particularly but not only, if associated with muscle damage. The result is renal failure or traumatic anuria which has a high mortality, because death commonly occurs before the kidneys are able to recover from the damage sustained.

Traumatic anuria is closely related to the crush syndrome, first recognized during the blitz in Britain. Its incidence was between 3 and 4 per cent. of air raid casualties, and the common feature in all cases was some form of crushing injury resulting in plasma loss into bruised tissues, sufficient to produce severe oligoëmic shock, with which was associated considerable muscle necrosis, mainly ischæmic in origin. Cases surviving the initial shock later developed anuria which was usually fatal. The characteristic renal lesion was a lower nephron degeneration similar to that found after mismatched transfusion, calculus anuria and sulphonamide poisoning, and many of the tubules were blocked with pigment casts of precipitated myoglobin.

It was subsequently found that traumatic anuria was not confined to crush injuries, but that it could complicate ordinary missile wounds where there had been a low blood pressure (70 mm. or less) for a period of the order of two hours or more. Although some muscle damage was always present in these cases, it was no more than in many other wounds. The common feature was the sustained hypotension. Balch, Meroney & Yoshio Sako (1955) have reported twenty-four cases among Korean war casualties, and nine cases have occurred in Far East Land Forces since 1950, five among wounded, three following traffic accidents and one after haemorrhage. Two of these recovered. The following was typical:
A police officer was shot in the thigh when a train was fired on, fracturing the femur. He received no proper splinting and on arrival at hospital two hours later was profoundly shocked. This was treated by transfusion, and after several hours resuscitation the blood pressure had risen sufficiently for wound toilet and splinting. His immediate post-operative condition was satisfactory with no further fall in blood pressure, but after forty-eight hours he began to develop progressive oliguria with a rising blood urea. This went on to complete anuria and he died twelve days later. This case well illustrates the danger to the kidney of a continued low blood pressure and the urgency of restoring it.

Much speculation has taken place regarding the relative importance of plasma loss into damaged tissues and of vaso-depressant products of muscle necrosis, such as adenosine triphosphate, in causing shock. There has also been considerable discussion as to the actual cause of the renal damage. Green & Stoner (1950) have shown experimentally that the shock-producing effect of adenosine triphosphate injection could be much reduced by the prophylactic administration of large amounts of normal saline solution, but only if this were given before renal damage has occurred. They also showed that the shock-producing action of this compound was much increased by raising the surrounding temperature; an observation which has an important bearing on shock management. Experience from British air raid casualties was that early alkalization of the urine was of real protective value, as was pressure bandaging the crushed limb. Alkalization was obtained by giving bicarbonate by mouth and lactate and plasma transfusion by veins, but it was agreed that in order to achieve success this way, it was necessary to give alkalies before and not after renal damage had occurred (Bywaters & McMichael, 1953).

The treatment of established renal failure is difficult and unsatisfactory. The main danger is a progressive disturbance of the electrolyte balance resulting in potassium intoxication and acidosis. The problem is to tide over the patient until the tubules have had the time to recover, and the usual methods of protein elimination from the diet and encouraging other routes of nitrogen excretion have proved disappointing in these cases. The American Surgical Research Team which investigated the syndrome in Korea tried the artificial kidney for some cases with reported success. Of eighteen cases dialysed, six survived, and it was considered these otherwise would have died, and of the remainder, renal failure was the immediate cause of death in one case only (Balch et al. 1955).

Traumatic anuria remains a serious complication of a small proportion of traumatic casualties, battle, air raid or even traffic. The common feature seems to be a continued low blood pressure and, once established, the condition carries a high mortality and is difficult to treat. Prevention therefore must be the basis of management and for this the following rules are useful:

(a) A sustained low blood pressure is a very dangerous condition. Every effort must be made, by prompt effective first aid measures, to prevent it occurring, and if it develops it demands most urgent and energetic treatment to restore the blood pressure before renal damage becomes irreversible.

(b) Cases with extensive muscle damage, specially from crush injury, are
particularly dangerous, partly from the intensity of the shock which develops and may be fatal and partly because of the special risk of traumatic anuria in survivors. They therefore require early surgical treatment, as soon as vigorous resuscitation has made this possible, with excision of necrotic muscle. This is just as important after crush injuries as after flesh wounds.

(c) When a limb has been crushed and the circulation obstructed, a pressure bandage should be applied as the limb is released and kept on until plasma transfusion has been started and the limb can be explored surgically if necessary. Removal of the bandage will produce a sharp fall in blood pressure as blood drains into the damaged tissues, the effect being similar to that of a major haemorrhage. Added to this sudden blood loss is the vaso-depressant action of released muscle toxins. It is essential therefore to avoid this until plasma or blood can be replaced as quickly as it is lost. The preliminary administration of intravenous saline, together with alkalis by mouth, will help protect the kidney from the effects of released myoglobin. At operation, the extent of muscle destruction resulting from the crush must be determined, non-viable muscle excised, or even amputation performed if it is not considered to be safe to preserve the limb. The subpectoral route is an easy and very effective way of continuing saline while blood is being transfused intravenously and considerable amounts can be absorbed in this way. A rectal drip, provided it is run in slowly, is also a good accessory route.

(d) Temperature plays an important part in determining the speed and extent of muscle necrosis following ischaemia, which is much retarded if the wounded or crushed limb is kept cold until adequate surgical toilet is possible. Deliberately chilling the limb in these circumstances is a rational procedure.

(e) Whenever the blood pressure has remained at 70 mm. or less for over an hour, or if there has been extensive muscle damage, the casualty must be regarded as a case of potential traumatic anuria and measures to protect the kidneys taken from the outset—i.e. “push” saline, make and keep the urine alkaline, eliminate protein from the diet and keep the damaged limb chilled. The urinary output and blood urea should then be carefully watched. It is dangerous, however, to continue to “push” fluids once urinary secretion begins to fail, and at this stage the fluid intake should be restricted as a precaution against pulmonary oedema.

REDUCTION OF METABOLIC REQUIREMENTS

Peripheral arteriolar constriction is a common feature of all forms of shock and is often associated with sweating. As a result the skin, particularly of the extremities, becomes pale, cold and clammy and the patient may feel cold. Because of this, the warming of casualties with shock, by blankets, hot water bottles or other means, has been long accepted without question as an essential part of shock management. But with increasing experience, many surgeons began to realize that when heating was pressed to the point of causing sweating, it merely increased both the fluid loss and the discomfort of the patient and was doing more harm than good. By the end of the last war, it was generally
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advised that heating the patient should be discontinued before it produced sweating. Nevertheless, wrapping a shocked casualty in blankets and applying hot water bottles is still common practice, even in hot climates.

It was during the latter part of the 1939-45 war that the wisdom of actively heating patients in shock was first doubted. It was observed, at Tobruk, that casualties who had been immersed in cold sea water for several hours were usually in better condition than those with similar wounds not immersed (Devine, 1943). Kay (1944) at Glasgow, reported the deleterious effect of overheating shocked cases. Experimental work confirmed these clinical observations. Bla­lock & Mason (1941) found that applying heat hastened the death of dogs with shock due to hæmorrhage, while cold delayed, although it did not prevent, death. Wakin & Gatch (1943), at Indiana University, reported that both heat and cold were harmful to shocked animals, while the experimental work of Allen (1943a) showed that not only was a slightly subnormal temperature harmless in shock, but it was probably beneficial. There is thus now considerable evidence, clinical and experimental, that the optimum temperature for a patient with traumatic shock is slightly subnormal, and that any heating should be done with considerable caution and should never produce sweating. A subnormal temperature, by reducing the metabolic requirements of the body, eases the burden on the failing circulation, while at the same time it inhibits the production of vasodepressants from damaged tissue which may intensify the shock (Green & Stoner, 1950). The skin is always cold and clammy in shock and the patient often feels cold, nevertheless to apply heat may well diminish his survival chances. In hot weather or in the tropics it is absolutely contraindicated and no form of external heat is needed no matter how cold the patient may feel.

During the war in Indo-China, the French carried this a stage further and used controlled hypothermia as a method of treating patients in severe shock and also prophylactically to forestall the development of shock in cases where this was anticipated. Simultaneously blood replacement was essential (Laborit & Huguenard, 1953; Creyssel & Deleuze, 1953). Although the rationale is sound to prevent tissue anoxia by reducing metabolic requirements and to inhibit enzyme activity, the risk of causing severe renal damage while doing so must be remembered. However, provided an adequate renal circulation can be maintained by blood replacement and a safe technique developed for maintaining hypothermia with serious battle casualties, artificial hibernation appears to have fascinating possibilities and be worthy of careful investigation and further trial.

SUMMARY

1. The principles of resuscitation of war-time casualties have been reviewed in the light of recent developments.

2. Revolutionary post-war advances include the use of massive blood replacement and intra-arterial transfusion, mechanical transfusion pumps, the discovery of morphine antagonists and the possible use of controlled hypothermia in the management of shock.
3. With improved methods of prevention and treatment, shock in the future should lose many of its terrors and resuscitation will become one of the most important tasks at field ambulance level.

REFERENCES