Chapter 28

DAMAGE CONTROL PHILOSOPHY IN CRITICAL CARE: PATIENT MANAGEMENT AND ORGAN SUPPORT

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INTRODUCTION

Following ballistic trauma, there are two clear goals of damage control during intensive care:

1. To recognize and treat the complications of all primary injuries.
2. To identify and manage the complications of therapy and prevent iatrogenic injury.

Ten years ago, United Kingdom (UK) and US military intensive care was provided as part of a sequential package following initial resuscitation and damage control surgery. Operative procedures during this stage were limited to 60 minutes so that surgeons focused on "life and limb" interventions, controlling bleeding, and removing contamination. Following temporary wound closure, the patient was resuscitated in critical care, with targeted interventions against the lethal triad of acidosis, hypothermia, and coagulopathy. With corrected physiology, the patient could later be returned to surgery for second-look procedures.

Experience gained in Iraq and Afghanistan has led to refinement of treatments and development of damage control resuscitation. Surgeons now follow an integrated model of care involving permissive hypotension, hemostatic resuscitation, and damage control surgery.[1-4] In combination with massive transfusion,[5,6] targeted warming, and near-patient testing of coagulation and arterial blood gases, damage control resuscitation is so successful that patients can potentially emerge from surgery normothermic, euvolemic, and with normal blood gases and clotting profile. These techniques have allowed surgeons to extend operating times and perform more surgical procedures, in particular vascular repairs, extensive debridement, and the external fixation of fractures, in a single episode of surgery.

Despite these advances, significant challenges remain for the deployed intensivist. Even with intraoperative resuscitation, changes in coagulation and volume status will likely develop due to ongoing consumption of clotting factors and third space losses. Not all patients have initial resuscitative surgery in Role 3 facilities, and consequently blood products and near-patient testing may be limited. These patients will require transfer to a Role 3 facility for further investigation, resuscitation, and management. During major incidents, patients may need direct admission to the critical care unit until an operating room table becomes available. Finally, those managed in Role 3 need appropriate preparation before transfer to tertiary services for second-look and definitive care. Consequently, critical care can be involved at any point on the damage control resuscitation and evacuation pathway.

Despite the complexity of their injury patterns and the large transfusions given, patients with major trauma can be saved if simple rules are followed. Clinicians must avoid assumptions, be meticulous, and follow a structured approach with an active, regular review process. Care must be proactive and targeted to prevent the lethal triad. It is good practice to follow the "ABCDE" approach, as explained below, and to remember the goals of treating all primary injuries and preventing iatrogenic insults.

AIRWAY

For any patient requiring mechanical ventilation, it is mandatory to use the correct size endotracheal tube, insert it to the correct length, and inflate the cuff to the correct pressure. Furthermore, the tube must be securely attached to the patient, mindful of the patient's injury pattern. Failure to apply these rules can cause a number of iatrogenic complications.

Currently, cuff pressures are rarely measured prior to admission to intensive care, and particularly with out-of-hospital intubations, cuffs are likely to be overinflated in the field to quickly secure the airway. Consequently, it is mandatory to check cuff pressures on admission to intensive care to limit long-term sequelae from mucosal ischemia such as subglottic stenosis.

Unrecognized endobronchial intubations can cause segmental lung collapse, which limits gas exchange and mimics hemothorax radiologically. In the presence of multiple injuries, these radiographic findings can lead to the unnecessary placement of chest tubes. Children are particularly at risk of endobronchial intubation because of their shorter airways.

Care should be taken when securing endotracheal tubes to ensure that they do not migrate. Local injuries may make it necessary to use adhesive tape, commercial tube holders, or sutures. Tape ties must not be allowed to obstruct neck veins or cause pressure areas.

BREATHING

Patients requiring mechanical ventilation should be managed with a protective lung strategy to prevent injury from excessive volume or pressure. To prevent such injuries, tidal volumes must be limited

to a maximum of 6 mL/kg. Carbon dioxide clearance is facilitated by increasing respiratory rates (up to 30 breaths per minute), although in the absence of a concurrent metabolic acidosis, hypercapnia can be tolerated providing the pH remains above 7.25. All patients should have end-tidal CO_2 monitoring as part of their respiratory care package.

Patients should be ventilated with 5 cm of positive end-expiratory pressure (PEEP) to prevent basal atelectasis, but PEEP can be increased if oxygenation is difficult. $Pa0_2$ greater than 8 kPa is an acceptable target for most patients.

A chest tube with suction system or Heimlich valve is mandatory for patients with a pneumothorax who need aeromedical evacuation,[7] but all chest drains should be checked to ensure they are correctly positioned in the chest cavity and that pneumothoraces and fluid collections are draining appropriately. Regular checks should ensure that the drains are intact, without disconnection, and that any suction is applied within safe limits. All drains should be removed at the earliest opportunity to reduce the risk of infection.

CIRCULATION

Maintaining Blood Pressure

Damage control resuscitation includes permissive hypotension (blood pressure targeted to a palpable radial pulse), hemostatic resuscitation, and damage control surgery. Permissive hypotension is permitted only during the first hour until control of massive hemorrhage has occurred and is not usually a feature of critical care.[2] Hemostatic resuscitation is a strategy using blood and blood products as primary resuscitation fluids. The UK policy is to give blood, fresh frozen plasma, and platelets in a ratio of 1:1:1, unit for unit. Patients receive doses of tranexamic acid and cryoprecipitate as guided by rotational thromboelastometry (ROTEM [Tem International, Munich, Germany]), and ionized calcium is kept above 1 mmol/L, monitored by arterial blood gas analysis. This policy is followed from point of wounding through critical care.

Clotting activity is known to fall by 10% for every 1°C fall in core temperature; consequently, it is important to correct hypothermia to interrupt the lethal triad. Blood and blood products (excluding platelets) are given through high volume infusion equipment (examples include the Level One Fast Fluid Warmer [Smiths Medical, Ashford, Kent, UK], or the Belmont Rapid Infuser [Belmont Instrument Corp, Billerica, MA]). The warming capabilities of these devices, in combination with forced air warming blankets and heated mattresses, ensure that patients achieve normothermia. Considerable care should be taken not to overheat patients, since the vasodilatation associated with warming might accentuate relative hypovolemia.

Guided by hemoglobin, lactate, bicarbonate, and base deficit, together with central venous pressure (CVP) and mean arterial pressure (MAP), blood products are often given in large volumes. As a consequence, infusions of vasopressors are seldom required in the operating room because most patients can be resuscitated to euvolemia. Despite concerns that stored blood might contribute to the metabolic acidosis of hypovolemic shock,[8,9] experience shows that the acidosis usually resolves during resuscitation. Failure to correct acidosis typically results from a missed or overwhelming injury.

Advanced cardiac output monitoring is usually not required in the operating room or intensive care units, but clinicians should avoid fluid overload once hemorrhage has been controlled. Although pulmonary edema can be readily treated by increased PEEP and doses of furosemide, lung contusions are common in trauma patients. The combination of mechanical injury (contusion) with iatrogenic insult (edema) can cause hypoxemia, which is poorly tolerated by those with concurrent head injury. Efforts to improve oxygenation in the injured lung by increasing PEEP can lead to barotrauma and raised intracranial pressure (ICP). Pulmonary edema is probably the only indication for furosemide in the first 24 hours of admission.

Complications of Fluid Administration

Patients who have received large volumes of blood products are at risk from hyperkalemia, arrhythmias, and cardiac arrest. Although these concerns are reduced by administering calcium as part of the transfusion protocol, calcium does not eliminate these risks. The presence of tented T-waves on electrocardiogram (Figure 28-1) indicates dangerous hyperkalemia and should be treated acutely with 10 units of insulin with 50 mL of 50% dextrose. Electrocardiogram changes (tented T waves, ST segment depression, and widened QRS > 0.12 sec) are expected to resolve and plasma potassium levels return to the normal physiological range following treatment. It is important to avoid episodes of hypoglycemia when managing hyperkalemia, and consequently arterial blood gases should be taken to measure both the response of glucose and potassium to this intervention.

Arbitrary targets for MAP (> 60 mm Hg) and CVP (>10 cm H_2O) can be set, but must be associated with improvements in metabolic function. Ventilated and sedated trauma patients tolerate hypovolemia poorly

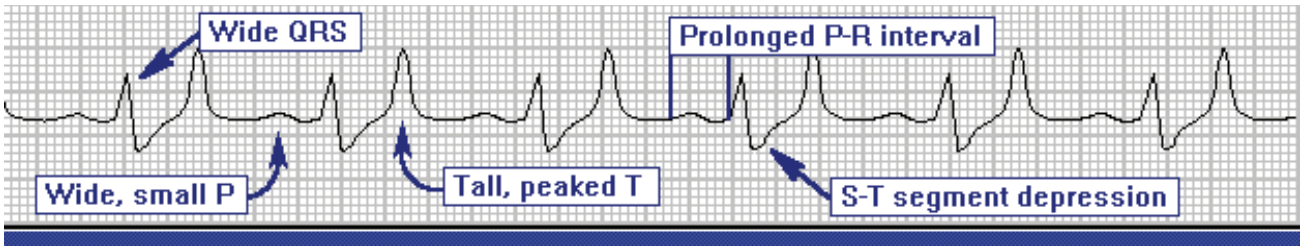

Figure 28-1. Changes in the T wave (tall, peaked, or tented) give the earliest indication of hyperkalemia. Other changes that might be seen include wide, flat, or absent P waves; prolonged P-R interval; S-T segment depression; and widened QRS complexes.

because positive pressure ventilation reduces venous return and cardiac output. This effect is compounded by the use of sedative drugs, which are negatively inotropic and vasodilating in effect. Following the completion of surgery and return to the intensive care unit, it may be necessary to use a low-dose vasopressor and inotropes to achieve a suitable MAP, provided the patient is euvolemic.

A urine output of 1 mL/kg/hour is desirable, but the stress response may reduce this amount by increasing water retention in the kidneys. If the patient's acid base, urea, and creatinine are normal (or correcting), there is no need to increase urine output with fluid boluses, and furosemide should not be administered. In clinical scenarios where rhabdomyolysis and myoglobinuria are demonstrated, it is desirable to have a good urine output, but the restoration of euvolemia and MAP are appropriate means to achieve this, rather than loop or osmotic diuretics.

Avoiding large volumes of crystalloid solutions during resuscitation has a number of advantages. The negative impact of these solutions on coagulation, acid base balance, and renal function (hyperchloremia) is avoided. Anecdotally, there appears to be less tissue edema in patients who have had hemostatic resuscitation, reducing the incidence of bowel dysfunction and abdominal compartment syndrome.

Compartment Syndromes

Compartment syndromes result from increased pressure in a closed body compartment, eg, tension pneumothorax, pericardial tamponade, and increased ICP, all conditions that are observed in the victims of ballistic trauma. Extremity compartment syndromes can occur following any mechanism of injury but are most common following fractures. Inflammation and edema following injury (and fluid resuscitation) cause swelling and increased pressure within fascial compartments. Microvasculature is compressed, causing ischemia and rhabdomyolysis, with subsequent lymphatic and venous drainage obstruction, resulting in venous congestion. If left untreated, compartment syndrome can have devastating consequences such as muscle necrosis, ischemic contractures, infection, and delayed union of fractures. For this reason, fasciotomies are often performed electively in patients with an injury pattern placing them at risk. When a fasciotomy is not performed, the intensivist must be alert to the possibility of compartment syndrome, because muscle necrosis may have commenced by the time peripheral pulses are lost.

Abdominal compartment syndrome (ACS) is described as intraabdominal pressure greater than 20 mm Hg associated with new onset end-organ dysfunction or failure.[10] Patients who have had an emergency laparotomy for trauma are at high risk for developing ACS even with a laparostomy. ACS has many causes, including intraabdominal packs; post-resuscitation visceral edema; intraperitoneal, retroperitoneal, or pelvic bleeding; and the failure of conservative measures to treat solid organ injury. Intraabdominal hypertension can affect the cardiovascular, respiratory, renal, alimentary, and central nervous systems. Raised intraabdominal pressure impedes venous return to the heart and causes sequestration of blood in the lower extremities. The diaphragm is pushed cephalad, decreasing thoracic compliance, and increased airway pressures are required for mechanical ventilation. Additionally, functional residual capacity falls, and ventilation perfusion mismatch impairs oxygenation. In the face of massive transfusion, these changes could be misinterpreted as acute lung injury.

Oliguria or anuria despite aggressive fluid resuscitation is a typical sign of ACS. The mechanisms for these conditions include direct compression of the renal parenchyma, decreased renal perfusion secondary to a fall in cardiac output, and increased water and sodium retention through activation of the renin angiotensin system.

ACS increases ICP by increasing intrathoracic pressure and impeding venous return from the brain. Gut

perfusion falls due to reduced cardiac output and increasing splanchnic vascular resistance, causing tissue ischemia.

The significant consequences of ACS and the danger of mistaking it for other conditions mandate that it be identified at the earliest opportunity by measuring intrabdominal pressure in all at-risk patients. ACS can occur in the absence of intraabdominal pathology, representing whole body ischemia reperfusion injury, and is associated with resuscitation from massive shock, eg, following multiple extremity fractures, traumatic amputations, pelvic fractures, and penetrating chest injury.

Although successful in treating coagulopathy and acidosis, hemostatic resuscitation is not without attendant risks. Hyperkalemia, pulmonary edema, and urticarial reactions are often seen. Transfusion-related acute lung injury is also a risk, and later complications such as infection and thromboembolism are currently being assessed. Therefore, it is important to use blood products appropriately and recognize the transition between active volume resuscitation against hemorrhage and the vasodilating, systemic inflammatory response syndrome when vasopressors may be more beneficial.

DISABILITY

The presence of traumatic brain injury (TBI) considerably complicates the management of trauma patients, but damage control principles can be used to limit secondary brain insults. Periods of hypotension are poorly tolerated by patients with TBI, and consequently permissive hypotension should not be practiced during initial resuscitation. In the field critical care unit, it is unlikely that invasive ICP monitoring will be available (although at the time of writing this situation is under review for UK forces). The intensivist must therefore make an assumption about ICP based on computed tomography imaging, pattern of injury, and clinical findings. Evidence suggests that neurological outcomes are worse if cerebral perfusion pressure falls below 60 mm Hg, so MAP must be targeted at 80 to 90 mm Hg, assuming an ICP of 20. This MAP level contrasts with the lower blood pressures tolerated in the absence of TBI, and may require the earlier use of vasopressors. Vasopressors can complicate the management of metabolic acidosis because they may be required before euvolemia has been achieved and can contribute to tissue ischemia through their vasoconstricting effects.

Patients should be adequately sedated and nursed head up (30°), ensuring that endotracheal tube ties do not obstruct venous drainage of the head and neck. Patients should be ventilated to normocapnia ($PaCO_2$ of 4.5–5.5 kPa, with $PaO_2 > 10$ kPa). Maintaining this ventilation may be challenging, necessitating protective lung strategies that might result in a degree of respiratory acidosis. In this potentially no-win situation, the intensivist must balance the consequences of increased ICP against the risk of acute lung injury and acute respiratory distress syndrome.

Furthermore, in the case of a lung contusion or edema, the clinician must weigh the risk of increased PEEP on ICP against an acceptable target for PaO_2. Efforts to improve the mechanics of ventilation should be considered, including muscle relaxation and relieving intraabdominal hypertension.

The intensivist must be cautious not to over-warm the patient while attempting to limit the coagulopathic consequences of hypothermia since pyrexia is known to elevate ICP. In the absence of ICP monitoring, mild degrees of hypothermia may represent the favorable risk.

Fluid and electrolyte disturbances secondary to TBI can be profound, and the clinical goal should be maintaining a euvolemic patient with normal sodium (145–150 mmol/L) and electrolytes. Doses of mannitol or hypertonic saline should not be given indiscriminately but only when clinically indicated. Hypoglycemia and hyperglycemia should be avoided in TBI.

Some patients may need to wear hard cervical spine collars because of real or anticipated injury. These devices can obstruct neck veins and cause local pressure areas and infection. Consequently, they should be removed in ventilated sedated patients and replaced with sand bags and tape, adjusted as necessary when the patient is turned as part of pressure area care.

ENVIRONMENT

Record-keeping and Imaging

Patients often receive definitive treatment at facilities remote from their initial resuscitation, so it is imperative that meticulous records are kept, including notes on injury patterns, investigations, procedures, drugs, and fluids administered. All patients should travel with a copy of these notes and any imaging performed.

Patients normally have a whole body computed tomography scan as part of the trauma screen to identify all injuries. Where practicable, the scan is done prior

to surgery, but in cases of refractory shock it may be delayed until after damage control surgery. It is vital that the critical care team have a full written report of all imaging, and that all surgical teams are made aware of the findings.

Gut

Damage control laparotomy for bowel injury can result in multiple resections, with blind ends left stapled and the bowel in discontinuity. Enteral nutrition is contraindicated, but a nasogastric tube should be left in situ to drain gastric collections. An alternative supply of calories can be provided with a 10% dextrose solution at 40 mL per hour. If enteral nutrition is expected to be delayed for a considerable period(> 72 hours), total parenteral nutrition should be considered, but it must not be commenced through dirty trauma lines. Blood glucose should be measured and kept within the range of 4 to 10 mmol/L. This range provides sufficient margin of error to limit iatrogenic hypoglycemia, while protecting against the adverse effects of hyperglycemia, especially in TBI. If the bowel is continuous and there are no primary anastomoses, enteral nutrition should start at the earliest opportunity.

Gastric Protection

All patients should receive gastric ulcer prophylaxis with H_2 receptor antagonists, and if the bowel is in continuity, prokinetics and aperients as indicated.

Venous Thromboembolism Prophylaxis

Trauma patients are at increased risk of embolic events and should use mechanical devices such as stockings and compression boots as allowed by their injury patterns. Low molecular weight heparins are indicated once there is no risk of bleeding and in the absence of TBI.

Intravascular Lines

Following initial resuscitation and surgery, any peripheral and central lines that are not being used should be removed to limit infection, prevent clots, and preserve veins for later use. Patients should be examined to ensure that all intraosseous devices have been removed, and all central lines should be transduced to confirm that they are placed in the venous system. Trauma lines sited in the emergency department and during initial surgery are by definition "dirty," but it is current practice to leave these in situ until second-look procedures at the tertiary facility.

Position

All patients should be nursed head up (30°) with attention to pressure areas, because this position aids ventilation and helps reduce microaspiration and ventilator-associated pneumonia. Similarly, spinal patients nursed supine should be managed by tilting the bed 15° to 30° head up.

Topical Negative Pressure Dressings

Care should be taken to ensure that topical negative pressure dressings are functioning correctly and that any measured losses are factored into the daily fluid balance. Although any active bleeding is expected to be controlled by surgery, ongoing drain losses associated with physiological changes should prompt urgent surgical review.

Transfer

To reach tertiary services, most patients require long journeys by air. During the flight, patients must be actively managed as an extension of their intensive care[7] (see Chapter 38, Air Transport of the Critical Care Patient).

Drugs

Any antibiotics and pain medications should be given according to local guidance and policy.

Regional Techniques

Once coagulopathy is controlled, peripheral nerve blocks and epidurals can be sited to treat acute pain and reduce the long term sequelae of chronic pain syndromes.

SUMMARY

The immediate survivors of ballistic trauma often have complex and life-threatening injuries. However, by using an integrated, targeted, and cooperative approach to trauma care, initiated early after wounding, many patients recover despite high injury severity scores.[11] Subsequently, the ability of these patients to accept and live with disability and their desire for rehabilitation vindicates the substantial medical assets deployed in their care.

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