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THE THERAPY OF SHOCK

SOME LESSONS FROM MILITARY SURGERY

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The state of peripheral circulatory failure, referred to as "shock," is so frequently associated with the extensive injuries incurred during military operations, it may properly be called a malady of warfare. Not only is it encountered more frequently, but its severity far exceeds that ordinarily seen in civilian practice. It is thus possible to draw from the experiences of military surgery many points of practical value in the treatment of shock.

Because the term "shock" has been used quite loosely to indicate any form of peripheral circulatory collapse, this discussion will be prefaced by a brief definition and classification. Shock is defined as a "state of actual, or impending, peripheral circulatory failure due to a reduction of the effective blood volume not primarily cardiac in origin." This excludes from discussion all forms of peripheral circulatory collapse in which failure of the heart is the primary cause. Depending upon the mechanisms involved, three types of shock may be recognised: *neurogenic*, a state of sudden loss of vasomotor tone with a redistribution and pooling of blood in dependent portions of the body; *vasogenic*, a consequence of direct injury to capillaries or small vessels; and *hematogenic*, the result of a primary loss of fluid from the circulation.⁴ The term "decompensated shock" is used to indicate the state of advanced collapse which follows a prolonged reduction of the effective blood volume, irrespective of the original cause or causes.

NEUROGENIC SHOCK

Neurogenic shock is a condition in which the total volume of blood is unaltered, but is ineffective because of a loss of constrictor tone, with a redistribution and pooling of blood in the lower portions of the body. It is manifested by pallor, sweating, a lowered blood pressure, and a slow pulse rate. It is usually a consequence of severe pain, fright, or other emotional disturbance, but may be associated with hematogenic shock. The syncope observed in blood donors is a familiar example. It is of interest that in severe hemorrhage this neurogenic reaction is mediated by vasomotor nerves, and occurs before the cardiac output is decreased.²

An understanding of the characteristics of neurogenic shock is of the greatest importance because, its occurrence in conjunction with the other forms of shock, makes an accurate appraisal of the therapeutic needs of a patient more difficult. Thus, on the one hand, blood or plasma may be needlessly given the patient who has fainted with a minor injury, and, on the other hand, recovery from neurogenic shock may temporarily obscure the fact that a concealed hemorrhage is actually progressing.

VASOGENIC SHOCK

The term "vasogenic shock" is used to include a number of factors such as infection, anaesthetic agents, or hypothetical toxins which, although the exact mechanism of their action is not known, are recognised as contributing to the circulatory failure of shock. Whether these agents cause a specific capillary injury, or a generalised loss of vascular tone associated with cellular necrosis throughout the body, is not yet established. The latter possibility seems more likely since progressive hemo-concentration is not a feature of severe infection, unless there is associated dehydration.⁷ From the practical point of view the precise mechanism is unimportant. It is important, however, to realise that specific measures to prevent or treat infection are of more value than measures taken to improve the circulatory failure which it produces.^{1, 7}

HEMATOGENIC SHOCK

Hematogenic shock is a consequence of a loss of fluid from the circulation. This may occur as blood, plasma, water and electrolytes, or any combination of these. It may be obvious, as in external bleeding or vomiting or, as is more often the case, it may be concealed as in bleeding into a body cavity or into tissue spaces. Irrespective of the type of fluid lost from the blood stream, two compensatory phenomena occur: the movement of available extra-cellular tissue fluid into the circulation, and peripheral vasoconstriction. These compensatory mechanisms may be sufficient to maintain the blood pressure despite a continued reduction of the blood volume, a fact of the greatest practical importance to the clinician.

DECOMPENSATED SHOCK

Decompensated shock is advanced or late shock. Practically speaking, it is the result of untreated hematogenic shock, and is often complicated by sepsis, anesthetic agents, exposure to cold, and variable neurogenic factors. Decompensated

shock* is characterised by low arterial pressure, diminished cardiac output, low blood volume, a decrease in the total circulating plasma protein, acidosis, and anoxia. If untreated, decompensated shock becomes irreversible in the sense that an adequate restoration of the blood volume has no effect upon the circulatory collapse. The nature of this "irreversible state" is not known. It has been thought that either as a consequence of "toxins" absorbed from the area of injury or prolonged anoxia, a generalised permeability of capillaries develops. That this may occur in the terminal stages seems likely, but there is increasing evidence to indicate that before the capillaries are injured a generalised "cellular" death occurs, particularly in highly specialised cells in the liver, adrenals, kidneys, and gastro-intestinal tract. Apparently the heart itself is affected for, even if the blood volume is restored to normal and the venous pressure raised by rapid infusions, the circulation fails to improve.

An example may serve to clarify precisely what is meant by each of these types of shock: a man sustains a perforating gunshot wound of the buttock and abdominal cavity. He falls to the ground, experiences moderate pain, realises he has been badly wounded, fears he may die, and passes into a state of collapse. If examined at this time he will be pale, sweating, and will have a low blood pressure. However, his cardiac output and blood volume if determined would prove to be normal. This is neurogenic shock. The man is picked up, and some two hours later arrives at an Aid Station. He has received a large dose of morphine, his pain is relieved, and his worst fear—that of dying neglected on the battlefield—has been proven groundless. He smokes a cigarette and begins to feel more cheerful. At this time he will no longer be sweating, and as a consequence of compensatory vasoconstriction his blood pressure will be normal, or even a little above normal, and his pulse rate may not exceed 100. Yet, by now he has lost 1,000 c.c. of blood—most of it "concealed"—and he shows evidence of early peritonitis. This man is now in hemato-genic shock. Before he is transported to the Field Hospital, replacement therapy with blood or plasma should be instituted.

Let us assume that no therapy is given. The man is transported another five miles to the rear, he is given another dose of morphine because of a recurrence of his pain, and he is exposed to cold and is badly shaken up in transport. Some six hours after injury he arrives at a Field Hospital. Examination will again show him to be in obvious collapse; he is pale, his face and extremities are cold, and his pulse is weak and thready. Despite continued vasoconstriction as evidenced by cold, white

extremities, the blood pressure will now be low. If determined, the blood volume would be found reduced by as much as half its original total. This is decompensated shock. If treated promptly by adequate replacement of the lost fluid, and if the therapy is continued throughout the necessary operative procedures, this patient may yet recover. However, if further delay ensues and peritoneal or retroperitoneal infection becomes established, or if therapy is inadequate and a general anaesthetic is given, there will develop a state of circulatory collapse which will no longer respond to the infusion of blood or plasma. Even though the blood volume is restored to normal the blood pressure will remain low and the extremities cold and nearly pulseless. This is an "irreversible state."

DIAGNOSIS AND TREATMENT

It has been emphasised repeatedly that the most important evidence of shock is obtained from an evaluation of the clinical findings.^{5, 6, 8, 10} This view has been confirmed by recent experiences in the campaign in Normandy. The inexperienced medical officer is lost when first confronted with even a single patient in profound collapse. Whereas an orderly and logical appraisal of each case, using clinical findings as the principal criteria for diagnosis and treatment, allows an experienced officer, with one or two enlisted men as assistants, to care for an entire ward full of patients in shock. The following "standard operating procedure" for the reception and management of the patient in circulatory collapse was established for this unit over two years ago. Although certain aspects of it may appear exceedingly elementary, it is presented here in detail as a guide for those who have had little or no experience with this problem.

When confronted with a patient in circulatory collapse the first, and most important, thing to do is to determine as nearly as possible under the existing circumstances the exact nature of the injuries. Usually sufficient history to provide the background for an immediate examination can be obtained in a word or two, such as "Jeep accident," or from a glance at the "Emergency Medical Tag," such as "Shell wound of chest and abdomen." A preliminary examination is made as follows:—

1. Determine the state of consciousness of the patient and ascertain whether or not he has any recollection of the accident—thus, in a second, including or excluding a cerebral injury.
2. Inspect and palpate the skull swiftly, but gently, for laceration, contusion, or depression; note the colour of the ears and lips for pallor or cyanosis.

3. Inspect the chest, looking particularly for a sucking wound; then compress the chest gently for evidence of fractured ribs. Auscultate the chest anteriorly, but do not do a detailed examination unless there are findings indicating a tension pneumothorax or cardiac tamponade which require urgent therapy.
4. Palpate the abdomen, merely to exclude tenderness and spasm.
5. Gently compress the wings of the ilium and palpate the symphysis pubis for evidence of pelvic fractures.
6. Feel and gently move, or have the patient move, all his extremities to exclude major fractures.
7. Note the colour and temperature of the hands and feet.
8. Finally, gently turn the patient just enough to slide a hand under the perineum, buttocks and back to exclude major wounds with continued and unrecognised hemorrhage.

While the surgeon is making this survey of the extent of the injuries, trained corpsmen should be preparing a plasma infusion, taking and recording on a graphic chart the pulse rate, respiration rate, blood pressure, and rectal temperature, and assisting in the judicious removal or cutting away of clothing, as indicated by the surgeon. Thus, at the conclusion of the examination everything is in readiness for treatment. It is usually wise to begin with an initial plasma infusion, at which time a sample of blood should be taken for determination of the serum protein and hematocrit. Thus, within a matter of minutes the case is evaluated, treatment is started, and the basis established for continuous therapy as indicated by changes in the chart or laboratory data. The following case reports are examples:—

1. *Truck Accident.*—Patient semi-comatose; no demonstrable injuries of chest, abdomen, or extremities. There is a small contusion of the scalp. BP 90/70; pulse 110; respiration 10. Impression: Head injury; withhold infusions, proceed with detailed neurological examination and appropriate therapy.
2. *Patient Run Over by Half-track.*—History of short period of unconsciousness. Complains of severe abdominal pain; laboured respiration; chest negative. Cold, white extremities. Abdomen spastic and tender, especially in left upper quadrant. BP 50/0, pulse 140, respiration 24. Impression: Hematogenic shock due to abdominal injuries; possible rupture of spleen with intraperitoneal haemorrhage and probably retroperitoneal haemorrhage. Start infusion at once—plan to give

at least 1,000 c.c. of blood. Subsequent course to be guided by response to infusion and result of more detailed appraisal of the abdomen. The presence of uncontrolled haemorrhage will make prompt surgery necessary. As the shock is being treated prepare for surgery.

3. *Gunshot Wound of Arm.*—Severe pain; patient pale, but extremities are warm. Examination discloses no other injury. No evidence of loss of blood from history or character of wound. BP 90/50, pulse 90, respiration 20. Impression: Neurogenic shock—withhold infusion; give morphine and warm drinks.
4. *Gunshot Wound of Chest.*—Marked dyspnea; chest shows shift of mediastinum to opposite side with tympany and absent breath sounds over the injured side; no other evidence of injury. BP 90/50, pulse 120, respiration 30. Impression: Tension pneumothorax. Insert flutter valve. Start infusion because some blood loss is inevitable. Watch carefully for signs of intrapleural bleeding as evidenced by increasing dullness over affected side. Keep in mind the fact that once the pneumothorax is relieved the patient can bleed to death into his pleural cavity without signs of respiratory embarrassment.
5. *Severe Burns of Trunk and Legs.*—BP 140/60, pulse 100, respiration 20. Despite apparently normal circulation, begin plasma infusion at once. Plan to give approximately 500 c.c. of plasma for each 10 per cent of body area burned. As soon as possible, apply pressure dressings to limit loss of plasma. Subsequent course to be guided by response to therapy and result of hematocrit and serum protein changes.

The type and amount of replacement fluid necessary is determined by: (1) the nature of the injury; (2) the response to therapy; and (3) the data provided by determination of the hematocrit and serum protein. In general, except for burns and peritonitis uncomplicated by hemorrhage, whole blood will be required in every case. Plasma is no substitute for blood if the losses are large, and particularly if extensive surgery must be done under a general anesthetic. If an infusion is needed at all, 1,000 c.c. represents a minimal requirement. If the injury is an extensive one, and particularly if the blood pressure is below 70 mm. systolic, at least double this amount will be required. The loss of blood, both externally and into the tissues, in the explosive type of wounds caused by shell fragments and land mines is appalling.

Attention is directed to the value of recording the pulse, respiration, and blood pressure on a

chart. It takes no longer to record it, and the graphic chart attached to the bed or litter allows one to appraise the status of the circulation at a glance. The first 1,000 c.c. of replacement fluid should be given rapidly, that is, each 500 c.c. within 15-20 minutes. The response to this initial infusion is a good guide to the severity of the shock. If the blood pressure promptly rises to well over 100 the shock is not severe, and immediate arrangements for surgery can be made. If the response is slow, either the degree of shock is extreme or there is continued bleeding. By this time laboratory data should be available. If the initial hematocrit is found to be low—and it is surprising how frequently readings of 25 or 30 are obtained—the former alternative is likely, and a second 1,000 c.c. should be given rapidly. If the initial hematocrit is not low, and especially if the nature of the injury makes continued uncontrolled bleeding likely, it is wiser to continue with the second 1,000 c.c. infusion more slowly, but to hasten preparation for surgery. In the presence of uncontrolled hemorrhage one cannot expect the blood pressure to return to normal. As soon as estimated losses have been restored, the general condition of the patient improves as evidenced by a return of colour and blood flow to the extremities, and the systolic pressure rises to 90 or 100, or higher, necessary operative procedures should be carried out, but *at the same time* transfusion must be continued. It is useless to transfuse and delay. It is folly to operate without continuing the transfusion. Amounts of 4,000 to 6,000 c.c. of blood given simultaneously, through two or more venipunctures, may be required. Indeed, there is no limit to the amount of blood which may be given if it is necessary to maintain the circulation in the face of continued and temporarily uncontrolled losses. The hypothetical danger of renal damage or toxic reactions to massive blood transfusions is no contra-indication to their use when the alternative is death.

The value of hemoglobin or hematocrit deter-

minations in acute hemorrhage has not been sufficiently appreciated. Particularly after early replacement therapy with plasma, but, at times, even in untreated patients, enormous reductions may occur within a few hours of injury. Often the degree of blood loss indicated by this laboratory finding is fully appreciated, but occasionally, and especially in non-penetrating abdominal injuries with concealed hemorrhage, very low values are obtained unexpectedly and furnish information of considerable value. In crushing wounds, or badly lacerated explosive wounds, hemorrhage is associated with a loss of additional plasma, thus making changes in the hematocrit variable and difficult to interpret. Moreover, the nutritional state and water balance of the patient at the time of injury will also affect the change in the hematocrit. Some of the possible variations in the hematocrit are shown in the accompanying table (Fig. 1).

In burns, peritonitis, dehydration, and following prolonged surgery, varying degrees of hemoconcentration occur. In burns, particularly, this rise in the hematocrit long precedes the fall in blood pressure. When the loss of plasma or fluid is severe and prolonged the following formulae may be used as guides to the volume of replacement required.

1. First-aid Formula: One pint of plasma for each 10 per cent of body area burned. (Harkins) (Fig. 2.)
2. Plasma required equals $(5 - \frac{500}{H^b}) \times 1000$, in which H^b is the hemoglobin observed after the injury (Black).
3. For each point the maematocrit is elevated above the normal of 45, give 100 cc. of plasma (Harkins).

It must be emphasised that these formulae only provide an approximate estimate of the requirement. Determination of the serum protein pro-

FIGURE 1

Clinical State.	Fluid Lost	Stage of Shock	Hematocrit or Hemoglobin
Clean wounds of major vessels, injuries of spleen, liver, etc.	Blood	—Early —After dilution by plasma infusion	—Normal-low —Low
Burns Crush	Plasma	—Early —After dilution by plasma infusion	—High —Normal
Crushing injuries with associated hemorrhage, extensive explosive wounds, compound fractures with extensive tissue injury, etc.	Blood and additional plasma	—Early —After dilution by plasma infusion	—Normal—high —Low—normal

vides an additional basis for estimating the degree of plasma loss. A high hematocrit associated with a normal, or slightly reduced, level of the serum protein is indicative of an enormous loss of plasma. In simple dehydration the plasma protein concentration as well as the hematocrit is high.

FIGURE 2.

Area Burned.	Plasma Required (approximate)
Head	300 cc.
Upper Extremity:	
Arm and Forearm	300 c.c.
Hand	100 c.c.
Total	400 c.c.
Trunk:	
Anterior surface	1000 c.c.
Posterior surface	900 c.c.
Total	1900 c.c.
Lower Extremity:	
Thigh	500 c.c.
Leg	350 c.c.
Foot	150 c.c.
Total	1000 c.c.

In evaluating both the laboratory and clinical data there are no substitutes for acumen and common sense. It is of vital importance to determine whether or not there is a cerebral injury, cardiac tamponade, tension pneumothorax, or an open sucking wound of the chest, or severe infection, for all of these require prompt, specific therapy, and do not of themselves require massive plasma or whole blood transfusions. Indeed, they may be made worse by it. Furthermore, it cannot be emphasised too strongly that no single sign, symptom, or laboratory determination is a reliable guide to the state of the circulation. Characteristically, the pulse is rapid and weak, but rates of 90-100 are not uncommon even in severe hematogenic shock. The blood pressure may be low because of neurogenic factors alone. It may be normal, or above normal, after severe injuries with marked loss of blood or plasma. The laboratory data may prove exceedingly accurate and valuable, or it may furnish information so out of keeping with the general picture that it has to be disregarded.

GENERAL SUPPORTIVE MEASURES

Thus far the replacement therapy of hematogenic shock has been the principal concern of this discussion. Although it constitutes the

essence of modern shock therapy, certain additional supportive measures require mention. The necessity for adequate splinting and immobilisation of extensive soft tissue wounds, as well as of fractures, is well appreciated. Morphine must be used to control pain, but overdosage should be carefully avoided. Unless there is uncontrolled pain or excessive restlessness, morphine may only depress the respiratory centre and add to an already existing anoxia. To avoid delayed absorption with possible cumulative action it is best administered intravenously.

The use of heated cradles and many hot-water bottles wrapped in blankets has been discarded as a specific form of therapy. It is actually harmful since it increases peripheral vasodilatation when the available blood volume is needed elsewhere. On the other hand, the use of blankets and heat to maintain normal body temperature is often essential.

It has been proposed by certain observers that exposure to cold may be beneficial in shock. There is no sound clinical grounds for this. Recent experiences in Northern France indicate that patients who have been exposed to severe cold require more replacement therapy, and react less favourably to transfusion, than patients who have been protected from exposure, but who have essentially the same injuries. In the present state of our knowledge, it is only common sense to try to maintain normal body temperature.

The use of the Trendelenburg position is a debatable point. As an emergency procedure in haematogenic shock until the blood volume can be restored, and as a means of combating the loss of constrictor tone in neurogenic shock its use seems reasonable. Since it causes a certain degree of respiratory and circulatory embarrassment, it should be discontinued as soon as the circulation is restored by transfusion.

Oxygen should be used only when specifically indicated by dyspnea, air hunger, or cyanosis. The anoxia of shock is primarily circulatory, and once the circulation is restored oxygen is unnecessary. Its routine use is cumbersome and often interferes with, or delays, more vital forms of therapy. It should be used whenever specifically indicated, but not routinely. In neurogenic shock the simple measures of the recumbent position, sedatives, and warm drinks appear to hasten recovery. Failure of neurogenic shock to respond promptly to these measures should stimulate renewed search for signs of concealed haemorrhage.

THE CHOICE OF AN ANESTHETIC

It is axiomatic that surgery is never done until shock is treated. Once the circulation has been

restored by replacement therapy the choice of an anesthetic agent must be determined largely by the extent of the surgery to be done. That a general anesthetic is a contributing factor in the production of an irreversible state is well established experimentally. For this reason local anaesthesia is the one of choice and should be used whenever possible. When local anaesthesia cannot be used, ether is the best all-round choice. In general, spinal and pentothal anaesthesia should be avoided; at times they may be employed in exceptional circumstances by the highly-experienced anesthetist, provided the circulation has been restored to normal by replacement therapy.

Attention is directed to the specific usefulness of local anesthesia for amputation of badly injured limbs, especially when the circulation fails to respond after adequate replacement therapy. If adequate blood and plasma infusion have been given and the blood pressure remains low, it is evidence of an irreversible state. To delay further is to accept a fatality. On several occasions the writer, or his colleagues, has had occasion to advise immediate amputation under local anaesthesia while the replacement therapy is continued. Following removal of the damaged limb a general improvement of the circulation has followed, with eventual recovery.

Surgery in the patient who has been in severe shock is not for the dilettante or the beginner. Since the duration of the anesthesia is a contributing factor to an irreversible state, what is to be done must be done expeditiously.

THE PROBLEM OF SHOCK

There can be no doubt that in the present war early and adequate restoration of the blood volume by massive blood and plasma infusion is enabling thousands of men to survive injuries which would have proved fatal in the last war. There remains, however, a comparatively small, but significant, group of cases in which adequate replacement therapy is without avail. In this group are very extensive burns (80-100 per cent of the body surface), multiple explosive wounds with extensive damage to muscle, and late decompensated shock, irrespective of original cause or causes, but especially when complicated by sepsis (peritonitis, retroperitoneal cellulitis, etc.). Two factors which contribute to this irreversible state are the extent of the destruction of tissue and the time which elapses between the injury and surgical measures designed to correct the condition (for example, the debridement of extensive muscle wounds or the amputation of badly mangled limbs). A third factor which affects the response of the circulation

to replacement therapy is the age and general condition of the patient. An elderly, malnourished individual cannot withstand losses of blood or plasma which hardly affect a young, hardened soldier. Very extensive injuries (i.e. a burn of 90 per cent of the body area), even in well-conditioned individuals and even when treated promptly, will not respond to therapy. Less extensive injuries such as bomb wounds (land mine) of both legs, if treated by replacement therapy and amputation or debridement, as indicated, do well. If delay ensues, however, no favourable response to replacement therapy occurs. A discussion of the causes of this irreversible state is not within the scope of this paper. As suggested recently, it may be a generalised cellular death in which the heart as well as other viscera is affected. Whether this is a consequence of a specific toxin absorbed from the injured area, or of an altered metabolism of the cells because of anoxia, is unknown. From the practical point of view, all that can be done at the moment is to prevent this state from developing by early and adequate replacement therapy, followed by prompt surgery. Surgery in explosive wounds involving muscle is as important in preventing shock as in preventing sepsis.

SUMMARY

Practical points in the diagnosis, treatment, and prevention of shock derived from experiences in military surgery are presented.

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FIRST AID TREATMENT OF BURNS

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There is considerable confusion at the present time regarding the first-aid treatment of burns. Many of the methods, which have been recommended, are unsuitable and interfere with the subsequent treatment. The first essential is to appreciate that every burn involving more than 5 per cent of the body surface and burns of the face, hands, feet and eyes should be admitted at