THE ROLE OF THE ANAESTHETIST IN THE ASSESSMENT AND TREATMENT OF INJURY AT THE ACUTE STAGE

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The anaesthetist may be involved directly in the immediate care of seriously injured patients in resuscitation, anaesthesia for urgent surgery, or both. In any case he must be able to assess the clinical state of the injured patient in conjunction with the surgical team.

The most important recent advance in the care of patients with major injuries has been the recognition of the extent of blood loss associated with injury and the benefit derived from its replacement by transfusion of stored blood. Studies of the extent of haemorrhage following trauma have resulted primarily from the development of techniques for determining blood volume, either from the Evans Blue plasma volume and haematocrit, or from the cell volume using erythrocytes labelled with radioactive phosphorus (P32) or chromium (Cr51) (Davies, 1956).

Corroborative evidence of the extent of blood loss can be derived from routine follow-up haemoglobin studies (Topley and Clarke, 1956). Blood loss during surgery can be estimated by swab weighing or measurement of blood sucked out of the abdominal cavity or aspirated from the chest. Indirect evidence of blood loss into the tissues can be obtained by measurement of limb swelling at the acute phase (Clarke, Topley and Flear, 1955). Correlation of the appearance of wounds or closed injuries with blood volume studies can facilitate the assessment of blood loss from the nature of the injuries (Clarke and Fisher, 1956). Experience with swab weighing in the operating theatre can train anaesthetists to make their own visual estimates with a useful degree of accuracy. Clinical observation of patients on whom blood volume studies have been carried out can assist in the assessment of local or general signs as evidence of the presence and extent of haemorrhage or oligaemia.

Our investigations and experience have shown that the general conclusions enunciated by Grant and Reeve (1951) following their investigations during World War II are fully relevant to an important proportion of civilian injuries. They stress the frequency and extent of whole blood loss in battle casualties, the need for larger blood transfusions, and the relative unreliability of the blood pressure and pulse rate as quantitative indicators of changes in blood volume.

We have emphasized the contribution of blood loss into the tissues in association with closed injuries, especially fractures, and have developed the thesis of Grant and Reeve that the nature and extent of injury frequently constitutes the best guide to the extent of blood loss.

BLOOD LOSS FROM CLOSED INJURIES

Blood loss from closed fractures of the limbs can be crudely assessed from the volume of primary swelling. The likelihood of continued bleeding into the tissues can be based firstly on direct observation, secondly on an understanding of the mechanism of injury and its anatomical extent, especially in relation to the probability of muscle damage or vascular injury. Many straightforward closed fractures of the ankle, os calcis, tibia and fibula, forearm, elbow or shoulder can be associated with the loss of one pint (1 litre) of blood into the extremity. This in itself need cause no anxiety and obviously does not need replacing, but a number of such injuries may add up to a significant loss of blood. More severe fractures of the tibia and fibula and many closed fractures of the femur are associated with a primary blood loss of the order of 2—4 pints (1—2 litres). Severe fractures of the shaft of femur with major swelling may lead to much greater loss of blood. On several occasions we have seen patients who...
have lost half their total blood volume into the tissues of an injured thigh. Recent studies have shown that wide extravasation of blood can be associated with fractures of the ribs, and that even without a major haemothorax moderate chest injuries can be associated with a blood loss of 1–1½ litres, without this being obvious from the surface. Closed fractures of the pelvis, particularly when associated with visceral injuries, can cause the loss of very much larger quantities of blood and continued massive transfusion may be necessary for resuscitation, particularly where major surgery is required for the repair of a ruptured urethra or damaged rectum.

**BLOOD LOSS FROM OPEN INJURIES**

Blood loss from open wounds which occurs before admission to hospital is difficult to assess. In battle casualties Grant and Reeve (1951) have suggested that a crude guide to the total blood loss in limb injuries can be based on the extent of tissue damage, roughly assessed in terms of the volume of the human fist. This guide is in our opinion of only partial value in the majority of civilian injuries. Small lacerations may divide medium sized blood vessels with haemorrhage out of proportion to the size of the wound. Many patients with major lacerations of the forearm and hand can lose 2–4 pints (1–2 litres) before admission to hospital. They can continue to bleed through dressings whilst awaiting operation. In the ordinary run of open fractures the majority of which are “ compound from within ”, we have found that the extent of blood loss to the outside is frequently of the same order as the extent of bleeding into the tissues, so that open fractures tend to cause the loss of about twice as much blood as do similar fractures without skin penetration. Some major injuries of a crushing type are associated with early obliteration of the vessels and relatively little blood is lost. On the other hand, patients with major amputations, particularly through the thigh, lose more blood than is usually anticipated and continue to lose blood into tissues around the amputation stump. They frequently require not only large primary transfusions but repeated transfusions to maintain a normal quota of red cells.

We have presented elsewhere (Clarke, Topley, and Flear, 1955; Clarke and Fisher, 1956) a number of case histories with photographs and detailed blood volume studies to illustrate the general conclusions here presented.

Important additional evidence on the extent of blood loss in certain types of battle casualties was obtained by American research workers during the Korean War (Howard, 1956; Prentice et al., 1954). They found evidence of disappearance of red cells even greater than was the general experience in World War II, and reached the conclusion that this red cell loss was primarily due to haemorrhage, although some of the disappearance of red cells could be explained by their use of uncrossmatched Group O blood and a degree of haemolysis that has not been encountered in our studies.

**BLOOD LOSS AND CLINICAL SIGNS OF “ SHOCK ”**

(Clarke, 1956)

Injured patients presenting the classical picture of established “ haemorrhagic shock ” with pale cold extremities, a systolic pressure below 70 mm of mercury, a rapid pulse rate and obvious bleeding have probably lost at least half their total blood volume by haemorrhage. When surgery is needed they will almost certainly require a transfusion of the order of their total blood volume (4–7 litres) and occasionally much more. Patients with less obvious haemorrhage but with diagnosable closed injuries presenting the same clinical picture must be regarded as suffering from haemorrhage of the same extent. They require the same quantities of blood replacement.

Patients with trivial injuries can for a very short period of time present a somewhat similar picture. There is certainly a form of purely neurogenic shock that needs to be differentiated from the effects of haemorrhage. But even this condition is predisposed by any pre-existent loss of blood. In the absence of continuing major irritation to the nervous system, this type of neurogenic “ shock ” will respond very rapidly if the patient is kept flat. In the presence of evidence of major injury it is very dangerous to assume that a patient reaching hospital in a state of apparent circulatory collapse is suffering from a purely neurogenic response with no underlying component of oligae mia.
Blood volume studies have now established that patients with 15–30 per cent of whole blood loss may show no evidence of circulatory upset. More frequently such patients are pale and their extremities take longer to warm up than those of an individual not suffering from haemorrhage. A proportion of patients with total blood loss of 25 per cent will be found to have a raised blood pressure, the "hypertensive phase" described by Grant and Reeve. This is sometimes associated with pain and anxiety. It appears to be more likely to develop in the presence of severe tension in an injured part even if this is not associated with pain. The raised blood pressure does not necessarily respond to morphine, usually disappears under anaesthesia, but may recur subsequently unless the blood volume is restored to normal. The presence of a high blood pressure is no indication for withholding transfusion from a patient known to be oligaemic. It is particularly important that this should not be overlooked in children because the hypertensive phase may be followed by sudden fatal collapse.

When patients are seen soon after injury (1–2 hours), a low blood pressure—less than 100 mm of mercury in a young man—or a low pulse pressure—20 mm of mercury—frequently indicates blood loss of 25 per cent or over.

The pulse rate may be of more significance than we had previously supposed and the quality of the pulse may give an important indication of deterioration. On the other hand, it is probable that a rise in pulse rate associated with moderate haemorrhage may be due to some other factor—developing pneumothorax or peritoneal irritation. Clinical evidence of the state of the circulation is useful but frequently gives a misleading picture of the degree of oligaemia, and impressions derived from such evidence should be ignored when the nature of the injuries clearly indicates a greater degree of haemorrhage.

SIGNIFICANCE OF BLOOD LOSS
So far we have discussed oligaemia in terms of whole blood loss. There is now an overwhelming body of evidence that in the majority of non-thermal injuries oligaemia is in fact due to whole blood loss. It is only in burns, the occasional crushing injury and in the presence of established peritonitis, that there is a loss of plasma-like fluid from the circulation unassociated with loss of red cells. It is probable that in many closed injuries there may even be reabsorption of fluid and electrolytes from the site of haemorrhage into the injured tissues even while further haemorrhage is still taking place, so that red cells are lost to a greater extent than the fluid component of blood. It is generally agreed that in severe haemorrhage transfusion of whole blood is the treatment of choice. When there is a shortage of whole blood, plasma and plasma substitutes may play a limited role in maintaining the circulation for a short time. With an adequate transfusion service there is little evidence that they are anything but a second or third rate substitute for whole blood.

Minor blood loss need not always be replaced in toto. Healthy subjects can lose 1–1½ litres of blood and even more without any immediate danger to life. If they do not need to be moved and do not require surgical treatment, the loss of such quantities of blood can be left to take care of itself. In patients requiring surgery, in the elderly and in the very young, and in all patients who are in any way systematically sick, even the loss of 1–3 pints of blood may decisively contribute to the risk to life. Many patients with fractures of the shaft of the femur have collapsed following transport without splintage, turning, anaesthesia, or surgery. A patient who has lost 1–2 pints of blood from closed fractures may collapse dangerously from quite minor bleeding lacerations unless the bleeding is promptly arrested. Many of the problems associated with anaesthesia in the injured patient are facilitated by recognition of the extent of blood lost and its accurate and timely replacement.

The role of transfusion, moreover, is not confined to the period of primary resuscitation and anaesthesia. Continued postoperative bleeding, especially from amputations and major orthopaedic interventions, is usually underestimated and may require continued transfusion.

On the other hand, moderate bleeding is followed by haemodilution and the blood volume may be restored by a transfusion which is inadequate to replace the lost red cells. In the past it
has been recognized that transfusion for anaemia plays a vital part in the treatment of established wound infections. The prevention of post-traumatic anaemia by full blood replacement appears to contribute to the prevention of infection and the better healing of wounds, especially where the circulation has been interrupted by trauma. The rapid correction of oligaemia certainly plays a part in the prevention of traumatic anuria, and there is evidence to imply that prolonged uncompensated oligaemia predisposes to liver failure, and to cerebral or coronary thrombosis in patients with degenerative conditions of the vascular tree. Clinical observations (Clarke, 1952) suggest that transfusion sufficient to maintain the red cell volume leads to quicker and smoother convalescence. Flear and Clarke (1955) have shown that this improved convalescence is associated with alterations in the metabolic and electrolyte response. Water and sodium retention are certainly reduced by adequate transfusion. Topley and Fisher (1956), Flear (1956) and others have suggested that a variety of complications which may follow surgery or trauma are favourably influenced in proportion to the adequacy of transfusion. There is probably no substitute for red cells lost by haemorrhage except whole blood transfusion and the evidence is increasing that stored blood is as effective for cell replacement as fresh blood. Those responsible for transfusion must consider not only the immediate effects of oligaemia but the long term events which may be adversely affected by a shortage of red cells. This is an additional reason for a cautious attitude to the use of plasma or plasma substitutes when whole blood has been lost.

TRANSFUSION RISKS

The advantages of replacing lost red cells by transfusion must obviously be weighed in the balance against the risks involved, and perhaps to a lesser extent against the organization required. The methods of assessing blood loss, as detailed previously, frequently make it possible to estimate blood requirements in time to allow for accurate grouping and full crossmatching. The overwhelming majority of transfusions for acute injury at the Birmingham Accident Hospital have been of type-specific blood. Patients with severe blood loss cannot wait, but the majority with moderate injuries—not obviously bleeding rapidly—can wait at least half an hour for grouping and emergency crossmatching, whilst many can wait two hours for full crossmatching. The primary grouping must include the rhesus factors in addition to the ABO group.

During the past eight years we have lost one patient from an incompatible transfusion. With an efficient organization and careful checking at all levels, the danger to life from transfusion itself need be no greater than the risk of expert anaesthesia for a minor operation on a fit patient. The greater risk is of transfusing too rapidly at the wrong time, but even this is very much less today than the risks involved in transfusing too slowly and too late.

In the presence of clear evidence of bleeding, the earlier a patient is seen the more rapidly can blood loss be safely replaced. The greater the extent of blood loss the more is speedy replacement urgently required. Patients with a blood loss of 40–50 per cent of their total blood volume, particularly when this is associated with severe injury, should be transfused as rapidly as possible. The intravenous route is usually as efficient as intraarterial transfusion. With the aid of pressure and a large bore Guest cannula a bottle of blood can be transfused into a single vein in about four minutes. Occasionally it is necessary to set up several transfusions at once.

With less severe haemorrhage the transfusion can be kept going as a continuous stream until it is estimated that the blood volume has been restored to normal. It can then be adjusted to a fast drip and further timing based on observation of further bleeding or on the patient's general condition. In my opinion the colour of the toes frequently provides a very useful guide to full blood replacement. For moderately severe injuries I aim to replace blood loss by the end of the anaesthetic administration and this should leave the patient not only with a warm and pink nose and ears but with warm pink toes. Further transfusion should be discussed with the surgeon concerned in relation to all the factors mentioned previously.

When there is delay in the patient's reaching hospital so that haemodilution is likely to have taken place, transfusion should be more cautious
except in a condition of advanced "shock". In old people and particularly in those where there is evidence of disturbance of pulmonary or cardiac function, greater care is needed. On the other hand, such patients stand blood loss and under-transfusion just as badly as a transfusion which is excessive in quantity or administered too rapidly.

With added accuracy in the assessment of blood loss it is possible to ensure that the majority of patients with moderate injuries come to the operating theatre not only with a normal blood pressure, pulse rate, skin temperature and colour, but with a blood volume which is nearly normal. Under such conditions definitive surgery can be carried out without undue haste and with a high degree of safety. This is of great importance in trauma because the early hours following injury frequently provide the only opportunity for adequate repair of injured tissues and adequate wound surgery to prevent infection.

When there is evidence that progressive bleeding particularly into the abdominal cavity, is taking place faster than can be replaced by transfusion, surgery becomes an essential part of the resuscitation process and must be carried out whilst the patient is still improving; transfusion must be continued throughout.

ANCILLARY METHODS OF TREATMENT

Our advancing knowledge of the role of transfusion is associated with clearer conceptions of the physiological disturbances associated with the response to blood loss in trauma. We no longer attempt to treat the coldness of the patient's extremities by artificial heating. A patient with a minor injury may be comforted with a blanket but there is never any indication for the use of hot water bottles or hot cradles. In warm weather the injured patient does not require more than a single blanket and even this is usually unnecessary during the course of anaesthesia.

It is now fully recognized that such drugs as morphine and pethidine should not be injected subcutaneously to a patient with an obvious deficiency of the peripheral circulation. These drugs are beneficial when given for pain or extreme restlessness and anxiety. When necessary in acute "shock" they should be given intravenously and in small doses, repeated as indicated by the response of the patient.

Position is important. The patient in a state of severe collapse can with advantage be nursed with the head of the table tilted down. Most other patients should be nursed flat except when chest injuries make it difficult for them to breathe properly in this position.

The need for oxygen is less than the need to clear the airway, particularly in the presence of bleeding from a fractured skull or jaw or lacerations around the mouth. Tracheal or bronchial suction or intubation may be required under such conditions; assisted respiration may be useful and many patients with head injuries, cut throats and paradoxical respiration with a loose segment of the chest wall, benefit from early tracheotomy carried out under local anaesthesia. A severely injured patient may be upset by any movement, although this may be necessary for examination and radiological investigation. All interference is likely to be safer if it can be delayed until the blood volume has been restored to something like normal.

Since many of the above procedures are closely related to the normal activities of the trained anaesthetist, and particularly as the anaesthetist will have a major responsibility during the anaesthetic period, I am convinced that wherever possible a member of the anaesthetic staff should be available as part of the resuscitation team at the earliest possible moment following the admission to hospital of a severely injured patient. He will not only play an invaluable part in the pre-operative handling of the patient but will have the opportunity to form independent assessments of the need for blood and of the indications for and timing of transfusion.

COOLING AND THE USE OF CHLORPROMAZINE

During the last few years there has been a great amount of discussion, research and clinical observation concerned with cooling, and with attempts to alter the general state of reactivity of sick and injured patients with chlorpromazine and allied drugs, the "lytic cocktail" in various forms, with or without artificial cooling.

In cardiac surgery it would appear that cooling can make a decisive contribution to the safety of
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...direct surgical intervention, particularly in association with methods of maintaining the circulation to essential organs by mechanical means. The early suggestion (Laborit, 1952) that the treatment of the severely injured could be transformed has not yet been substantiated. There is a widespread clinical impression that chlorpromazine is a useful adjunct to premedication and anaesthesia. There are reports of individual patients who appear to have benefited from cooling and chlorpromazine, and of other new drugs which have contributed to the possibility of safe prolonged states of sedation.

The general conception of altering the whole nature of the individual response to trauma, so that excessive reactions are modified and the general metabolic needs decreased, is of profound interest from a long term point of view, but in my opinion we need to know much more about the physiological disturbances present during all phases of the illness of trauma before we can assess the practicability of this method of approach. From the purely empirical point of view we need very detailed observations on all patients receiving treatment of this kind.

It is important, moreover, that the complex difficulties, which underlie the making of a full diagnosis in severe injuries, should not be further complicated by drugs whose detailed actions have not yet been fully evaluated, without overwhelming evidence of the beneficial character of their clinical action.

A number of other observers have advocated the use of noradrenaline in states of “shock”. This is likely to be even more dangerous as a general approach. There may be individual patients who can benefit from the “lytic cocktail” and others who might benefit from noradrenaline. The majority of patients reported in the literature could well have recovered with transfusion alone in adequate doses at the right time. The whole subject still requires much careful clinical and research study.

Meanwhile, no illusion that “shock” is a single simple entity or that it can respond to a specific drug, should distract from the careful assessment of blood loss and the continued evaluation of its quantitative replacement.

REFERENCES


