Shock in Trauma

Caroline Leech, MBChB, FRCEM, FIMC RCSEd, FEWM[a,b,*],
Jake Turner, BMedSci, BMBS, FRCA, FIMC RCSEd[b,c,d]

KEYWORDS

• Shock • Trauma • Hypovolemia • Physiology • Resuscitation

KEY POINTS

- Understanding the cardiovascular responses to hemorrhage and injury aids interpretation of the vital signs following trauma.
- Hypotension following injury can be caused by several conditions other than, or in addition to, hypovolemia.
- There are several clinical pitfalls if clinicians use only pulse and blood pressure to assess the severity of hypovolemia.

INTRODUCTION

Shock is a life-threatening condition of circulatory failure leading to inadequate organ perfusion and inadequate tissue oxygenation. Left untreated, poor perfusion leads to anaerobic metabolism, lactic acidosis, and progressive cellular and organ dysfunction resulting in irreversible multiorgan failure and death.

Inadequate perfusion may result from failure of the pump (the heart), inadequate circulating blood volume (absolute or relative), or obstruction to the flow of blood through the circulatory system. Traditionally, shock has been subdivided into 4 main subtypes (**Fig. 1**). In practice, there is often considerable overlap, with different types of shock coexisting in the same patient. In trauma patients, this includes patients with more than one system injury (eg, traumatic brain injury [TBI], hypovolemia) or medical events (eg, sepsis, cardiac, neurological) contributing to the mechanism of injury resulting in a mixed picture of shock.

In healthy, uninjured patients, oxygen consumption (V_{O_2}) is closely regulated and serves as a carbon acceptor in the generation of ATP by mitochondria. In shock states when oxygen delivery decreases, reducing V_{O_2} below a critical level to maintain cellular metabolic demands, mitochondrial aerobic function is impaired. This results

[a] University Hospitals Coventry & Warwickshire NHS Trust, Coventry CV2 2DX, UK; [b] The Air Ambulance Service, Blue Skies House, Butlers Leap, Rugby, Warwickshire, CV21 3RQ. UK; [c] Anaesthetic Department, Nottingham University Hospitals NHS Trust, Derby Road, Nottingham NG7 2UH, UK; [d] Lincs & Notts Air Ambulance Headquarters, HEMS Way, Lincoln LN4 2GW, UK
* Corresponding author. Emergency Department, University Hospitals Coventry & Warwickshire NHS Trust, Walsgrave, Coventry CV2 2DX, UK.
E-mail address: Caroline.Leech@uhcw.nhs.uk

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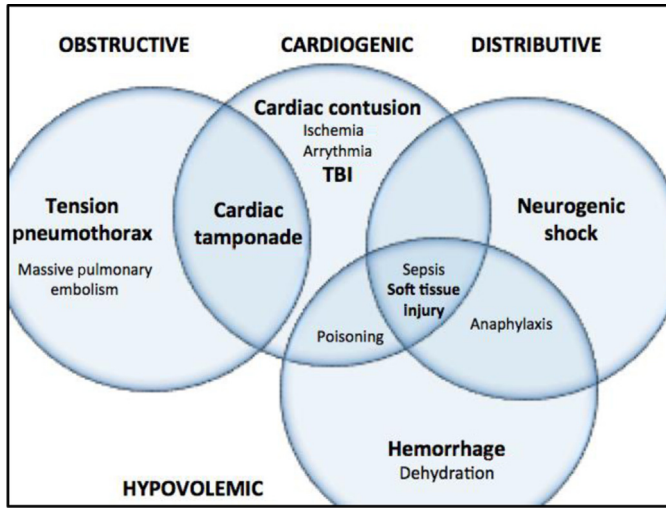

Fig. 1. Venn diagram showing the different subtypes of shock. TBI, traumatic brain injury.

in a cellular oxygen debt and accumulation of metabolic acids generated by anaerobic processes.[1] As oxygen debt progresses, the likelihood of cellular injury increases with a reduction in cellular membrane integrity, osmosis, and swelling. Intracellular organelles become damaged, cellular synthetic mechanisms cease, and lysosomes are activated resulting in cellular necrosis and death. Tissues with the greatest basal metabolic oxidative requirements (brain, myocardium, liver, kidneys) are particularly vulnerable, and depending on the extent and severity of cellular injury, can develop multiorgan dysfunction and failure.[2]

Hypovolemic shock results from inadequate circulating blood volume secondary to hemorrhage or excessive fluid loss. In the context of major trauma, hypovolemic shock secondary to uncontrolled hemorrhage is the most common cause of death in the military setting,[3] and the second most common cause of death (after TBI) in the civilian setting.[4] Bleeding may occur from damaged veins, arteries, solid organ parenchyma, and/or fractured bones. The rate of hemorrhage, presenting physiology, and trajectory of deterioration will depend on the anatomy of injury, source of bleeding, and patient factors (age, comorbidities, polypharmacy). Alternative or coexisting causes of shock in trauma often complicate the clinical assessment and management of these patients and are discussed in more detail later.

CASE SCENARIOS

Case 1: Incisional Torso Hemorrhage

A 25-year-old male patient has sustained an incisional wound to the left chest in the anterior axillary line 7th intercostal space. On arrival to the resus room, the patient is agitated, diaphoretic with no palpable radial pulses, BP 180/154, and a thready slow carotid pulse (HR 50). The patient has unrecordable peripheral oxygen saturations and looks centrally pale (lips, tongue, and conjunctivae). The prehospital team has placed an intravenous cannula and administered tranexamic acid.

The trauma team leader (TTL) tasks the anesthetic team to prepare for a rapid sequence intubation to facilitate ongoing management and resuscitation of the patient, but not to undertake this intervention until appropriate volume resuscitation

has been undertaken. The TTL confirms with the team that this patient is most likely critically hypovolemic and to disregard the spurious blood pressure. The anesthetic team sedates the patient with judicious doses of ketamine and full monitoring and oxygen is applied.

The trauma team fully exposes the patient (to exclude additional incisional wounds) and undertakes a primary survey. There is reduced air entry on the left side and absence of pleural sliding on ultrasound. There is no emphysema or active bleeding from the wound. An extended FAST scan excludes any significant pericardial tamponade but does identify free fluid within the left splenorenal fossa. Bilateral wide bore intravenous access is obtained and warmed blood products are started as there is no recordable blood pressure. A left-sided thoracostomy is performed (lung down, comes up, no blood) with insertion of a chest drain. Invasive blood pressure monitoring confirms that the patient is a transient responder to volume resuscitation with an opening BP of 40/30, improving to 75/52. The patient's heart rate improves from an initial 42 beats per minute, to 118 with volume resuscitation as the underfilled left ventricular C-fiber–mediated bradycardia resolves.

The TTL, in collaboration with the consultant surgeon, agrees that the patient should be taken directly to theater for a trauma laparotomy for hemorrhage control.

Case 2: Blunt Polytrauma

A 67-year-old male patient has been run over by a heavy goods vehicle and sustained lower abdominal, pelvic, and bilateral femoral injuries. On arrival, the patient is confused, has palpable radial pulses and a NIBP of 100/40 with a heart rate of 75. The pulse oximetry trace is poor, but intermittently reading 94% on 15L oxygen. The prehospital team stated that the patient was initially unconscious but has become confused en route to hospital. Drug history includes bisoprolol, amlodipine, ramipril, aspirin, and furosemide.

Primary survey identifies bilateral rib fractures, no surgical emphysema, lower abdominal contusions and tenderness, pelvic tenderness with a pelvic binder that is correctly positioned over the greater trochanters, bilateral closed femoral fractures splinted with traction devices, and left frontotemporal bruising with bilaterally reactive pupils. The patient has a Glasgow Coma Score (GCS) of 13 (M5, V4, E4), is cool peripherally, and has palpable radial pulses with a consistent BP of 98/45 and HR of 70. A venous blood gas confirms a normal baseline hemoglobin, but with deranged acid-base status (pH 7.18, BE -8.2, Lac 3.4) consistent with a shock state.

The TTL summarizes to the team the current injuries and main issues: this is an older patient with clinical signs of significant hypovolemia for his baseline physiology. Plan is for careful volume resuscitation to a MAP 80 to 90, and when able, a whole-body CT due to the high blunt-mechanism injury burden. The anesthetic team is asked to place a radial arterial line to help guide ongoing volume resuscitation and blood products are commenced. The patient responds to 2 units of blood and is transferred to CT, which demonstrates the femoral fractures, complex pelvic fractures, bilateral rib fractures, and pulmonary contusions but with no active bleeding.

PHYSIOLOGY OF HYPOVOLEMIA

Shock Classification

The traditional ATLS classification of hypovolemic shock with 4 classes defined by % blood loss, heart rate, blood pressure, respiratory rate, and GCS has been shown to overestimate the degree of tachycardia and hypotension associated with increasing

blood loss.[5] The physiological response to bleeding in trauma is complex, variable, and dependent on both injury and patient-related factors.[6,7]

Hemodynamic Reflexes to Hypovolemia

There are 3 main compensating reflexes that need to be understood when considering the physiological response to acute hypovolemia: the arterial baroreceptor reflex, the reflex elicited by activation of the cardiac C-fiber afferents, and the arterial chemoreceptor reflex (**Table 1**). Only a proportion of patients with hypovolemia will present with hypotension and tachycardia.

Simple hemorrhage without significant soft tissue injury (eg, *penetrating trauma*) is often biphasic with an initial baroreceptor-mediated reflex tachycardia and peripheral vasoconstriction. As hemorrhage progresses, there is a subsequent vagally mediated depressor response resulting in bradycardia and peripheral vasodilation. This is referred to as a biphasic response and may be secondary to cardiac C-fiber–mediated reflexes (**Fig. 2**).[8] This second reflex may confer some degree of protection with the bradycardia supporting diastolic filling, increasing stroke volume, and improving coronary perfusion.[7]

With tissue injury and ischemia in *blunt trauma*, it is the baroreceptor-mediated tachycardia and peripheral vasoconstriction response which predominates. This intense vasoconstriction redistributes blood flow and oxygen delivery from metabolically active organs such as the gastrointestinal tract, increasing oxygen debt, and promoting subsequent multiorgan dysfunction.[7]

In military battlefield casualties, explosions and *blast injury* are currently the primary mechanism of trauma.[9] Casualties are likely to have extensive tissue damage with severe blood loss from a combination of blunt and penetrating trauma, with a minority having hypoxia secondary to blast lung. The cardiorespiratory response to blast lung causes a characteristic triad of bradycardia, prolonged hypotension, and apnea followed by rapid shallow breathing.[10] This reflex will augment any biphasic response to severe hemorrhage as a consequence of secondary blast injury.

Administration of *opioids* for analgesia may have a deleterious impact on hemodynamic stability in the context of hypovolemia. In simple hemorrhage, morphine may

Table 1
Hemodynamic reflexes

Reflex	% Blood Loss	Receptor Location	Mechanism	Effects	Clinical Signs
Arterial baroreceptor	10-15%	Carotid sinus, aortic arch	As pulse pressure decreases, activity decreases	↓ Vagal activity = predominant sympathetic activity	Tachycardia ↑ SVR
Cardiac C-fibers	> 20%	Left ventricular myocardium	Contracting around empty chamber	↑ Vagal activity	Bradycardia
Arterial chemoreceptor	> 20%	Carotid and aortic bodies	Hypoxia, hypercarbia, acidosis	↑ vagal & slight ↑ sympathetic activity	↑ SVRBradycardia ↑ SVR

Abbreviations: RR, respiratory rate; SVR, systemic vascular resistance.

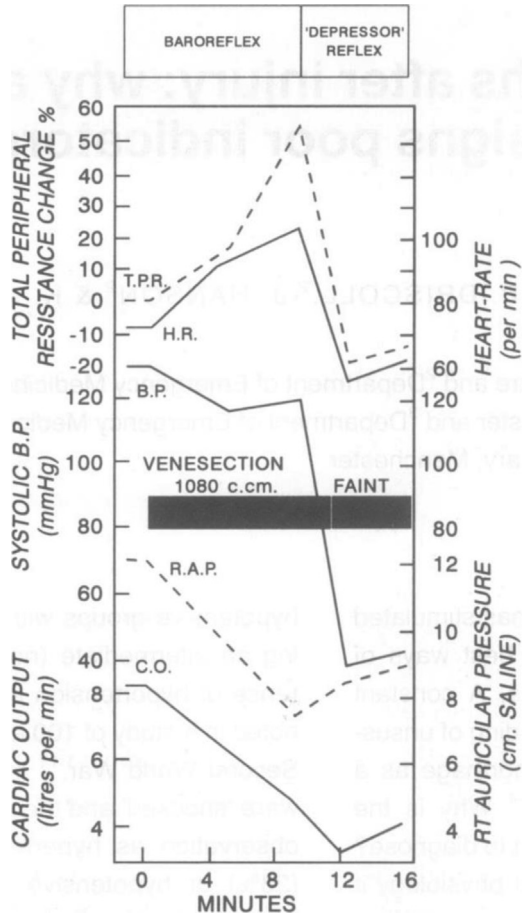

Fig. 2. Changes in HR, systolic arterial blood pressure, cardiac output, peripheral resistance, and right atrial pressure during controlled hemorrhage in male human volunteer.[6]

attenuate (reduce) the biphasic response (autonomic mediated bradycardia and vasodilation), which is protective for end-organ and coronary perfusion. Even hemodynamically stable opioids such as fentanyl can precipitate hemodynamic collapse in patients with propound hypovolemia and should be used judiciously.[11]

Arterial Injury Shock

Arterial injury shock is the physiologic response to a major arterial vascular injury whereby elastic arterial diastolic recoil is impaired across the entire arterial system. This results in immediate and profound hemodynamic instability with a disproportionate loss of diastolic pressure and subsequent reduction in left ventricular coronary perfusion. Characteristically there is minimal or no response to volume resuscitation, as the underlying shock etiology is one of diastolic recoil failure, rather than hypovolemia.

Patients with significant defects to large arterial structures are at risk of this phenomenon. It tends to occur more frequently in penetrating trauma as elastic arterial structures are more resistant to the shear forces of blunt trauma. Arterial injury shock

will lead to a very rapid and profound hemodynamic decompensation, often resulting in cardiac arrest within minutes of injury, unless early prehospital vascular control can be achieved. In comparison, exsanguinating small arterial, venous, solid organ, or bony bleeding often results in a slower physiologic deterioration, with either complete or transient response to volume resuscitation.

Clinical Assessment

A thorough assessment of the primary survey, prodromal events, mechanism/kinematics of injury, and familiarity with bleeding mimics are essential in identifying the etiology of shock in the trauma patient.

The 'hateful eight' of hypovolemia

Compensatory mechanisms, biphasic autonomic responses to hypovolemia, and unreliability of noninvasive blood pressure (NIBP) contribute to the challenges of shock assessment. A broader clinical assessment of the patient, in correlation to the mechanism of injury is key. This clinical assessment is sometimes referred to as the 'hateful eight' and can be used to identify patients with life-threatening hemorrhagic shock (**Box 1**).

Compensated shock

Hypovolemic, cardiogenic, and obstructive shock states are all characterized by a reduced cardiac output. To offset this reduction in stroke volume and maintain cardiac output, the sympathetic nervous system increases the heart rate and stimulates peripheral vasoconstriction diverting blood centrally to restore preload. This is recognized by pale and sweaty skin, prolonged capillary refill time, reduced pulse pressure, and acidosis-driven increased minute ventilation. In these early stages, cardiac output and blood pressure are maintained. Although the blood pressure is maintained, perfusion of peripheral tissues is impaired, and an oxygen debt accumulates.

Distributive shock states may not present with the classic skin changes or tachycardia described earlier. Pathological vasodilatation may prevent compensatory vasoconstriction, resulting in flushed warm peripheries in the early stages. Tachycardia may also be absent in neurogenic shock with high cord lesions, due to unopposed vagal tone.

Decompensated shock

When compensatory mechanisms fail, perfusion to the vital organs becomes compromised and decompensation occurs. The brain relies on a constant blood flow to

Box 1
Clinical signs of hypovolemia

- Air hunger
- Low end-tidal CO_2
- Sweaty/clammy
- Pallor (lips, tongue, palms, soles)
- Venous collapse
- Abnormal pulse (brady or tachycardia)
- Hypotension
- Altered mental status (agitated, unconscious)

maintain function and as blood flow decreases, cognition is impaired, resulting in confusion/agitation. The presence of peripheral pulses and NIBP measurements are unreliable indicators for decompensated shock.

The speed at which decompensation occurs will depend partly on the physiological reserve of the patient and the cause of the shock state. Patients in cardiogenic and distributive shock states have a limited ability to compensate and therefore are liable to decompensate rapidly. Other confounding factors can affect the patient's response to shock (**Table 2**), and a high index of suspicion is essential in these patient groups if shock is to be identified.

'Low flow' or 'no flow' states

Patients with ongoing hemorrhage will deteriorate from decompensated shock into cardiac arrest with no palpable central pulse. During the early stages of hypovolemic cardiac arrest, the patient will be in a low output state, and may have a rapid or normal rate narrow complex pulseless electrical activity. Cardiac activity may still be present on ultrasound and this finding can prioritize management away from chest compressions (which will impede right ventricular filling[13]) and toward volume replacement with blood products. As the patient further declines, the electrocardiographic complexes will widen, slow, and decline into asystole with no cardiac activity on ultrasound (**Fig. 3**): this requires chest compressions. End-tidal CO_2 should be monitored throughout as a surrogate for right ventricular stroke volume and return of spontaneous circulation. Hemorrhage control, oxygenation, and volume resuscitation is the priority for these patients.

Clinical Pitfalls

Palpable pulses. Traditionally, palpation of the carotid, femoral, and radial pulse has been used to estimate the blood pressure and perfusion of trauma patients. A loss

Table 2
Factors affecting the physiologic response to shock

Confounders	Changes in Response to Hypovolemia
Advancing age	Older patients have less physiological reserve and can decompensate earlier. Resting blood pressure is higher and hypotension may be present at systolic blood pressures <110 mm Hg.[12]
Comorbidities	Major cardiovascular, renal, hepatic, and endocrine disorders may tolerate shock states poorly, rapidly accumulating a critical oxygen debt with an increase of risk end-organ injury, dysfunction, and failure.
Medications	Medicines that slow the heart rate and block the sympathoadrenal axis can mask early signs of hemorrhagic shock by preventing a compensatory tachycardia and peripheral vasoconstriction.
Pacemaker	A pacemaker with a fixed rate will limit the ability of the patient to mount a compensatory tachycardia and lead to earlier decompensation.
Younger patients/athletes	The resting heart rate may be in the region of 50 bpm. This should be taken into account when assessing for relative tachycardia.
Pregnancy	Blood may be shunted from the uterine and placental circulation into the maternal circulation to the detriment of the fetus. Significant hypovolemia may occur before signs of shock are evident.
Hypothermia	Hypothermia can reduce RR, HR, and BP independent of hypovolemia.

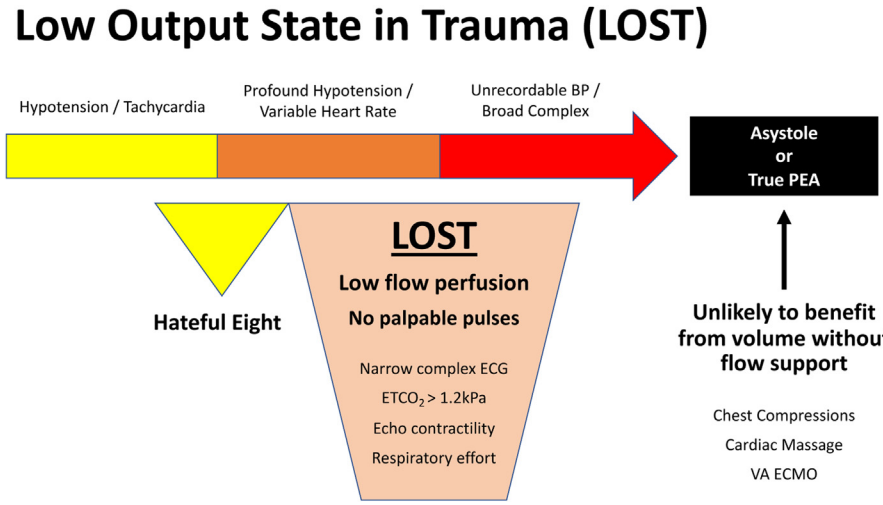

Fig. 3. The physiological spectrum of a low output state due to hypovolemia. PEA, pulseless electrical activity.

of the radial pulse is used as a surrogate marker for the need for fluid replacement in many trauma protocols. However, in some patients, a radial pulse may only become absent when the systolic blood pressure is below 60 mm Hg and therefore this clinical sign may underestimate the degree of shock.[14]

Noninvasive blood pressure. NIBP is standard monitoring for all patients but is prone to several pitfalls. An accurate measurement depends on cuff size and fit and no vascular limb injury. In low output states, an erroneous reading is common (eg, 200/180). Studies demonstrate that it is imprecise when compared to invasive blood pressure monitoring.[15] Interpretation relies on a trend rather than an individual reading and mean blood pressure measured noninvasively is more accurate than systolic blood pressure. The reference ranges for different ages of children need to be available and consideration that older patients have higher baseline blood pressures when well. Pulse pressure is the difference between systolic and diastolic blood pressures. In trauma patients with a blood pressure in the normal range, a narrow pulse pressure is an independent early predictor of active hemorrhage requiring blood product transfusion and surgical intervention for hemorrhage control.[16,17]

Shock index (SI). SI is the heart rate divided by the systolic blood pressure. A SI greater than one is considered to be abnormal. SI performs better than hypotension alone for triaging patients with critical bleeding and need for emergent injury. However, it includes the pitfalls of noninvasive blood measurement and undertriages patients who have a bradycardic response to shock.

Permissive Hypotension

The underlying principles for resuscitation of the shocked patient focus on restoring perfusion and eliminating oxygen debt. However, for patients with active noncompressible torso hemorrhage, contemporary management advocates damage control resuscitation, until definitive hemorrhage control can be achieved.[18] Permissive hypotension is a key component of this approach and describes the permissive acceptance of adequate, but suboptimal blood pressure and end-organ perfusion.

The role of permissive hypotension (palpable central pulse or SBP 80–90 mm Hg) in the management of the actively bleeding trauma patient has become widespread, and is supported by several national guidelines such as the European Guideline on Management of Major Bleeding and Coagulopathy Following Trauma[19] and the National Institute for Health and Care Excellence (NICE) Guidelines for the Management of Bleeding Trauma Patients.[20] Despite this, permissive hypotension remains contentious, with risks when applied generically to trauma patients, especially in the context of TBI.[21]

The NICE guidelines[20] advocate a restrictive approach to volume resuscitation until early definite control of bleeding, titrating volume resuscitation to a palpable central pulse. However, in patients with concurrent TBI, the guidance advocates a restrictive approach if bleeding is the predominant factor, and a less restrictive approach if TBI is the predominant condition. These challenges are compounded by the fact that physiological parameters are of limited diagnostic value in assessing the severity of major hemorrhage[22] and that up to 45% of UK trauma patients with TBI are intoxicated, making pre-CT exclusion of significant brain injury challenging.[23] We also know that hypotension in TBI increases mortality,[24,25] and both the Brain Trauma Foundation (SBP >110 mm Hg) and Association of Anaesthetists (MAP 80 mm Hg) advocate normal hemodynamic targets to maintain penumbral cerebral perfusion and minimize secondary brain injury.[26,27]

A 2014 Cochrane meta-analysis examining the timing and volume of fluid administration in hemorrhage concluded there was 'no evidence for or against the use of early or larger volume intravenous fluid administration in uncontrolled haemorrhage.'[28] There are several relevant limitations for the studies included within this Cochrane review; exclusion of TBI, utilization of large volumes of cold crystalloid fluids as the comparator group, predominantly younger patients with a high incidence of penetrating trauma, immortality bias, and exclusion of patients with comorbidities.[21] The vast majority of UK trauma is multisystem and blunt in nature (penetrating trauma accounting for <3% of cases),[29] making the extrapolation of data supporting permissive hypotension for penetrating trauma challenging.[30] The studies analyzed by the Cochrane meta-analysis also do not reflect modern trauma care with early access to balanced blood product resuscitation, a high incidence of concurrent TBI, and an aging trauma population with increasing comorbidities tolerating periods of hypoperfusion poorly.

Hypotension should be recognized as the decompensation of a bleeding trauma patient, often requiring immediate blood product resuscitation to maintain adequate cerebral and end-organ perfusion. We need to consider patient factors (age, comorbidities), injury factors (type and severity), and duration of shock when providing individualized modern trauma care. The remaining components of damage control resuscitation should be aggressively pursued with the normalization of coagulopathy, avoidance of hypothermia/hypocalcemia/hyperkalemia, and rapid surgical or interventional radiologically achieved hemorrhage control.

BLEEDING MIMICS

Prodrome and Comorbidities

Prodromal events such as ingestion of illicit substances, exertion, fear, and severe anxiety can all precipitate a sympathomimetic response to injury, mimicking early shock physiology. These mimics are short-lived and readily managed with reassurance, time, and judicious doses of analgesics.

Incisional trauma, breach of pleural and peritoneal membranes, and blood irritation of thoracic and abdominal cavities can precipitate a profound vagal reflex mimicking a

biphasic C-fiber–mediated response to major hemorrhage. As with prodromal mimics, vagal reflexes may resolve with reassurance, time, and analgesia.

Cardiogenic Shock

Cardiogenic shock occurs when there is myocardial dysfunction in the presence of adequate left ventricular filling pressures, resulting in inadequate end-organ oxygen delivery. Without intervention, cardiogenic failure will result in reduced cardiac output, impaired coronary perfusion, and a spiral of worsening myocardial function (**Fig. 4**).

Myocardial dysfunction may be preexisting (ischemic heart disease, cardiomyopathy, heart failure), but in the context of major trauma can be secondary to a variety of factors; commotio cordis, cardiac contusion, myocardial injury, coronary injury, valvular disruption, and cardiomyopathy.

Commotio cordis

Commotio cordis or 'concussion of the heart' is caused by a blunt impact to the chest wall over the heart during the vulnerable phase of the cardiac cycle.[31] This can result in an electrophysiological R-on-T phenomenon causing ventricular fibrillation. There is no mechanical damage to the myocardium or surrounding organs, and cardiac arrest should be managed as per advanced life support guidance.

Cardiac injury

High-energy blunt trauma to the thorax can result in myocardial contusion, valvular disruption, coronary dissection, and rib fracture-associated penetrating trauma to the heart and pericardium. Myocardial contusions may impair contraction, and in severe cases result in cardiogenic shock. The right ventricle is most commonly injured in blunt trauma because of its position behind the sternum. Diagnosis includes ECG,

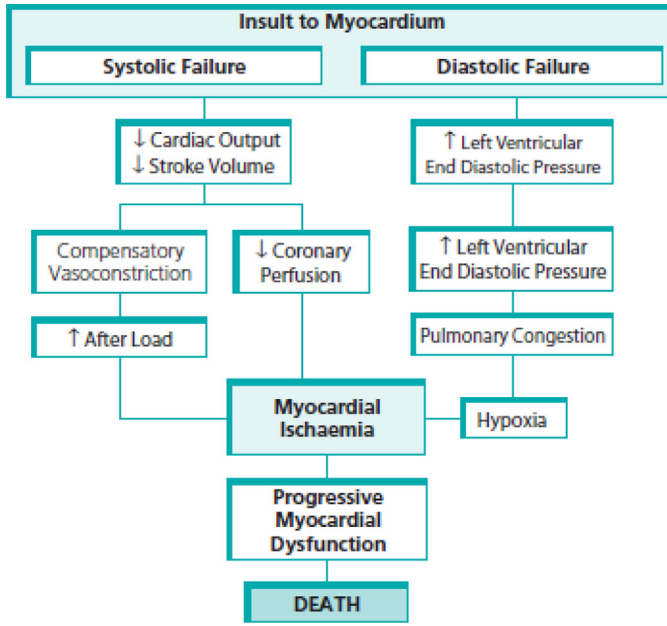

Fig. 4. Cardiogenic shock. Tim Nutbeam, Matthew Boylan, ABC of Prehospital Emergency Medicine, Wiley, 2013.

cardiac troponins, and echocardiography. ECG changes include nonspecific ST-segment and T-wave changes, conduction delays, dysrhythmias, and sinus tachycardia.[32] Cardiac troponins are highly sensitive for myocardial injury, but may also be raised due to hypoperfusion from hypovolemia or other causes of hypotension.[32] A normal 12-lead ECG and troponin blood test excludes cardiac contusion. Echocardiography is a useful screening tool for patients who develop cardiac complications or unresponsive hypotension after blunt chest wall trauma. Segmental wall motion abnormalities representing regional hypokinesia and RV dilatation are characteristic findings in severe cardiac contusion. Echo is also helpful to diagnose valvular disruption or pericardial effusions in the context of coronary lesions and myocardial lacerations.[32]

Penetrating trauma to the thorax may cause a pericardial effusion, but can also cause coronary disruption, valvular/papillary injury, and damage to the conduction system of the heart. The assessment and clinical management of penetrating trauma to the chest remains unchanged (see below for more detail in the obstructive shock section), but in patients with refractory shock states and conduction abnormalities, direct cardiac injury should be considered.

Cardiomyopathy

Cardiomyopathy is defined as a disease of the myocardium that impairs its ability to contract due to dilation, thickening, or stiffening. Although patients may have preexisting cardiomyopathy, this condition can also occur acutely in the context of major trauma, complicating the clinical assessment and management of patients with shock in trauma.

Following a TBI or multisystem trauma with a high tissue-injury burden, excess catecholamines are released, depleting the myocardium of energy stores (**Fig. 5**),[33] impairing contractility and resulting in a stunned myocardium with regional wall motion abnormalities in the basal and midventricular regions. This pattern of myocardial impairment is referred to as a Takotsubo cardiomyopathy.[34–37] Concurrent hypoxia

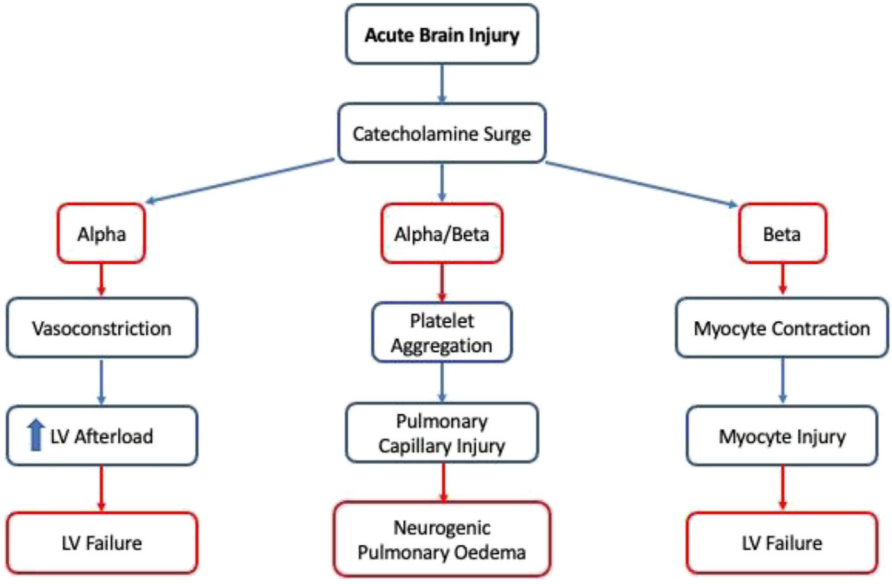

Fig. 5. Acute brain injury and associated cardiorespiratory compromise.

and hypercarbia from concussive injury to the brain, resulting in abnormal ventilation or apnea, can compound this catecholamine-driven myocardial injury.[38] Recognition of a stunned myocardium/cardiomyopathy can be challenging, and a thorough clinical examination, assessment of the patient's physiology, and correlation with echocardiography may guide diagnosis. Management requires judicious volume resuscitation and inotropic support as required.

Patients with sustained or profound myocardial hypoperfusion due to prolonged hypovolemia, particularly on a background of preexisting ischemic heart disease, may sustain irreversible myocyte injury. This can result in delayed (24–48 hours) cardiac failure and multiorgan dysfunction despite aggressive and early resuscitative efforts.

Obstructive Shock

Extracardiac obstruction to blood flow can cause impaired ventricular diastolic filling or excessive right ventricular afterload, impairing cardiac output and resulting in end-organ oxygen debt. Causes include cardiac tamponade, tension pneumothorax, and pulmonary embolism.

Cardiac tamponade is most commonly caused by incisional trauma to the torso including the supraclavicular region, lower neck, thorax, or epigastrium. There have also been case reports of pericardial effusion and tamponade caused by blunt trauma, either due to rib fractures and bone-fragment injury, or due to blunt cardiac chamber lacerations or coronary vessel disruption.[39] Cardiac tamponade must be a differential diagnosis for all patients with decompensated shock in the context of penetrating trauma. Signs of cardiac tamponade include tachypnea, cardiovascular compromise, electrical alternans (alternating height of consecutive QRS complexes), pulsus paradoxus (fall of systolic blood pressure of >10 mm Hg during the inspiratory phase of respiration), muffled heart sounds, and distended neck veins (absent if concurrent hypovolemia). In patients with compensated shock, further imaging may guide diagnosis: CT or bedside transthoracic echocardiography with visualized effusion and right atrial/ventricular collapse have a high specificity.[40] For patients who are periarrest or in cardiac arrest, immediate resuscitative thoracotomy is indicated within 15 minutes of loss of vital signs.[41] Note that a proportion of neurologically intact survivors from prehospital thoracotomy have a presenting rhythm of asystole, and therefore this should not be used as a prognostic determinant for this cohort of patient.[42]

Tension pneumothorax in the spontaneously ventilating patient has a very different pathophysiology to the positive pressure ventilated patient.[43] In spontaneous ventilation, a one-way pleural defect is required for gas accumulation in the intrapleural space, and progressive tachypnea, dyspnea, pleuritic chest pain, and hypoxia (masked by high-flow oxygen) are the presenting features. Hemodynamic compromise only occurs at the point of hypoxic cardiac arrest and concurrent hypovolemia should always be considered in spontaneously ventilating patients with a tension pneumothorax and cardiovascular collapse. In positive pressure ventilated patients, a nonvalved pleural defect will suffice for gas accumulation in the intrapleural space, increasing the incidence of tension physiology. This will result in rapid hemodynamic compromise, high ventilatory pressures, decreased oxygen saturations, and subsequent cardiovascular collapse. Both distended neck veins and a deviated trachea are unreliable and rarely identified signs of a tension pneumothorax.[44] The acuity of cardiovascular collapse is also related to the volume status of the patient, with concurrent hypovolemia decreasing this threshold in patients with a tension pneumothorax when positive pressure ventilated.

Dynamic hyperinflation (gas trapping) due to excessive positive pressure ventilation in patients with severe bronchospasm may also reduce venous return sufficient to cause an obstructive shock state, particularly in the presence of hypovolemia. Consideration of mechanical ventilatory strategies and gentle bag-valve ventilation of hypovolemic patients can minimize hemodynamic compromise by reducing the inspiratory pressure.

Distributive Shock

A reduction in peripheral vascular resistance can unmask compensated hypovolemia, or in the context of profound vasodilation/vasoplegia will decrease right ventricular preload, resulting in reduced cardiac output and subsequent end-organ oxygen debt. Septic, anaphylactic, and neurogenic shock are the most common subtypes of distributive shock.

Neurogenic shock occurs secondary to damage to the spinal cord above the level of T10, with loss of sympathetic outflow leading to unopposed peripheral vasodilatation. Injuries to the spinal cord above T6 may also damage the cardioaccelerators and result in bradycardia, compounding this vasoplegic-mediated neurogenic shock. Clinical suspicion of spinal cord injury with neurogenic shock is supported by spinal pain/tenderness, limb paralysis and paresthesia, flaccid areflexia, diaphragmatic breathing, flushed warm skin despite hypotension, and priapism. The timing of onset of neurogenic shock can vary considerably (minutes to hours post injury) and can also develop in patients with anatomical injuries below T5.[45] The whole length of the sympathetic cord supplies innervation to the vasculature and therefore interruption at any level has the capacity to produce the vasoplegic shock independent of heart involvement. Log-rolling or repositioning may precipitate vasovagal stimulation. In the early phases of neurogenic shock, there may be tachyarrhythmias making it hard to differentiate from hypovolemic shock.[46] The hypotension that characterizes neurogenic shock may lead to hypoperfusion of the spinal cord with subsequent ischemia and secondary injury. Treatment after exclusion of hypovolemia on imaging and fluid resuscitation

Table 3
Summary of trauma shock etiologies, and diagnostic features

Etiology of Shock	Clinical Diagnosis
Tension pneumothorax	Respiratory compromise Chest ultrasound, chest x-ray, or thoracostomy
Hypovolemia	Hateful eight signs Blood gas Hb (very late sign) eFAST, chest x-ray/pelvic x-ray
Cardiac tamponade	Mechanism Distended neck veins Cardiac ultrasound
Cardiac contusion	Mechanism Abnormal ECG, troponin T, echo
Neurogenic shock	Clinical signs of spinal cord injury – motor and sensory deficit Vasodilatation – warm peripheries, distended peripheral veins, priapism Bradycardia with hypotension
Traumatic brain injury	External signs of head injury, prehospital low GCS Alternating bradyarrhythmia and tachyarrhythmia
Soft tissue injury	Mechanism, signs of limb injury Unresponsive tachycardia

to maintain blood volume, includes preventing fluid overload, monitoring urine output/fluid balance, and judicious use of vasopressors to maintain an MAP of 85 to 90 mm Hg.[47]

Rewarming hypothermic major trauma patients with cutaneous vasoconstriction, or administration of anesthetic agents with vasodilatory profiles can precipitate cardiovascular collapse in patients with compensated hypovolemia. This should be anticipated when rewarming a bleeding polytrauma patient and treated with balanced blood product resuscitation as required. The use of vasopressors to offset this phenomenon should be avoided, as may worsen end-organ microcirculatory perfusion, oxygen delivery, and trauma-induced coagulopathy.

Table 3 summarizes the common causes of shock in trauma patients.

As **Fig. 1** illustrates, there are also several medical conditions that can mimic bleeding and may also account for the primary cause of the injury. Examples include relative hypovolemia from dehydration or sepsis causing collapse, drug use/overdose leading to behavioral disturbance, and cardiac ischemia or arrhythmia leading to syncope. The importance of interpreting the prodromal events, patient comorbidities, physical examination findings, and response to treatment are all crucial in recognizing these trauma mimics and managing them appropriately.

SUMMARY

Shock in trauma is complex and varied in presentation and etiology. The physiological response to major hemorrhage is dependent on a variety of autonomic reflexes, mechanism of injury, bleeding source, and the patient's baseline physiology. A number of bleeding mimics can also result in trauma shock, occurring in isolation or in combination with major hemorrhage. A thorough understanding of how patients respond to bleeding, the differential diagnoses, and recognition of individualized tolerability to shock is essential when assessing and resuscitating major trauma patients.

CLINICS CARE POINTS

- Hypovolemia may induce bradycardia via cardiac C-fiber and arterial chemoreceptor reflexes.
- Penetrating injury without tissue damage may produce a biphasic hemodynamic response.
- Traumatic brain injury or multisystem trauma with a high tissue-injury burden may cause release of excess catecholamines resulting in a stunned myocardium and cardiogenic shock.
- Rewarming hypothermic major trauma patients can precipitate cardiovascular collapse in patients with compensated hypovolemia.

DISCLOSURE

None of the authors have any financial or professional conflicts of interest to declare.

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