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RECENT ADVANCES IN HAEMORRHAGE AND SHOCK*

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I wish to say, first, that it is a privilege to be asked to address this Conference of Surgeons of the St. John Ambulance Brigade. In tendering my thanks for the opportunity I would like to acknowledge my debt to my research associates and clinical colleagues at the Birmingham Accident Hospital. This is important, for these observations would not have been possible without the existence of an independent unit attempting to focus the potential of modern medicine and scientific research on the treatment and study of patients suffering from the effects of injury.

In the past I have been critical of inadequacies in the teaching of first aid, and in particular of many of the first-aid textbooks. A year ago I opened a discussion at a meeting of industrial medical officers and nurses with some sharp comments on first-aid orthodoxy. I tried to be provocative, and it is perhaps due to that attempt that I have been invited to give this address.

Meanwhile I have delved more deeply into the historical origins of ideas which many of us now consider to be out of date. I had not previously appreciated the weight of medical scientific authority which lay behind the treatment of "shock" by artificial heating, hot sweet tea, and rubbing the limbs. The argument is presented in theory and practice in the Medical Research Committee's green book on "Wound Shock and Haemorrhage" based on observations during the first world war of a team of workers which included such historic names as Cannon (1919) and Cowell (1919).

It is true that Blalock (1940) had developed the theory that shock was due to loss of fluid from the circulation, but a new stimulus was needed to shift the balance from the approach which Cannon had so ably presented.

Problem of Blood Loss in Shock

The old attitude is illustrated by Cannon when he poses the question of "the problem of the lost blood in shock." Everyone knew that shock was often associated with bleeding. It was widely known that studies of plasma volume showed that conditions of severe collapse following injury were associated with a severe fall in blood volume and at times an even greater disappearance of red cells, but for many years no reliance was placed on the evidence. Measurements of plasma volume were considered to be unreliable when it was assumed that plasma-like fluid was leaking from the circulation in shock and that this leak was not confined to the site of injury. Furthermore, it was almost univer-

sally assumed that the observed haemorrhage could not possibly account for the disappearance of blood. It was therefore argued that the missing blood must still be inside the circulation, not measurable by blood-volume techniques because it was no longer circulating. Evidence was produced to show that it *could not* be in the arteries and *was not* in the veins—therefore it *must* be in the capillaries.

In the light of our present knowledge we believe this to have been a fundamental error—but a very good case was made out. In certain clinical states which follow injury, with and without severe infection, particularly when death is imminent, the capillaries lose their tone and a patient can bleed to death into the capillary bed in spite of transfusion. The mistake was to conclude that at an earlier stage the fall in circulatory blood volume was due to capillary stasis. The objective of treatment was to mobilize the missing blood back into effective circulation: hence the heat and the friction.

It was known that patients with moderate injuries could partially restore their own blood volume by absorption of fluid from outside the circulation. When this process was slow or ineffective it was assumed, sometimes correctly, that the patient's fluid reserves were deficient. In any case, it appeared to be sometimes possible to increase the blood volume more rapidly by forcing fluids. In addition, severe states of shock were associated with acidosis, which was made worse by anaesthesia. Hence the hot sweet tea—hot as a "stimulant," sweet to provide glucose to combat acidosis, wet to provide fluid. Originally it was laced with alkalis, but these seem to have been left out during the years that followed.

A further fallacy underlies the older approach: we now know that, except in burns, in the occasional crush injury, and in abdominal injuries with peritonitis, the fluid lost from the circulation in the overwhelming majority of injuries is whole blood. For a time, and even well on into the period of the second world war, the emphasis was on the need to restore the blood volume when it was low. We are convinced to-day that this was only a second-best objective even when effective, and that the restoration of red cells can be almost as important as the restoration of total circulating volume.

Blood Banks and Transfusions

I shall return to the story of the missing blood, but first I must stress the fact that the older observations contained almost all the evidence, although the basic

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conception here presented did not receive any wide acceptance until the emergence of the modern blood bank with the possibility of transfusing large quantities of stored blood.

During the first world war transfusion was used in very small quantities. A litre (2 pints) was a large transfusion. It was the experience of the transformation effected by transfusion of much larger quantities of blood to patients with severe injuries that established the need for drastic revision of theory and the reassessment of old therapeutic practices. Blood banks were established in Moscow and Chicago by the early 'thirties. Durán-Jordá (1939) organized a modern transfusion service for the casualties in Barcelona during the Spanish Civil War. Blood banks were being developed in Britain during 1938 and 1939. The experience of the second world war led to their establishment as a normal part of our Health Service.

With stored blood available, transfusion came into its own. Early transfusion of large quantities of blood gave results that were clearly a major advance on all previous experience. Further investigations showed that the blood-volume methods were more reliable than had been suspected. New knowledge of blood groups and new techniques of cross-matching reduced the danger from incompatible transfusions. New methods of preparation and storage reduced other transfusion hazards, and blood can be stored for longer periods.

British, American, and Canadian research teams came to similar conclusions during the 1939-45 war, and by 1946 it was possible for some authorities to suggest that traumatic shock was nothing more than the effect of loss of blood or other fluid from the circulation. This was an over-simplification, but the new thinking required for the treatment of injuries at all stages must be based on the accumulating evidence that *the most important single factor underlying states of shock and leading to early death following injury is loss of whole blood by bleeding*.

Assessment of Extent of Bleeding

The most important clinical and research study which emerged from the second world war is the Grant and Reeve (1951) classic "Observations on the General Effects of Injury in Man," based on a study of 120 patients in England during the air raids and a further 110 patients seen in forward areas in Italy in 1944 and 1945. These authors emphasized the extent of haemorrhage and the role of early rapid transfusion, especially in "large" and "very large" wounds. They elaborated the possibility, particularly in limb injuries, of assessing the extent of blood loss from the nature of the injury. They concluded that the word "shock" was misleading and that existing theories were inadequate.

Observations at the Birmingham Accident Hospital (Clarke, 1952; Clarke, Topley, and Flear, 1955; Clarke and Fisher, 1956) have confirmed those of Grant and Reeve. They are particularly important, because we have been dealing with civilian casualties and have been in a position to carry out very much longer follow-up studies. The last 15 years, moreover, have seen the development of new methods for the determination of blood volume, using red cells labelled with radiophosphorus or radiochromium. These methods are not only more accurate than the plasma-volume techniques but they measure the red-cell loss directly. The radiochromium technique labels the red cells more permanently, so that their subsequent fate can be studied. In a large proportion of patients the life-span of transfused cells is similar to that of the patients' own erythrocytes. The fact that we now have a variety of techniques has meant that

we can cross-check one against the other and determine the blood volume at more frequent intervals. We are now satisfied that blood-volume studies carried out in the acute phase of injury and even during states of clinical shock can provide important information on the depletion of blood volume and the extent to which this is due to loss of whole blood. Compared with Grant and Reeve we would pay less attention to the blood pressure when it is normal and have had a greater experience in studying the effects of more adequate transfusions.

The first theoretical issue that we have been up against is the controversial question of "where all the blood goes to." From repeated blood-volume studies we have been able to show that when seriously injured patients are resuscitated by transfusion to clinical states resembling normality this change has never been associated with any apparent return of missing red cells. This alone might not be considered decisive, so we have concentrated attention on measuring or assessing the actual extent of bleeding.

We soon became aware that blood was missing from the circulation just as frequently in major closed fractures as it was in open wounds. The disappearance of blood appeared to be proportional to the extent of injury, and particularly to the size of the swelling. We therefore measured the amount of swelling in injured limbs soon after injury. The order of swelling frequently corresponds to the amount of blood missing from the circulation. From these measurements and a variety of clinical observations we are now in a position to make a more accurate clinical diagnosis, not only of the extent of blood loss already present but of that which is likely to occur later. The fact that swellings of a given order with recognizable surface appearance can conceal such extensive blood loss can be vividly illustrated by attempting to reconstruct such swellings, for which purpose we have used measured volumes of surgical felt (Clarke *et al.*, 1955; Clarke and Fisher, 1956).

In addition to investigating blood loss in closed fractures, we have studied blood loss from open wounds. During surgery, bleeding can be measured by swab-weighing. Bleeding between admission and surgery can be measured by collecting the blood in a "polythene" bag wrapped round the limb. Bleeding at the place of injury, on clothes, on stretchers, and on the floor is difficult to measure, but we have toned up our capacity to estimate such loss by photographing known amounts of blood distributed in clinically relevant forms. In addition photographs and clinical observations of a wide range of injuries have been correlated with blood volume and laboratory studies.

During the last four to five years we have carried out repeated studies of blood volume on over 100 patients suffering from non-thermal injuries, many of whom have had very detailed clinical and laboratory investigation.

Our general observations have been further confirmed by reports from the American research team in Korea (Prentice *et al.*, 1954). They had large quantities of blood available and were able to resuscitate patients with such severe injuries that they would certainly not have survived at an earlier period of history. Although some of these observers speculated on the fate of the blood lost, it is now generally agreed that frank haemorrhage from the surface or into the tissues is enough to explain the loss of blood and that there is no fundamental mystery. *We are now convinced that the blood loss observed in traumatic shock is simply due to haemorrhage—to loss of whole blood, including red cells, irretrievably from the circulation as a result of direct injury to blood vessels and not due to pooling or stasis in the capillaries.*

Indications for Transfusion

The recognition that blood loss following injury has in the past been repeatedly underestimated has led not just to a policy of giving larger transfusions earlier and with greater speed, but to a careful attempt to establish a more accurate approach to transfusion indications at various stages. At

the same time we have had an opportunity to review other methods of treatment in the light of the effects of transfusion.

We are now convinced that any patient who has lost more than 2-4 pints (1-2 litres) of blood, even when this has taken place slowly and when no early surgery is required, warrants consideration for blood replacement by transfusion. When such a patient has to be submitted to surgery and anaesthesia, or when further bleeding is likely to occur into dressings or into the injured part, transfusion is more definitely indicated. In the presence of more severe blood loss, large transfusions are indicated at an early stage, and in very severe injuries we are using transfusions which frequently exceed the patient's total blood volume.

As a result of our experience with larger primary transfusions we are convinced that blood replacement plays a vital part not only in the treatment and prevention of "shock" but in allowing more adequate primary surgery and in influencing the whole pattern of illness following injury so that convalescence is greatly shortened. We have carried out detailed haematological and biochemical studies which begin to suggest how undertransfusion can lead to illness, and adequate transfusion to its reversal or cure. At a later stage transfusion is invaluable in the treatment of infected wounds. Adequate primary transfusion plays a part in the prevention of wound infection and delayed healing.

In summary, blood transfusion is indicated when blood loss has occurred after injury: (1) for the treatment of established traumatic shock; (2) for the prevention of shock and other complications of a low blood volume and red-cell mass; (3) for the facilitation of adequate primary surgical repair, whether this be life-saving, for the arrest of haemorrhage or the prevention of infection, or merely to limit disability following injury to various tissues from bone to skin; and (4) for the prevention of many features of the later illness of trauma.

Dangers of Delay

Our attitude to transfusions as the primary weapon in the prevention and treatment of complications of injury has altered our point of view on the timing of other methods of treatment. When transfusion is not indicated or possible, and in the presence of injuries that do not require surgery, a policy of expectancy may be in order. It may be justifiable to wait during the course of establishing a detailed diagnosis or for accurate cross-matching so that transfusion can be safe. In moderate injuries there is rarely *any* indication for delay *before* the patient is transferred to expert medical care. In more severe injuries even without overt shock, a policy of delay without the facilities for diagnosis and transfusion is dangerous. Even when the patient appears fit, processes are taking place in connexion with moderate or severe injuries that tend towards deterioration rather than recovery. A large proportion of injured patients will begin their recovery only when transfusion and surgery have been carried out. Time is not on our side until a detailed diagnosis and assessment has been made and treatment planned and begun.

During recent years we have had considerable experience of the result of delay in transferring patients from outlying hospitals. Our experience corroborates the observations of Grant and Reeve. Patients with moderate and severe injuries do not benefit by an hour's rest which is associated with an hour's delay in carrying out effective treatment. When it was imagined that the fall of blood volume was not due to actual loss of blood one can appreciate the focusing of attention on rest. The patients that recovered did so because they were likely to improve anyway, but too many did not recover at all. A patient who has lost 25-40% of his blood volume from bleeding to the outside or into the tissues, even if he looks fairly fit, is in grave danger of major complications, particularly renal failure, if his blood volume is allowed to remain low for two to three hours. Some of our most successful experiences with severe injuries have been with patients transferred very early, or, when this has been

impossible, when the surgical team has been transported to the patient.

I remember in particular a boy of 18 injured in the Sutton Coldfield train smash. Both legs were torn to shreds from the lower thigh downwards and he had a fractured pelvis which required abdominal exploration to exclude a lesion of the gut. He was put straight in an ambulance at the site of the incident and reached the Birmingham Accident Hospital, 15 miles away, about 45 minutes later. He was in a state of profound haemorrhagic shock, but with the aid of transfusion of 11 bottles of blood, equal to his total blood volume, he survived early bilateral amputation and abdominal exploration and proceeded to make an uneventful recovery. Half an hour's delay without full transfusion might well have been fatal.

Some Traditions

The next orthodoxy which I wish to discuss is the tradition of hot sweet tea. Except when a very long delay is inevitable before full medical attention can be reached, the amount of good that can be done by giving oral fluid is negligible. The real objection to ill-advised administration of fluids or solids to injured patients is the risk that this may lead to vomiting under anaesthesia, one of the common causes of anaesthetic disaster in patients requiring emergency operations. When transfusion is needed, fluids by mouth are unnecessary. Quite apart from abdominal injuries any patient with an injury that might conceivably require surgical treatment under anaesthesia, and this includes many minor wounds and fractures, should have nothing by mouth whatever until a medical decision has been taken that no early operation is necessary. The only exception is that patients likely to be longer than two to three hours before reaching hospital may be given sips of water to allay their thirst. I remember the full operation of the older policy during my first few days in a casualty clearing station in North Africa. The hot sweet tea was given by the reception staff followed by large white tablets to prevent infection, and milk chocolate by the padres. The result was so dramatic that the routine was soon altered. Hot sweet tea is required only for patients who have not been significantly injured. Friction is not indicated at all and smelling-salts should be relegated to the Victorian drama.

More complex is the question of the use of heat in the treatment of the injured. It is perhaps logical from a lay point of view to heat anyone who feels cold. In fact, a patient who is cold and pale from a shut-down of the surface blood supply has insulated himself against heat loss. If he is shivering he is creating heat. The patient whose skin feels cold to the touch and who complains of feeling cold may have a raised internal temperature. The patient who is cold because he has lost a moderate amount of blood will warm up spontaneously unless exposed to the elements. Heating the surface of a patient who has lost a moderate amount of blood may open up the blood supply to the skin and side-track blood from circulation through more essential organs. The patient who is shocked or in a state of lowered consciousness following a head injury is liable to be burnt by the use of hot cradles or hot-water bottles even when the latter are supposed to be applied outside blankets. Finally, the skin appearance and temperature are valuable clues to the state of the circulation in the body as a whole and of an injured limb in particular. The use of heat confuses the evidence. In general there is no place whatever for hot-water bottles or hot cradles in the treatment of injured patients. The more serious the injury the less need there is for blankets. Blankets should be used for (1) the comfort of the patient with a minor injury, and (2) in cold weather for the protection of the patient with moderate injuries.

The head-down position is valuable when the pulse is thin or absent and it appears that consciousness has been interfered with as a result of severe blood loss. It is necessary only in severe conditions.

Splintage is important but needs to be simple. When a patient is to be transported a short distance and will reach hospital within half an hour of injury, it is not justified to take 20 minutes to apply a Thomas splint when tying the legs together will do just as much good.

Morphine and allied drugs are sometimes of value in the presence of pain, distress, and major anxiety. In general, however, the more severe injuries do not give rise to great pain except on movement of injured parts. Such drugs must never be used as treatment for "shock." Large doses of morphine, particularly when repeated subcutaneously in the presence of a sluggish circulation, are dangerous.

Shock: A Complex of Reactions

The main lesson I would suggest from the point of view of first aid is that there is *no treatment for shock* as such, and that in this country rapid evacuation to hospital is the decisive objective. Before concluding with some concrete proposals on the formulation of first-aid principles in this connexion I would like to deal further with the theoretical question of the nature of the complex of reactions commonly known as "shock." The best early description I can find is that of the famous sixteenth-century French surgeon Ambroise Paré. It appears in Johnson's translation as "sowning":

"Sowning is a sodain and pertinacious defect of all powers, but especially the vitall; in this the patients lie without motion and sense, so that the Ancients thought that it differed from death only in continuance of time. The cause of sowning which happens to those that are wounded is *bleeding*, which causeth a dissipation of the spirits: or *fear*, which causeth a sodain and joint retirement of the spirits to the heart. . . . Also sowning happens by a putrid and venenate vapour, carried to the heart by the arteries, and to the brain by the nerves; by which you may gather, that all sowning happens by three causes. The first is, by dissipation of the spirits and native heat, as in great bleeding. And then by the oppression of these spirits by obstruction, or compression, as in fear or tumult. . . . Lastly, by corruption, as in bodies filled with humours, and in poysonous wounds. The signs of sowning are paleness, a dewy and sodain sweat arising, the failing of the pulse, a sodain falling of the body upon the ground without sense and motion, a coldness possessing the whole body, so that the patient may seem rather dead, than alive. For many of these who fall into a sown die unless they have present help."

Since Paré's day there has been a continuing battle between the advocates of the three main theories—that shock is due to haemorrhage, nervous action, or toxins. To deal with the last category first, it is true that a state indistinguishable from the shock we are talking about can result from severe infection following injury, particularly from gas gangrene or peritonitis. Even in these conditions there is probably some blood loss and widespread extravasation of fluid. A low blood volume plays a major part in the clinical condition, which can frequently be reversed by a combination of chemotherapy, surgery, and intravenous transfusion of blood or other fluids. In the terminal stages it is perhaps true to say that the capillary bed is paralysed by toxins whose full nature has yet to be elucidated. From the point of view of first aid such conditions are unlikely to develop within four hours of injury, and the answer is still rapid evacuation to hospital.

We are left with the effects of haemorrhage and loss of fluid from the circulation and with the effects of reflex nervous action. In the absence of significant local or general injury, a transient state of collapse, with pale, cold extremities, a feeble pulse which may be slow, a very low blood pressure, nausea, vomiting, sweating, and loss of consciousness, may result from reflex nervous action resulting in redistribution of blood within the circulation. In the presence of bleeding this response may be of value to the patient in that the blood pressure falls and arrest of bleeding is facilitated (Edholm, 1952; Barcroft, 1957). But the responses can happen without any bleeding at all from the fright of the injury and the events associated with it. from a

needle-prick, the sight of blood, or even the sight of a needle-prick in a film or on television. The thought of blood is almost enough in some people to induce this type of faint or vasovagal attack. The blackout may be of secondary benefit to the patient in that the full impact of unpleasant events is obliterated from consciousness.

It may be possible that we are dealing here with a basic unconditioned type of reflex response to a falling blood volume which in the course of evolution and conditioning can now be fired off in man by a variety of stimuli not infrequently associated with haemorrhage. Such responses may possibly acquire new significance in the course of this evolutionary emergence. The primitive "fight or flight" response of Cannon (1923) may not be the only basic pattern of primary response to injury. Cowell's (1919) "primary shock" may vary greatly in its prognostic significance, but is a clinical state that still happens and requires explanation. The stimulating hypothesis of Slome and O'Shaughnessy (1938) that the primary reaction was due to fluid loss and the secondary collapse had its nervous component, warrants renewed consideration at a higher level of complexity and in the light of further evidence from the human subject now that we can measure the degree of oligæmia.

The real difficulty is that it is often clinically *impossible* to distinguish this reaction from true haemorrhagic shock, and that this type of response may be the first overt reaction of the organism to a serious injury with severe bleeding.

The shock reactions which result from bleeding or loss of fluid may in the first place be precisely similar to the classical neurogenic response, but may pass over to another type of circulatory response. This is characterized by constriction of blood vessels tending to keep the blood pressure raised as opposed to the dilatation of deep blood vessels especially in muscles, which has been demonstrated in the first type of response. All the patterns of redistribution of the circulation which can follow soon after injury are operated through the nervous system by reflex action, so that once there is significant injury of any kind it is no longer a question of this or that type of shock—it is a question of a particular pattern of nervous reaction fired off by particular combinations of causative factors, of which haemorrhage is likely to be the most important. The nervous response determines the reaction of the circulatory system.

While some of the early patterns of so-called circulatory failure following injury are essentially beneficial to the organism, the continued operation of the underlying processes, especially when associated with further bleeding, may change their fundamental character for the worse without obvious external change. The continued operation of a beneficial type of response beyond a given period of time may lead to dangerous complications. This is particularly so when the shock reaction involves vasoconstriction. The blood pressure may be maintained, but the blood flow through essential organs can be so reduced that their subsequent failure is inevitable. Partial resuscitation may produce an apparent improvement, but the patient dies from interference with the functions of such essential organs as the kidney, liver, heart, lungs, and brain.

A considerable amount of recent medical research and literature has focused attention on the endocrine glands, particularly the suprarenal cortex and the pituitary. These organs undoubtedly play a major part in producing the total reaction of the patient, but their activity is intimately linked with that of the nervous system, and theories which attempt to attribute the whole "stress reaction" (Selye, 1950) to action of these glands are, to say the least, one-sided.

A further new concept has emerged in recent years. It is suggested that following injury or other severe disease the patient may be suffering chiefly from harmful excessive reactions which can be modified by transforming the total reactivity of the organism. This can be done by cooling or by the use of such drugs as chlorpromazine and the "lytic cocktail" or by these combined with cooling (Laborit, 1952; Laborit and Huguenard, 1954). The whole question is

complex, and I think it fair to say that at present there is not enough evidence to warrant the use of cooling or hibernation in the treatment of severe injuries, with the possible exception of certain head injuries. Further elucidation of the detailed responses of the organism to injury may allow us to replace heating by cooling as a definitive approach to treatment, but this belongs to the future.

Suggestions for Teaching of First Aid

To return to the point from which we started, the teaching of first aid and the content of the first-aid textbooks. The full report of Grant and Reeve did not appear until 1951, six years after their research had been completed. It is then hardly fair to criticize the fact that the last edition of the *St. John First Aid to the Injured*, which was revised in 1950, reflects a position intermediate between the teachings derived from the first world war and those which are now in process of emerging from more recent clinical and research observations.

The time is now ripe for the incorporation of these advances and their implications into the teaching of first aid, and with this in mind I shall conclude with suggestions for the section on "Shock" in the *St. John* textbook for consideration by those responsible for its revision.

SHOCK

Shock is a state of collapse following injury or disease. It is characterized essentially by changes in the circulatory system. Its severity varies with the nature and extent of injury. It may develop early or late. It is a common cause of death following major injury. Prompt efficient hospital treatment is frequently of the utmost importance.

The most important cause of shock is loss of blood or other fluid from the circulation. In most injuries other than burns, severe shock within a few hours of injury is due to bleeding to the outside or into tissues or body cavities.

TYPES OF SHOCK

There are two types of early shock following injury which may be clinically indistinguishable. The one may follow the other with or without an interval. Nerve shock is a reflex nervous response not necessarily associated with any reduction in the volume of circulating blood. The other type of shock is associated with a reduced blood volume from bleeding or other fluid loss. In addition, any patient with a moderate or severe injury must be regarded as in a state of latent or concealed shock. The blood volume is reduced with little evident disturbance of the circulation. Obvious clinical shock can be precipitated by movement, further bleeding, emotional stimuli, surgery, and anaesthesia, or may develop with the passage of time.

Toxic shock may complicate the development of infection following severe injuries to the limbs or trunk. It is rarely of significance within four hours of injury and requires immediate transfer to hospital.

NERVE SHOCK

All forms of shock involve reflex nervous action, but it is possible to distinguish a type of early nerve shock associated with minor injury which results from purely nervous or psychological factors.

Signs and Symptoms.—These may range from a faint or "vasovagal" attack with little evident circulatory change except a slow pulse accompanying transient loss of consciousness to a state of severe collapse with extreme pallor, coldness of the extremities, a feeble pulse which may be undetectable, sweating, vomiting, and unconsciousness.

Course.—In the absence of serious injury or bleeding this condition will pass off within a few seconds or minutes. In the presence of significant injury and bleeding it may pass imperceptibly into a state of severe shock or may recur following movement or further bleeding. It can be diagnosed with confidence only when recovery is rapid and it is clear that the injury is trivial.

Treatment.—Lay the patient flat on the floor, stretcher, or couch. Reassure when conscious. Examine for evidence of significant injury. Cover with a blanket but do not heat. If recovery does not take place rapidly, medical care must be obtained urgently or the patient sent to hospital. When the

patient is conscious and it is clear that no operation will be necessary, he may be given a drink of tea or whatever other drink is available or preferred.

LATENT SHOCK

The majority of patients with more than trivial injuries are liable to develop some form of shock. This may appear to be due to pain or movement, but is always more likely in the presence of blood loss. The extent of bleeding distinguishes certain types of injury and makes them more likely to develop late collapse or a persisting state of shock.

A patient with a single fracture of the femur can lose 20-30% of the blood volume (2-4 pints) into the thigh without a wound. A patient with a fracture of the pelvis can lose much more. In compound fractures of the tibia and fibula 15-20% of the total blood can be lost. In multiple injuries each individual injury contributes its quota of bleeding. The risk of collapse depends on the amount and rapidity of such bleeding.

The patient with suspected major fractures, severe wounds, multiple minor injuries, or significant trunk injuries can lose enough blood to cause severe dangerous shock. For a time the patient can compensate for the blood loss, but his situation becomes more critical with the passage of time unless the blood volume is restored to a near normal level by transfusion. When surgery is urgently necessary the risk increases with every delay.

Signs and Symptoms.—Patients with latent or compensated shock may be pale with cold extremities, they may be warm and pink. They may have a rapid or slow pulse, a raised or lowered blood pressure. They may be anxious and in pain or listless and semi-stuporous. They may be thirsty, sweating, or vomiting, or *apparently perfectly fit*. The severity of this condition depends on (1) the extent of blood or fluid loss, (2) the extent and nature of the injury, and (3) the time that has elapsed since injury.

Treatment.—(1) Move as little as possible, and that gently but swiftly. Wounds must be bandaged and bleeding controlled. Major fractures must be splinted in the simplest way consistent with efficiency and speed. (2) Give nothing by mouth to any patient who may require an operation later unless there is an unavoidable delay of several hours before reaching hospital. (3) The patient may be covered with a blanket, but in warm weather care must be taken not to increase sweating. Hot-water bottles and hot cradles are dangerous. The unconscious patient requires no heat by way of first-aid treatment. (4) *Evacuate to hospital as quickly as possible.*

ESTABLISHED SHOCK

This is characterized by extreme pallor and coldness of the extremities, sometimes an ashen-grey colour, a feeble pulse which may be very rapid. The blood pressure is low and may be unobtainable. This dangerous condition may be associated with obvious major injuries, but is none the less dangerous when the injuries are deep and blood loss is not evident.

Treatment.—There is no first-aid treatment for shock. When the journey to hospital is likely to be short—for example, less than 45 minutes—transfer immediately to hospital with the minimum of movement except transfer to a stretcher. It is unwise to delay for as much as five minutes for any form of first-aid treatment, except for (1) severe wound bleeding, (2) a sucking chest wound, (3) obstruction to the respiratory passages, or (4) failure of respiration. In any case delay may be fatal. Heat is not needed. Friction is contraindicated. Rapid, firm bandaging of injured limbs may limit further blood loss. The minimum of effective splintage is required. Morphine is valuable for severe pain, but requires the presence of a doctor. The stretcher should be tilted so that the level of the head is lower than the rest of the body. When rapid evacuation is impossible, careful splintage is of great importance, and fluids may be given if the patient is conscious and not vomiting. Overheating is dangerous.

Such patients may recover only with major transfusion and surgery. The severity of the apparent state of collapse makes such treatment a matter of grave urgency.

Conclusion

Finally I must refer briefly to two matters of great importance. Firstly, what are the dangers of blood transfusion? With a 24-hour laboratory service and a well-organized transfusion service the risks of transfusion need be no greater than the risks of anaesthesia for a minor operation in the hands of a trained anaesthetist. Infinitely greater harm is done to-day by inadequate transfusion. There remain

dangers from transfusion at the wrong time and the wrong speed, and we are concerned with learning how the judgment necessary to reduce these risks can be acquired and taught.

The second question is that of supplies. In some parts of the world there are difficulties, but in Britain there are big reserves of voluntary donors yet untapped. The difficulties are organizational. If blood is needed it can be obtained. As for cost: what is it worth to save a life or a hundred lives? Certainly with full modern resuscitation we can do more than save lives—particularly when our injured patients get to hospital quickly. Modern transfusion allows the latest developments in plastic and orthopaedic surgery to be applied at an earlier stage. The lessons we have learnt at the Birmingham Accident Hospital must be shared more widely and especially with our first-line allies—the first-aid workers—so well typified by the thousands who have been trained by the surgeons of the St. John Ambulance Brigade.

Summary

Traditional teaching on the first-aid treatment of "shock" is criticized. The older practices are traced to their theoretical origin in observations based on the experience of the first world war.

First-aid teaching needs to be revised on the basis of a recognition of the role of blood transfusion. This in turn must be based on an understanding of the extent of blood loss. Further evidence has been produced to show that blood loss is as important in civilian injuries as in battle casualties. Modern transfusion has been made possible by the development of blood banks. Modern techniques for the investigation of blood volume have helped the development of clinical aids to the assessment of blood loss and transfusion indications.

An appreciation of the role of transfusion in the prevention and treatment of shock requires reconsideration of the need for early diagnosis and treatment, with early transfer to hospital as a basis of first aid for moderate and major injuries. Fluids by mouth are rarely indicated in first aid. The disadvantages of heat treatment are discussed.

The significance of "primary shock" is discussed, and it is suggested that many forms of early circulatory change following injury are not necessarily harmful unless associated with loss of blood or other fluid from the circulation.

Suggestions are made for revision of the section on "Shock" in the St. John manual *First Aid to the Injured*.

Brief reference is made to the dangers of transfusion and the possibilities of obtaining adequate supplies of stored blood.

REFERENCES

- Barcroft, H. (1957). *Fainting*. *Triangle*, 3, 53.
 Blalock, A. (1940). *Principles of Surgical Care, Shock, and Other Problems*. Kimpton, London.
 Cannon, W. B. (1919). *Med. Res. Comm. Spec. Rep.*, No. 25, p. 109.
 — (1923). *Traumatic Shock*. Appleton, Philadelphia.
 Clarke, R. (1952). *Ann. roy. Coll. Surg. Engl.*, 11, 87.
 — and Fisher, M. R. (1956). *Brit. J. clin. Pract.*, 10, 746.
 — Topley, E., and Flear, C. T. G. (1955). *Lancet*, 1, 629.
 Cowell, E. M. (1919). *Med. Res. Comm. Spec. Rep.*, No. 25, p. 99.
 Durán-Jordá, F. (1939). *Lancet*, 1, 773.
 Edholm, O. G. (1952). In *Ciba Foundation Symposium on the Visceral Circulation*. Churchill, London.
 Grant, R. T., and Reeve, E. B. (1951). *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 277.
 Laborit, H. (1952). *Réaction Organique à l'Aggression et Choc*. Masson, Paris.
 — and Huguenard, P. (1954). *Pratique de l'Hibernothérapie en Chirurgie et en Médecine*. Masson, Paris.
 Prentice, T. C., Olney, J. M., jun., Artz, C. P., and Howard, J. M. (1954). *Surg. Gynec. Obstet.*, 99, 542.
 Selye, H. (1950). *The Physiology and Pathology of Exposure to Stress*. Montreal.
 Slome, D., and O'Shaughnessy, L. (1938). *Brit. J. Surg.*, 25, 900.

SYSTEMIC LUPUS ERYTHEMATOSUS*

BY

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Arthritis and Arthralgia

Joint involvement was the commonest clinical feature in this series. Of 49 patients, 45 showed joint symptoms at some time. In 28 the first evidence of systemic lupus was a clinical picture indistinguishable from rheumatoid arthritis; a further 10 began their illness with vague joint symptoms, often monarticular or asymmetrical.

TABLE V.—Incidence of Clinical Features in 49 Cases

Albuminuria ..	38	Splenomegaly ..	14
Rheumatoid arthritis ..	34	Adenopathy ..	14
Secondary infection ..	28	Arthralgia or atypical arthritis ..	12
Respiratory ..	11	Raynaud's phenomenon ..	7
Other ..	17	Pericarditis ..	7
Rash ..	24	Hypertension ..	4
Nervous system ..	23	Gastro-intestinal ..	4
Psychiatric ..	13	Myalgia and vague pains ..	3
Epilepsy ..	5	Blood dyscrasia ..	3
Neuropathy ..	5	Alopecia ..	2
Pleurisy ± fluid ..	19		
Hepatomegaly ..	16		

The high incidence of arthropathy (Table V) in these cases might reasonably be thought to reflect a special interest in the rheumatic diseases; it is in fact little greater than that reported by many workers who have not this special interest. Harvey *et al.* (1944), for example, record arthritis or arthralgia in 95 of 105 cases; in 66 one or other was the presenting symptom. Similar figures were reported by Dubois (1956), 90%, Jessar *et al.* (1953), 77%, and Shearn and Pirofsky (1952), 85%.

Joint symptoms fall broadly into three main types—arthralgia, migratory polyarthritis, and chronic arthritis of the rheumatoid type.

Arthralgia and Myalgia

The aching pain in the muscles or around the joints is usually described as "fibrositis"; it may be migratory and accompanied by transient swelling and tenderness over a few joints. This muscular pain and tenderness is occasionally the predominating symptom, as in the following case.

Case 1.—A woman of 37 first noted stiffness, swelling, and slight pain in her fingers eight months before admission to hospital, followed in two months by tenderness of the thigh and upper arm muscles. Later, all her muscles became so tender and painful on movement that a diagnosis of dermatomyositis was considered. The facial rash and L.E. cells, however, suggested the true diagnosis, which was confirmed at necropsy two weeks later.

In my experience, arthralgia or myalgia as a first symptom seems to be of unfavourable significance. Of 10 patients presenting in this way, three died within two and a half years, two were in very precarious health about two years after the onset, and three others were only kept in partial remission by steroid therapy. Some of Kersley's (1951) cases of senile myalgia may belong to this group. He described a syndrome of muscle pain with systemic reaction in elderly patients, predominantly women. Their symptoms were unusually persistent and refractory to conservative therapy but responded promptly to cortisone. Bagratuni (1956) has reported a similar series.

Migratory Polyarthritis

This is easily confused with rheumatic fever, especially as it seems to occur mainly in younger patients. The incomplete response to salicylates may suggest the diagnosis.

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