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Review Article

Shock in Surgery

Siniša Franjić*

Independent Researcher, Croatia

***Corresponding Author:** Siniša Franjić, Independent Researcher, Croatia.

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Abstract

In everyday speech, we often use the word "shock" with meanings that differ from its medical definition. Shock in the medical sense means a life-threatening condition of disturbed blood circulation that can have various causes and mechanisms of occurrence.

Keywords: *Shock; Blood; Haemorrhage*

Introduction

Shock is a pathological, life threatening condition in which the oxygen supply to the tissues of the body fails [1]. The cause is usually one of the following:

- Hypovolaemia (bleeding)
- Sepsis
- Acute anaphylaxis: from allergy or drug reaction
- Neurogenic (after spinal trauma)
- Heart failure (left ventricular failure).

Shock is a life-threatening medical emergency that requires rapid intervention by the health care team [2]. A person in shock is in a state of circulatory collapse. Without immediate treatment, organ damage and death occur.

Shock is defined as inadequate tissue perfusionin which there is insufficient delivery of oxygen and nutrients to the body's tissues and inadequate removal of waste products from these tissues or, more simply, an imbalance between oxygen supply and demand. The decrease in tissue perfusion leads to impaired cellular metabolism, which in turn leads to tissue hypoxia. Tissue hypoxia results in hypo-perfusion of vital organs and cell death. All body systems are affected by reduced oxygen supplies. The resulting injury to the body can be treated in the early stages of shock, but if shock is prolonged, it leads to irreversible cell damage and death. By the time blood pressure drops, cellular and tissue damage have already occurred. Therefore, it is important to identify patients at risk for shock and carefully monitor them to detect early signs and symptoms.

The findings in a patient with shock are markers of hypoperfusion and the body's attempt to compensate [3]. Signs of early shock may be subtle and must be carefully sought to prevent the cascading events. It is imperative to underscore patently abnormal vital signs as a late finding in shock, particularly hypotension. Patients with a normal/near normal blood pressure may still have profound hypoperfusion; failure to recognize this will lead to increased mortality and morbidity. Findings may vary between the forms of shock and can provide clues as to the underlying etiology.

The autonomic nervous system is able to alter heart rate and peripheral vascular resistance in response to changes in blood pressure detected by the carotid sinus and aortic arch baroreceptors [4]. Changes in systemic vascular resistance may alter venous return by changing the amount of fluid circulating in the cutaneous and splanchnic vascular beds. Venous return determines stroke volume; increasing venous return causes an increase in stroke volume, the heart acting as a permissive pump (Starling's law: the output depends on the degree of stretch of the heart muscle at the end of diastole).

Volume regulation is achieved by the kidney, in particular by the regulation of sodium loss by the renin– angiotensin–aldosterone system and antidiuretic hormone (ADH) produced by the posterior pituitary which controls water loss in the renal tubules and collecting ducts. In addition, a fall in circulating volume prompts the sensation of thirst, stimulating increased fluid intake.

Shock may be produced as the result of severe infection from either Gram-positive or, more commonly, Gram-negative organisms. The latter are seen particularly after colonic, biliary and urological surgery, and with infected severe burns. The principal effect of endotoxins is to cause vasodilation of the peripheral circulation together with increased capillary permeability. The effects are partly direct and partly due to activation of normal tissue inflammatory responses such as the complement system and release of cytokines such as tumour necrosis factor (TNF).

Disseminated intravascular coagulation (DIC) results from activation of the clotting cascade and may lead to blockage of the arterial microcirculation by microemboli. Fibrin and platelets are consumed excessively, with resultant spontaneous haemorrhages into the skin, the gastrointestinal tract, the lungs, mouth and nose.

Most of the blood volume at rest is contained within the venous system [5]. The effect of the return of this venous blood to the heart produces ventricular end-diastolic wall tension, a major determinant of cardiac output. Gravitational shifts in blood volume distribution are rapidly compensated for by active and passive alterations in venous capacity. In the normal heart, most changes in cardiac output are a reflection of alterations in preload. Changes in position, intrathoracic pressure, intrapericardial pressure, and circulating blood volume produce major changes in cardiac output.

The normal circulating blood volume is maintained within narrow limits by balancing salt and water intake with external losses by the kidney's ability to respond to alterations in hemodynamics and the hormonal effects of renin, angiotensin, and antidiuretic hormone. In the acute setting, the changes in the venous tone, systemic vascular resistance, and intrathoracic pressure come into use. In addition, the net effect of preload on the ventricle also responds to the cardiac determinants of ventricular function, including coordinated atrial contraction, which augments ventricular diastolic filling, and tachycardia, which drops the effect of preload on the ventricle by compromising diastolic filling time.

Haemorrhage

The diagnosis is based on finding the signs of hypovolaemic shock - a pale, anxious, sweaty patientwith cold extremities, a rapid, thready pulse, tachypnoea and hypotension [6]. These signs occur when the body redistributes the circulation in an attempt to maintain the blood flow to the vital organs (heart and brain). Other organs, such as the skin, intestine and kidneys, become inadequately perfused and poorly oxygenated. This homeostatic response is brought about by the sympathetic nervous system causing a tachycardia and vaso-

constriction in the extremities. The skin becomes cool and clammy. The systolic blood pressure is usually maintained at first, but the pulse pressure (the pressure difference between systolic and diastolic pressures) may be reduced by a rise in the diastolic pressure. The rate of respiration increases to try to improve oxygenation.

Patients who arrive in the accident and emergency department without overt haemorrhage but who exhibit these signs have almost certainly lost 1 - 2 litres of blood. It is important to remember that young, fit patients can often tolerate considerable blood loss before they develop, often very suddenly, any signs of hypovolaemic shock, whereas elderly patients, especially those on beta-blockers or digitalis, tolerate quite small amounts of blood loss less well.

Clinical examination in conjunction with history and measured losses will assist the assessment of the patient's circulatory status [7]. Throughout the spectrum of the response to haemorrhage, heart rate, arterial pulse pressure and central venous pressure are useful indicators. Assessment of cardiac output and tissue flow, however, is more difficult.

Although it is relatively easy to recognise the effects of profound shock, the early manifestations (tachycardia and peripheral vasoconstriction) are more difficult to recognise. End-organ perfusion can assist in this assessment process and the clinical evaluation of urine output and peripheral circulation are important.

The classic division of grade of shock is further polluted by the differing physiological reactions of different age groups to haemorrhage. Very young children have an undeveloped sympathetic nervous system rendering them unable to influence blood pressure and flow significantly. In addition to a fixed stroke volume, tachycardia is their only response. Young fit adults can mount such a significant compensatory response that blood pressure may only be affected after there is a loss of a third of the patient's blood volume. In contrast, elderly patients have a limited cardiac reserve and with a relatively fixed vascular tree, signs of severe shock can become evident after a loss of only 10% of blood volume.

Haemorrhage may present with subtle signs or with shock depending on the site and rate of blood loss [8]. Mild acute blood loss is characterised by cold, clammy skin and decreased capillary refill of more than two seconds due to cutaneous vasoconstriction, tachycardia and orthostatic hypotension. With greater blood loss over thypotension and oliguria develop. Anabdominal examination is helpful if there is progressive distension. The output in drains can be misleading because the drains are often blocked with clot when there is active haemorrhage. It is important to note that acute haemorrhage does not immediately reduce the haematocrit level; serial haematocrit measurements will show precipitous fall after fluid resuscitation.

Clinical features

Common clinical manifestations of all types of shock include relative hypotension, tachycardia, poor skin perfusion, and organ system dysfunction [9]. Signs of organ system dysfunction include oliguria (renal hypoperfusion) and altered mental status (brain hypoperfusion). In this patient with relative hypotension and tachycardia, we must suspect this patient may be in the early stages of shock. We should determine what his baseline blood pressure and heart rate values are in order to better interpret his current vital signs. He has an elevated serum creatinine which may reflect renal hypoperfusion, but we should determine what his baseline creatinine is. His clinical presentation is concerning for abdominal sepsis and septic shock from perforated viscus.

Regardless of the underlying cause, shock is characterised by an acute alteration of the circulation in which inadequate perfusion leads to cellular damage, dysfunction and failure of major organ systems [10].

The clinical features of shock are so variable that they cannot be used to define the shocked state. Although the terms 'hypotension' and 'shock' are often taken to be synonymous, cellular perfusion may be inadequate despite a normal blood pressure. Perfusion describes

blood flow but also implies the supply of substrates (including oxygen) and the removal of waste products. Use of the terminology 'inadequate tissue perfusion' rather than 'reduced perfusion' is important since blood flow and substrate supply may be increased in hypercatabolic states (e.g. trauma and sepsis) and yet inadequate for the demands of the tissues due to increased metabolism and failure to extract substrates from the circulation, especially in septic shock.

In the shocked state, the distribution of blood flow is important. While certain viscera preserve flow through autoregulation (e.g. heart, kidney), others cannot (e.g. skin, gut) and may be hypoperfused preferentially. Intestinal hypoperfusion may occur in the face of a normal pulse and blood pressure and, following a brief hypotensive episode, a prolonged period of intestinal hypoxia may occur, with generation of cytokines and the onset of systemic inflammation.

Clinical assessment (and data gathered from charts and the notes) will usually point to a typical cause [11], but should begin by a targeted examination to establish the likely form of shock. Warm peripheries will point to a 'distributive' shock; that is to say where there is a failure of peripheral resistance (BP = (HR × CO) × TPR). This usually indicates systemic inflammatory response ± sepsis, but may occur in anaphylaxis or 'neurogenic shock' (such as spinal cord transection). Cool peripheries with signs of reduced circulating volume (low JVP, signs of dehydration, obvious fluid losses) may point to hypovolaemic shock. Cool peripheries and high JVP suggest a 'pump failure' – this may be intrinsic 'cardiogenic shock' if there has been a cardiac event (MI, arrhythmia) but can also occur secondarily to extrinsic compromise ('obstructive shock'), tension pneumothorax, cardiac tamponade or pulmonary embolism.

Further definitive management depends on the exact cause and any easily reversible causes should take priority. The movement of the patient to a critical care area and the institution of invasive monitoring is invariably required, as established shock is not often rapidly reversible. Hypovolaemic states require expansion of circulating volume; low peripheral resistance states also require fluid replacement (as the circulating volume requirement increases) but often require inotropic support to increase arteriolar tone. Pump failure situations may require a combination of careful pre- and after-load management and in the case of cardiogenic shock, management is very difficult requiring expert cardiological input.

Septic shock

When infections fail to respond or are deemed unlikely to respond to medical and antimicrobial treatments alone, surgical intervention might be needed [12]. The list of surgical infections necessitating surgical management is long, but the most common ones include abscesses, empyema, necrotizing skin and soft tissue infections (NSSTIs), intra-abdominal infections, and Clostridium difficile (C difficile) colitis. Many of these surgical infections can become life threatening by causing sepsis, septic shock and multiple organ dysfunction syndrome (MODS) if not controlled and treated promptly. Spreading can occur through tissue planes (necrotizing infections), abscess/fistulae formation, or through the lymphatic system and the bloodstream. Spread of an infection through the bloodstream (bacteremia) can lead to distant seeding of bacteria and subsequent abscess formation (eg, brain, liver, adrenal glands, heart valves).

Septic shock occurs when severe sepsis is associated with hypotension or hemodynamic instability. When septic shock leads to progressive dysfunction of multiple organs such as the brain (delirium), lungs (hypoxia and respiratory failure), heart (hypotension, pulmonary edema), and kidneys (oliguria and renal failure), it is referred to as multiple organ dysfunction syndrome (MODS). Bacteremia is the presence of viable bacteria in blood, often documented through blood cultures. Transient bacteremia (eg, following dental work) is common, benign, and self-limited. It usually has no clinical implications except in patients with damaged heart valves; cardiac, vascular, or orthopedic implants; or impaired immunity. Bacteremia happening in a patient with an uncontrolled source of infection can be devastating and may result in severe sepsis, septic shock, and MODS.

Transport

Modern studies have consistently shown that shock or compromised delivery of life-sustaining oxygen (as evidenced by regional or systemic markers of anaerobic metabolism) are associated with surgical complications, organ failure, and death [13]. Entire military and civilian trauma systems, have been built to deliver systematic and aggressive care within Golden Hour after injury. Observed decreases in trauma-associated multiorgan failure and mortality are likely attributable to early resuscitation. The principle of prompt intervention where oxygen delivery is impaired has sound physiologic basis and support from decades of observations that reductions of delays improve survival.

Observational studies have documented an important second peak in mortality between one and six hours after injury. Many of these injuries may be responsive to timely control of hemorrhage and restoration of oxygen delivery.

Recent studies of jurisdictions that place a strong emphasis on aggressive prehospital care were not able to show a difference between aggressive field resuscitation and rapid transport approaches. It is possible that a combination of technical factors leading to inadvertent hypoxia or hyperoxia, aspiration, hypocapnea, hypotension, or intracranial hypertension during intubation can compound or exacerbate the primary injury and compromise recovery. Until these factors are better characterized individually, it is reasonable to apply advanced life support measures in the prehospital setting selectively and with care to avoid hypoxra and hypotension and to continue to expedite patients' transfer to definitive care.

Conclusion

Blood loss is a major factor in the onset of shock. External bleeding is sometimes unreasonably considered strong (because blood is seen), while internal is unjustifiably underestimated (because blood is not seen). Shock can be caused by various diseases. It occurs when the "pump" of the bloodstream fails, ie when the heart is unable to create enough pressure to move the blood satisfactorily. The most common cause of this condition is a heart attack. Shock can also be caused by an allergic reaction to medications or an insect bite. Shock can occur in diseases that lead to large fluid loss through vomiting, diarrhea and sweating.

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