Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

SHOCK

Methodical recommendations
to prepare 5th year students for practical classes
Discipline "Emergency and urgent medical care"

Kharkiv – 2021

UDC 616.6-009.831(072)
S55

Reviewers:

Dubivska S. S. – Doctor of Medical Sciences, Professor of the Emergency Medicine, Anesthesiology and Intensive Care Department, Kharkiv National Medical University, Kharkiv;
Tykhonova T. M. – Doctor of Medical Sciences, Full Professor of the Internal Medicine Department, V. N. Karazin Kharkiv National Unyversity.

Approved for publication by the decision of the Scientific and Methodical Council of V. N. Karazin Kharkiv National University (Protocol № 4 of December 16, 2021)

S55 **Shock**: methodical recommendations to prepare 5th year students for practical classes Discipline "Emergency and urgent medical care" / compilers Mariia S. Matvieienko, Igor V. Belozorov, Nadiia V. Baranova. – Kharkiv : V. N. Karazin KhNU, 2021. – 48 p.

The methodical recommendations set out the main aspects of the management of patients with shock. For 5th year students to prepare for practical classes in the discipline "Emergency and urgent medical care".

UDC 616.6-009.831(072)

CONTENTS

LIST OF ABBREVIATIONS

ICU			Intensive Care Unit
SIRS			Systemic inflammatory response syndrome
MOF			Multiple organ fealure
BP			Blood pressure
HR			Hart rate
RR			Respiratory rate
CO			Cardiac output
SV			Stroke volume
SVR			Systemic vascular resistance
ED			Emergency department
PE			Pulmonary embolism
ACTH			Adrenocorticotropic hormone
CI			Cardiac index
RBC			Red blood cell
BUN			Blood urea nitrogen
CNS			Central nervous system
SvO_2			Mixed venous oxygen saturation
NO			Nitrouse oxide
LV			Left ventricular
RV			Right ventricular
AMI/MI			Acute myocardial infarction
ECG			Electrocardiogram
CXR			Chest radiograph
IV			Intravenous
CO_2			Carbon dioxide

1. BASIC KNOWLEDGE, SKILLS, HABITS NECESSARY FOR STUDYING THE TOPIC

Names of previous disciplines	Acquired skills
Foreign Language	Be able to work with foreign sources to obtain up-to-date data on methods of diagnosis and treatment different variants of shock
Medical informatics	Apply modern computer programs and be able to work with them, have statistical methods for processing the results of clinical trials, analyze research results, be able to evaluate and interpret the results of clinical trials
Human anatomy Normal physiology Histology, cytology and embryology	Know the normal structure, functions and regulation of the cardiovascular, pulmonary, central nervous system understand and determine the relationship of its structure and functions with other organs and systems of the human body
Pathomorphology Pathophysiology	Know the typical pathological processes: mechanisms of development, changes in the human body, compensatory reactions of the body, the development of connections that have the character of "cause-effect", in the pathology of the whole organism.
Pharmacology	Be able to navigate the range of drugs. Know the mechanisms of action of drugs, their pharmacodynamics, indications and contraindications to their use. Know the features of clinical pharmacology of drugs used in the treatment of hypovolemic, cardiogenic, distributive and obstructive shock, features of the pharmacological action of these drugs in different categories of patients. Make a reasonable choice of individual drugs and treatment regimens taking into account the principles of evidence-based medicine, optimization of treatment regimens, evaluate the effectiveness and safety of pharmacotherapy taking into account the individual characteristics of the patient, the presence of comorbidities.
Propaedeutics of internal medicine	Conduct a physical examination of patients, analyze the results of basic laboratory and instrumental research methods. Identify the leading syndromes and symptoms. Be able to make a differential diagnosis, substantiate and formulate a diagnosis on the basis of physical examination and data of additional methods.

The student should know:

- anatomy and functions of the cardiovascular sistem;
- anatomy and functions of the pulmonary sistem;
- definition and epidemiology of shock, kind of shock;
- main causes and pathophysiology of shock;
- difference between compensated, uncompensated and irreversible stage of shock;
- clinical features of different types of shock;
- diagnostics, peculiarities of the course, intensive therapy for various types of shock (hemorrhagic, traumatic, burn, anaphylactic, septic, dehydration, cardiogenic).
- the different therapeutic approaches to the various types of shock.

The student should be able to:

- be familiar with the new classification of types of shock;
- understand the different ethyology of the four main categories of shock;
- understand the different pathogenesis and pathophysiology of the four main categories of shock;
- conduct a diagnostic of any type of shock in patients;
- conduct the difference between compensated, uncompensated and irreversible stage of shock
- to make a differential diagnosis of different types pf shock;
- to make a plan for managment of the patient.
- conduct a treatment of any type of shock in patients;
- define hypovolemic shock;
- define cardiogenic shock;
- define distributive shock;
- define obstructive shock.

2. INTRODUCTION

Shock in its various forms presents a very common challenge in the Intensive Care Unit (ICU). Over the past decade, we have developed new terminology to better understand shock.

In the first descriptions of shock the focus was exclusively on traumatic hemorrhagic shock, but later this changed and five different types of shock came to be distinguished. Although it is true that all types of shock can lead to the same final stage of multiorgan failure as a result of the imbalance between

oxygen demand and supply, the differences in their pathogenesis and pathophysiology make it desirable to change their classification, partly for teaching purposes, but also, especially, because different therapeutic measures are needed for the different types of shock.

In an attempt to develop a common language, a consensus conference of the Society of Critical Care Medicine and the American College of Chest Physicians was held in August 1991 to produce a series of universal definitions for the systemic inflammatory response syndrome (SIRS). This interplay of cellular and systemic responses, which may be modulated by cytokines, may replace the terms septic, cardiogenic, hypovolemic, distributive, and obstructive shock. This enhanced understanding of the pathophysiology of shock syndromes and SIRS may not only give us a common language but also may aid in the development of treatment protocols. We now understand that the immediate recognition of and institution of treatment for SIRS and shock are paramount in the ICU.

3. DEFINITION

Shock is a multifactorial syndrome resulting in inadequate tissue perfusion and cellular oxygenation affecting multiple organ systems.

Shock defines a state in which the delivery of oxygen and nutrients to the tissue is insufficient to meet basal metabolic needs, leading to tissue hypoxia, and, if persistent, to multiple organ fealure (MOF) and death. Shock results from tissue hypoperfusion and microcirculatory dysfunction.

4. CLASSIFICATION

There are only four major categories of shock, each of which is mainly related to one of four organ systems [Fig. 1].

Classification of types of shock

- Hypovolemic shock
- Distributive shock
- Cardiogenic shock
- Obstructive shock

Hypovolemic shock relates to the blood and fluids compartment while distributive shock relates to the vascular system; cardiogenic shock arises from primary cardiac dysfunction; and obstructive shock arises from a blockage of the circulation. Hypovolemic shock is due to intravascular volume loss and is treated by fluid replacement with balanced crystalloids. Distributive shock, on the other hand, is a state of relative hypovolemia resulting from pathological redistribution of the absolute intravascular volume and is treated with a combination of vasoconstrictors and fluid replacement.

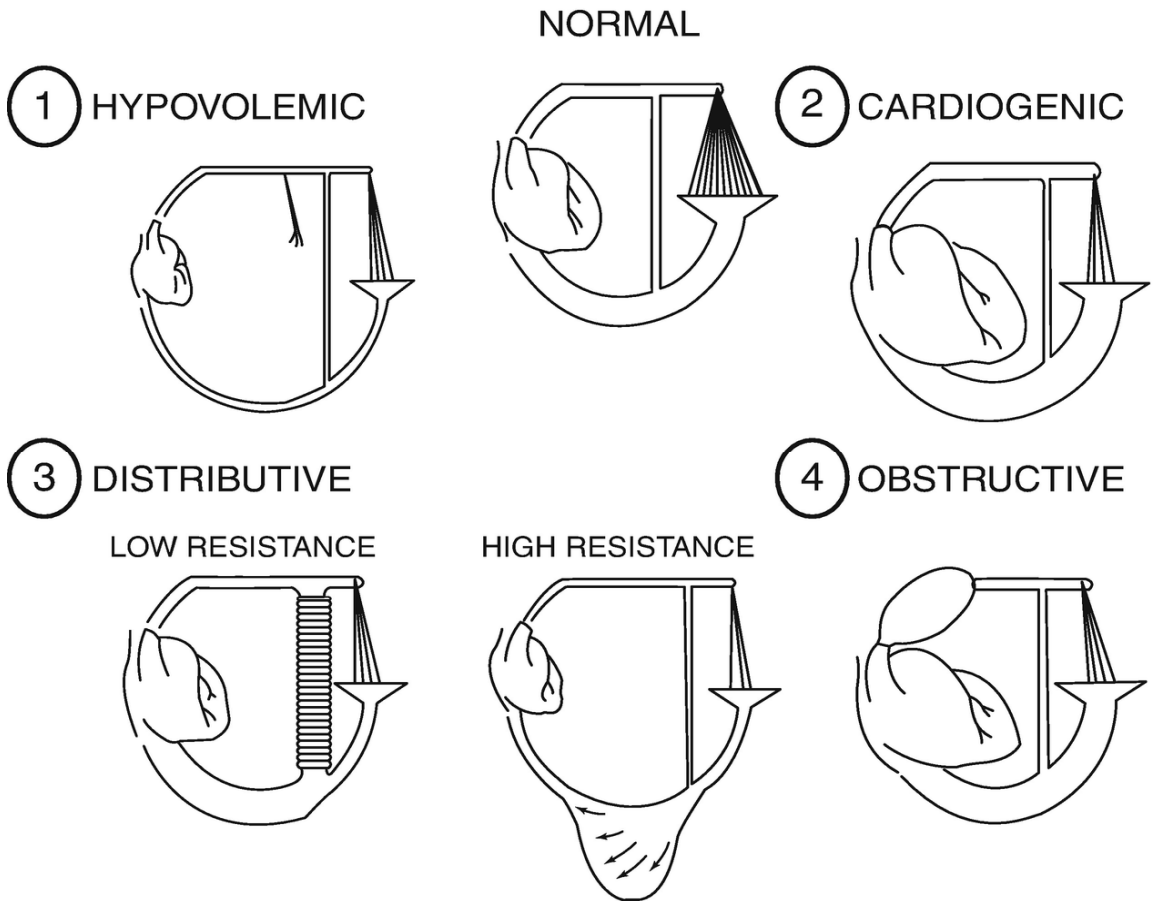
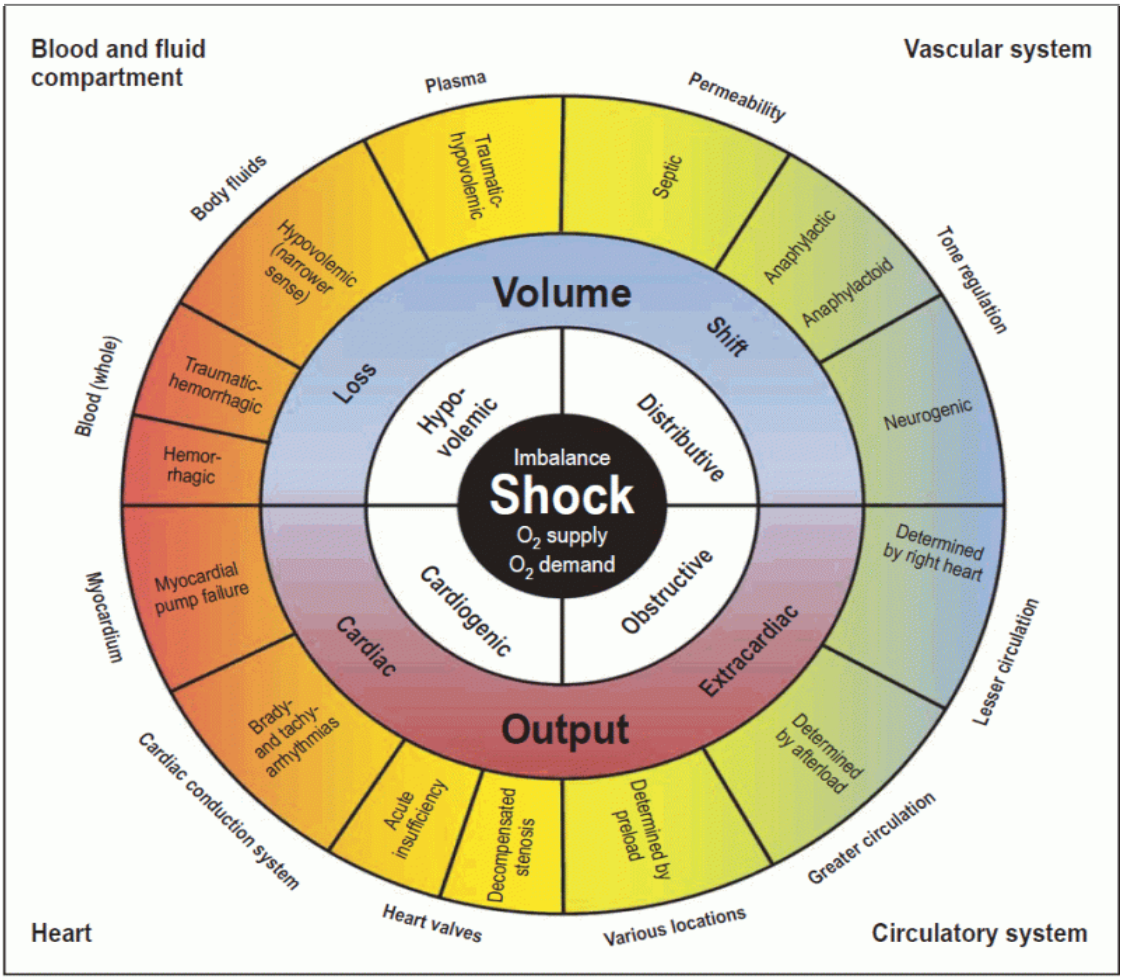


Fig. 1. The types of shock [1]

Cardiogenic shock is due to inadequate function of the heart, which shall be treated, depending on the situation, with drugs, surgery, or other interventional procedures. In obstructive shock, hypoperfusion due to elevated resistance shall be treated with an immediate life-saving intervention [Fig. 2].



Synoptic view of the four types of shock (inner, white field) with the organ systems primarily associated with them (outer corners), sites and mechanisms of manifestation (outside the circle), and pathogenetic and pathophysiologic features (outer and middle sectors of the circle). To maintain clarity, mixed types of shock are not depicted.

Fig. 2. Synoptic view of the four types of shock [5]

5. BASIC CARDIAC PHYSIOLOGY

Blood pressure (BP) is defined as the force that blood exerts against the inner walls of blood vessels. It most commonly refers to pressure in arteries supplied by the aortic branches, even though it actually occurs throughout the vascular system [Fig. 3].

Cardiac output (CO) is the blood volume the heart pumps through the systemic circulation over a period measured in liters per minute.

Stroke volume (SV) is defined as the volume of blood discharged from the ventricle with each contraction. An average adult male's stroke volume is about 70 mL. Stroke volume is classically determined by preload, myocardial contractility, and afterload.

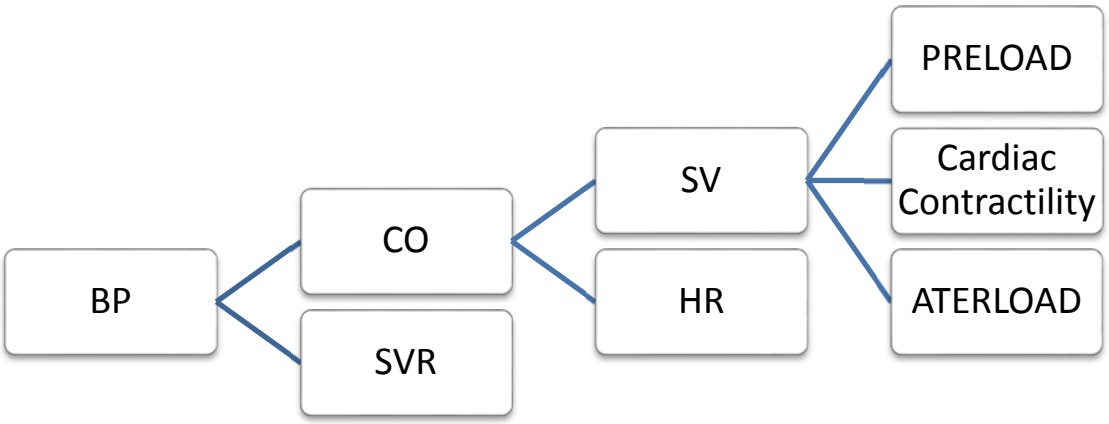


Fig. 3. Components of blood pressure summary

Preload the volume of venous blood return to the left and right sides of the heart, is determined by venous capacitance, volume status, and the difference between mean venous systemic pressure and right atrial pressure. This pressure differential determines venous flow.

The venous system can be considered a reservoir, or capacitance, system in which the volume of blood is divided into two components:

1. *The first component* represents the volume of blood that would remain in this capacitance circuit if the pressure in the system were zero. This component does not contribute to the mean systemic venous pressure.
2. *The second component* represents the venous volume that contributes to the mean systemic venous pressure. Nearly 70 % of the body's total blood volume is estimated to be located in the venous circuit. Compliance of the venous system involves a relationship between venous volume and venous pressure. This pressure gradient drives venous flow and therefore the volume of venous return to the heart. Blood loss depletes this component of venous volume and reduces the pressure gradient; consequently, venous return is reduced. The volume of venous blood returned to the heart determines myocardial muscle fiber length after ventricular filling at the end of diastole. According to Starling's law, muscle fiber length is related to the contractile properties of myocardial muscle.

Myocardial contractility is the pump that drives blood loss pathophysiology early circulatory responses to blood loss are compensatory and include progressive vasoconstriction of cutaneous, muscular, and visceral circulation to preserve blood flow to the kidneys, heart, and brain. The usual response to acute circulating volume depletion is an increase in heart rate in an attempt to preserve cardiac output. In most cases, tachycardia is the earliest measurable circulatory sign of shock. The release of endogenous catecholamines increases peripheral vascular resistance, which in turn increases

diastolic blood pressure and reduces pulse pressure. However, this increase in pressure does little to increase organ perfusion and tissue oxygenation.

Afterload, also known as **peripheral vascular resistance**, is systemic (SVR). Simply stated, afterload is resistance to the forward flow of blood. For patients in early hemorrhagic shock, venous return is preserved to some degree by the compensatory mechanism of contraction of the volume of blood in the venous system. This compensatory mechanism is limited. The most effective method of restoring adequate cardiac output, end-organ perfusion, and tissue oxygenation is to restore venous return to normal by locating and stopping the source of bleeding. Volume repletion will allow recovery from the shock state only when the bleeding has stopped. At the cellular level, inadequately perfused and poorly oxygenated cells are deprived of essential substrates for normal aerobic metabolism and energy production. Initially, compensation occurs by shifting to anaerobic metabolism, resulting in the formation of lactic acid and development of metabolic acidosis. If shock is prolonged, subsequent end-organ damage and multiple organ dysfunction may result.

6. THE MAIN CAUSES, PATHOGENESIS AND ORGAN RESPONSE

Shock may result from:

Common causes
Hypovolemic shock
Hemorrhage (e.g., internal and external bleeding, severe trauma)
Volume depletion (e.g., vomiting, diarrhea, diuretic over-usage,
Ketoacidosis, burns)
Internal sequestration (ascites, pancreatitis, intestinal obstruction)
Inadequate fluid administration (even if the patient clinically appears to be overloaded with fluid)
Adrenal insufficiency
Hypothermia
Hypocalcemia
Cardiogenic shock
Myopathic (acute myocardial infarction (MI), dilated cardiomyopathy, acute decompensated heart failure (ADHF), myocarditis)
Mechanical (acute mitral regurgitation, ventricular septal defect, severe aortic stenosis, severe ventricular septal defect)
Arrhythmic (atrial, ventricular, bradyarrhythmia)

Distributive shock

Sepsis

Anaphylaxis

Neurogenic (e.g., spinal cord injury)

Toxic overdoses

Endocrinologic (Addison's disease, myxedema)

Obstructive shock

Pericardial tamponade

Massive pulmonary embolism (PE)

Tension pneumothorax

Microcirculation. Normally when cardiac output falls, systemic vascular resistance rises to maintain a level of systemic pressure that is adequate for perfusion of the heart and brain at the expense of other tissues such as muscle, skin, and especially the gastrointestinal tract. Systemic vascular resistance is determined primarily by the luminal diameter of arterioles. The metabolic rates of the heart and brain are high, and their stores of energy substrate are low. These organs are critically dependent on a continuous supply of oxygen and nutrients, and neither tolerates severe ischemia for more than brief periods. Autoregulation, i.e., the maintenance of blood flow over a wide range of perfusion pressures, is critical in sustaining cerebral and coronary perfusion despite significant hypotension. However, when mean arterial pressure drops to ≤65 mmHg, flow to these organs falls, and their function deteriorates.

Arteriolar vascular smooth muscle has both α- and β-adrenergic receptors. The α_1 receptors mediate vasoconstriction, while the β_2 receptors mediate vasodilation. Efferent sympathetic fibers release norepinephrine, which acts primarily on α_1 receptors in one of the most fundamental compensatory responses to reduced perfusion pressure. Other constrictor substances that are increased in most forms of shock include angiotensin II, vasopressin, endothelin 1, and thromboxane A_2. Both norepinephrine and epinephrine are released by the adrenal medulla, and the concentrations of these catecholamines in the bloodstream rise. Circulating vasodilators in shock include prostacyclin [prostaglandin (PG) I_2], nitric oxide (NO), and, importantly, products of local metabolism such as adenosine that match flow to the tissue's metabolic needs. The balance between these various vasoconstrictor and vasodilator influences acting upon the microcirculation determines local perfusion.

Transport to cells depends on microcirculatory flow; capillary permeability; the diffusion of oxygen, carbon dioxide, nutrients, and products of metabolism through the interstitium; and the exchange of these products across cell membranes. Impairment of the microcirculation, which is central to the pathophysiologic responses in the late stages of all forms of shock, results in the derangement of cellular metabolism, which is ultimately responsible for organ failure.

The endogenous response to mild or moderate hypovolemia is an attempt at restitution of intravascular volume through alterations in hydrostatic pressure and osmolarity. Constriction of arterioles leads to reductions in both the capillary hydrostatic pressure and the number of capillary beds perfused, thereby limiting the capillary surface area across which filtration occurs. When filtration is reduced while intravascular oncotic pressure remains constant or rises, there is net reabsorption of fluid into the vascular bed, in accord with Starling's law of capillary-interstitial liquid exchange. Metabolic changes (including hyperglycemia and elevations in the products of glycolysis, lipolysis, and proteolysis) raise extracellular osmolarity, leading to an osmotic gradient between cells and interstitium that increases interstitial and intravascular volume at the expense of intracellular volume.

Cellular Responses. Interstitial transport of nutrients is impaired in shock, leading to a decline of intracellular high-energy phosphate stores. Mitochondrial dysfunction and uncoupling of oxidative phosphorylation are the most likely causes for decreased amounts of ATP. As a consequence, there is an accumulation of hydrogen ions, lactate, and other products of anaerobic metabolism. As shock progresses, these vasodilator metabolites override vasomotor tone, causing further hypotension and hypoperfusion. Dysfunction of cell membranes is thought to represent a common end-stage pathophysiologic pathway in the various forms of shock. Normal cellular transmembrane potential falls, and there is an associated increase in intracellular sodium and water, leading to cell swelling, which interferes further with microvascular perfusion. In a preterminal event, homeostasis of calcium via membrane channels is lost with flooding of calcium intracellularly and a concomitant extracellular hypocalcemia. There is also increasing evidence for a widespread but selective apoptotic loss of cells, contributing to organ and immune failure.

Neuroendocrine Response. Hypovolemia, hypotension, and hypoxia are sensed by baroreceptors and chemoreceptors, which contribute to an autonomic response that attempts to restore blood volume, maintain central perfusion, and mobilize metabolic substrates. Hypotension disinhibits the vasomotor center, resulting in increased adrenergic output and reduced vagal activity. Release of norepinephrine from adrenergic neurons induces peripheral and splanchnic vasoconstriction, a major contributor to the maintenance of central organ perfusion, while reduced vagal activity increases the heart rate and cardiac

output. Vagal tone is also recognized to downregulate the innate immunity inflammatory response. The effects of circulating epinephrine released by the adrenal medulla in shock are largely metabolic, causing increased glycogenolysis and gluconeogenesis and reduced pancreatic insulin release. Epinephrine also inhibits production and release of inflammatory mediators through stimulation of β-adrenergic receptors on innate immune cells.

Severe pain and other severe stress cause the hypothalamic release of adrenocorticotropic hormone (ACTH). This stimulates cortisol secretion, which contributes to decreased peripheral uptake of glucose and amino acids, enhances lipolysis, and increases gluconeogenesis. Increased pancreatic secretion of glucagon during stress accelerates hepatic gluconeogenesis and further elevates blood glucose concentration. These hormonal actions act synergistically to increase blood glucose in the maintenance of blood volume. Many critically ill patients have recently been shown to exhibit low plasma cortisol levels and an impaired response to ACTH stimulation. Low levels of cortisol in response to stimulation are linked to a decrease in survival. The importance of the cortisol response to stress is illustrated by the profound circulatory collapse that occurs in patients with adrenal cortical insufficiency .

Renin release is increased in response to adrenergic discharge and reduced perfusion of the juxtaglomerular apparatus in the kidney. Renin induces the formation of angiotensin I, which is then converted to angiotensin II, an extremely potent vasoconstrictor and stimulator of aldosterone release by the adrenal cortex and of vasopressin by the posterior pituitary. Aldosterone contributes to the maintenance of intravascular volume by enhancing renal tubular reabsorption of sodium, resulting in the excretion of a low-volume, concentrated, sodium-free urine. Vasopressin has a direct action on vascular smooth muscle, contributing to vasoconstriction, and acts on the distal renal tubules to enhance water reabsorption.

Cardiovascular Response. Three variables – ventricular filling (preload), the resistance to ventricular ejection (afterload), and myocardial contractility – are paramount in controlling stroke volume. Cardiac output, the major determinant of tissue perfusion, is the product of stroke volume and heart rate. Hypovolemia leads to decreased ventricular preload, which in turn reduces the stroke volume. An increase in heart rate is a useful but limited compensatory mechanism to maintain cardiac output. A shock-induced reduction in myocardial compliance is frequent, reducing ventricular end-diastolic volume and hence stroke volume at any given ventricular filling pressure. Restoration of intravascular volume may return stroke volume to normal but only at elevated filling pressures. Increased filling pressures also stimulate release of brain natriuretic peptide (BNP) to secrete sodium and volume to relieve the pressure on the heart. Levels of BNP correlate with outcome following severe stress. In addition, sepsis, ischemia, myocardial infarction, severe tissue trauma,

hypothermia, general anesthesia, prolonged hypotension, and acidemia may all impair myocardial contractility and also reduce the stroke volume at any given ventricular end-diastolic volume. The resistance to ventricular ejection is significantly influenced by the systemic vascular resistance, which is elevated in most forms of shock. However, resistance is depressed in the early hyperdynamic stage of septic shock, thereby initially allowing the cardiac output to be maintained or elevated.

The venous system contains nearly two-thirds of the total circulating blood volume, most in the small veins, and serves as a dynamic reservoir for autoinfusion of blood. Active venoconstriction as a consequence of α-adrenergic activity is an important compensatory mechanism for the maintenance of venous return and therefore of ventricular filling during shock. On the other hand, venous dilatation, as occurs in neurogenic shock, reduces ventricular filling and hence stroke volume and cardiac output.

Pulmonary Response. The response of the pulmonary vascular bed to shock parallels that of the systemic vascular bed, and the relative increase in pulmonary vascular resistance, particularly in septic shock, may exceed that of the systemic vascular resistance. Shock-induced tachypnea reduces tidal volume and increases both dead space and minute ventilation. Relative hypoxia and the subsequent tachypnea induce a respiratory alkalosis. Recumbency and involuntary restriction of ventilation secondary to pain reduce functional residual capacity and may lead to atelectasis. Shock is recognized as a major cause of acute lung injury and subsequent acute respiratory distress syndrome. These disorders are characterized by noncardiogenic pulmonary edema secondary to diffuse pulmonary capillary endothelial and alveolar epithelial injury, hypoxemia, and bilateral diffuse pulmonary infiltrates. Hypoxemia results from perfusion of underventilated and nonventilated alveoli. Loss of surfactant and lung volume in combination with increased interstitial and alveolar edema reduces lung compliance. The work of breathing and the oxygen requirements of respiratory muscles increase.

Renal Response. Acute renal failure, a serious complication of shock and hypoperfusion, occurs less frequently than heretofore because of early aggressive volume repletion. Acute tubular necrosis is now more frequently seen as a result of the interactions of shock, sepsis, the administration of nephrotoxic agents (such as aminoglycosides and angiographic contrast media), and rhabdomyolysis; the latter may be particularly severe in skeletal muscle trauma. The physiologic response of the kidney to hypoperfusion is to conserve salt and water. In addition to decreased renal blood flow, increased afferent arteriolar resistance accounts for diminished glomerular filtration rate, which together with increased aldosterone and vasopressin is responsible for reduced urine formation. Toxic injury causes necrosis of tubular epithelium and tubular obstruction by cellular debris with back-leak of filtrate. The depletion of renal

ATP stores that occurs with prolonged renal hypoperfusion contributes to subsequent impairment of renal function.

Metabolic Derangements. During shock, there is disruption of the normal cycles of carbohydrate, lipid, and protein metabolism. Through the citric acid cycle, alanine in conjunction with lactate (which is converted from pyruvate in the periphery in the presence of oxygen deprivation) enhances the hepatic production of glucose. With reduced availability of oxygen, the breakdown of glucose to pyruvate and ultimately lactate represents an inefficient cycling of substrate with minimal net energy production. An elevated plasma lactate/pyruvate ratio is consistent with anaerobic metabolism and reflects inadequate tissue perfusion. Decreased clearance of exogenous triglycerides coupled with increased hepatic lipogenesis causes a significant rise in serum triglyceride concentrations. There is increased protein catabolism, a negative nitrogen balance, and, if the process is prolonged, severe muscle wasting.

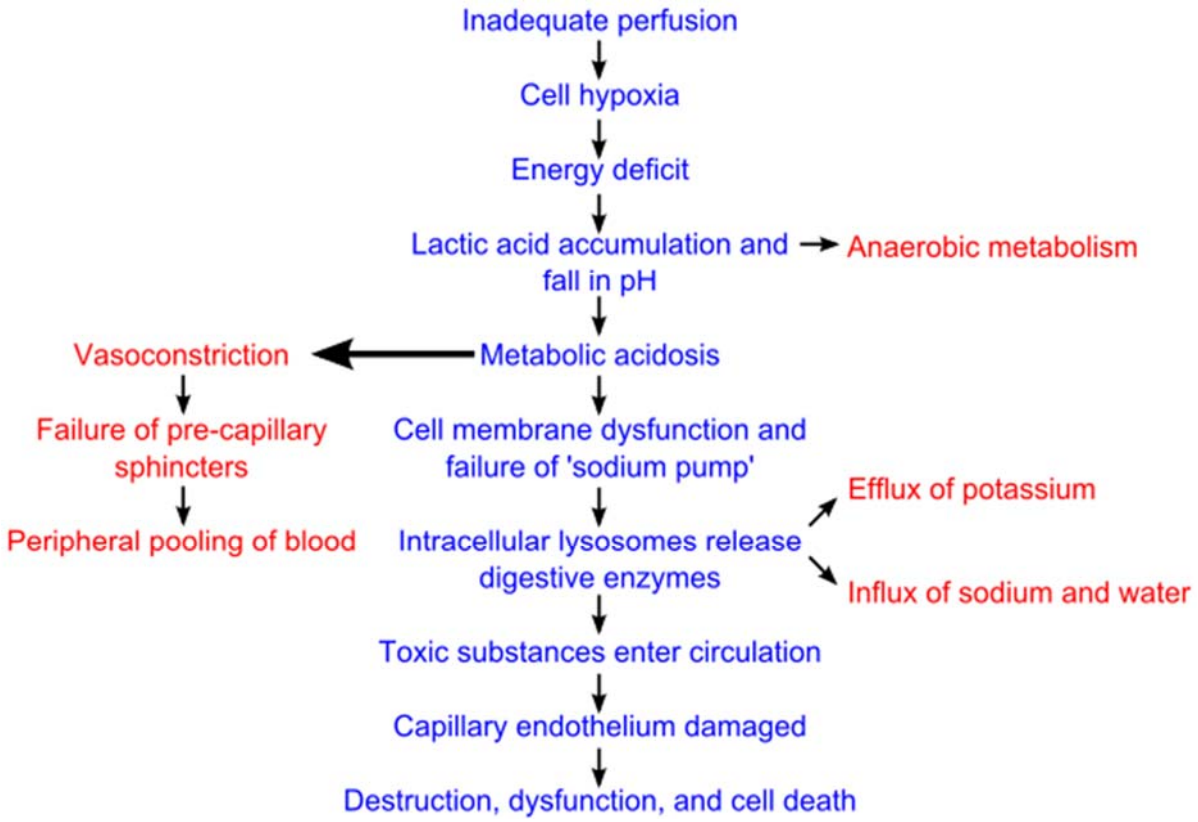


Fig. 4. Pathogenesis

Inflammatory Responses. Activation of an extensive network of proinflammatory mediator systems plays a significant role in the progression of shock and contributes importantly to the development of organ injury and failure (Fig. 5). In those surviving the acute insult, there is a delayed endogenous counterregulatory response to "turn off" the excessive proinflammatory response.

If balance is restored, the patient does well. If the immunosuppressive response is excessive, the patient is highly susceptible to secondary nosocomial infections, which can then drive the systemic inflammatory response syndrome (SIRS) and lead to delayed multiple organ failure.

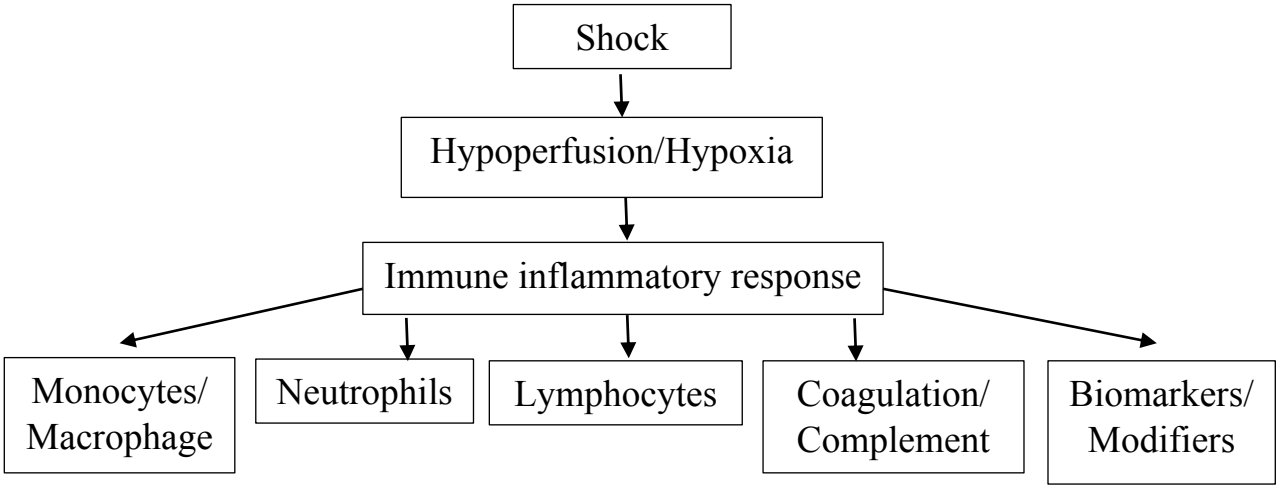


Fig. 5. A schematic of the host immune inflammatory response to shock

Table 1

Stages of shock

1. ***Compensated shock or nonprogressive***
- compensatory mechanisms of the circulation can return CO and BP to normal levels - baroreceptor reflexes → sympathetic stimulation → constrict arteriols in most parts of the body and venous reservoirs → protection of coronary and cerebral blood flow - angiotensin-aldosteron, ADH → vasoconstriction, - water and salt retention by the kidneys - absorption of fluid from interstitial fluid and GIT, increased thirst
2. ***Decompensated reversible shock or progressive***
- circulatory system themselves begin to deteriorate, without therapy shock becomes steadily worse until death. - positive feedback mechanisms are developed and can cause vicious circle of progressively decreasing CO - Cardiac depression - ↓ coronary blood flow, ↓ contractility - Vasomotor failure - ↓ cerebral blood flow - Release of toxins by ischemic tissues: histamine, serotonin, tissue enzymes - Intestines hypoperfusion → mucosal barrier disturbance → endotoxin formation and absorption → vasodilatation, cardiac depression - Vasodilation in precapillary bed - Generalised cellular deterioration → first signs of multiorgan failure

3. ***Decompensated irreversible shock***

- despite therapy circulatory system continues to deteriorate and death enssues
- marked hypoxic tissue damage
- endothelial dysfunction → adhesive molecules, neutrophils, macrophages → inflammation
- progressive acidosis microcirculation failure → plasma proteins leak to interstitium
- advanced disseminated intravascular coagulation

Decompensation occurs when normal compensatory mechanisms are overwhelmed and can no longer allow the body to maintain adequate blood pressure and tissue perfusion (Table 1). This leads to a complex cascade of life-threatening failures.

The body's responses to shock may vary according to the cause. For example, distributive shock may be characterized by low cardiac output. This variability may progress in all forms of shock to a set of common organ effects (Table 2).

Table 2

Common Effects of Shock on Organs

Systemic
• Capillary leak • Formation of microvascular shunts • Cytokine release
Cardiovascular
• Circulatory failure • Depression of cardiovascular function • Arrhythmia
Hematologic
• Bone marrow suppression • Coagulopathy • Disseminated intravascular coagulation (DIC) • Platelet dysfunction
Hepatic
• Liver insufficiency • Elevation of liver enzyme levels • Coagulopathy

Neuroendocrine
• Change of mental status • Adrenal suppression • Insulin resistance • Thyroid dysfunction
Renal
• Renal insufficiency • Change in urine electrolyte levels • Elevation of blood urea nitrogen (BUN) and creatinine levels
Cellular
• Cell-to-cell dehiscence • Cellular swelling • Mitochondrial dysfunction • Cellular leak

7. PRINCIPLES OF DIFFERENTIAL DIAGNOSIS THE DIFFERENT TYPES OF SHOCK

Differentiation between the four categories of shock can be made using several haemodynamic monitoring tools, including a pulmonary artery catheter, cardiac echocardiography and pulse contour analysis. Whatever the technique used, classification of shock relies on the determination of cardiac output and evaluation of intravascular pressures or volumes.

Measurement of cardiac output is essential to discriminate between the different types of shock: cardiac output and oxygen delivery are decreased (hypodynamic shock) in hypovolaemic, cardiogenic and obstructive shock, while it may be preserved and even increased in distributive shock (hyperdynamic shock).

To identify further the different components of hypodynamic shock, the determination of intravascular pressures (CVP) is essential: it is decreased in hypovolaemia, but increased in cardiogenic and obstructive shock. Of note, hypovolaemia can complicate any type of shock.

Echocardiography is mandatory to differentiate obstructive from cardiogenic shock (even though some indexes can be inferred from the pulmonary artery catheter or measurements of intravascular volumes with transpulmonary thermodilution) and to determine the exact cause of cardiogenic shock (contractility, valve disease).

Table 3

Physiologic characteristics of the various forms of Shock

Type of shock	*Preload*	*Pump function (CO)*	*Systemic vascular resistance (SVR)*	*Tissue perfusion (SvO_2)*
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Distributive	↓	↑(early), ↓(late)	↓	↑
Obstructive (pulmonary embolism, tension pneumothorax)	↓	↓	↑	↓
Obstructive (pericardial tamponade)	↑	↓	↑	↓

Although many clinical features are common to all types of shock, there are certain aspects in which they differ (Table 3).

To recognize and manage all forms of shock, clinicians must maintain a high level of suspicion and carefully observe the patient's response to initial treatment. Initial determination of the cause of shock requires an appropriate patient history and expeditious, careful physical examination.

Table 4

General Symptoms of Shock

CNS Changes

- Confusion
- Coma
- Combative behavior
- Agitation
- Stupor

Skin Changes

- Cool
- Clammy
- Warm
- Diaphoresis

Cardiovascular

- Increase or decrease in heart rate
- Arrhythmia
- Angina
- Low, high, or normal cardiac output
- Changes in preload (see Table 2)

Pulmonary

- Increased respiratory rate
- Increase or decrease in end-tidal CO_2
- Decrease in O_2 saturation
- Increased pulmonary pressures
- Respiratory failure
- Decreased tidal volume

Renal

- Decreased urine output
- Elevation in BUN and creatinine levels
- Change in urine electrolyte levels

Evaluation of symptoms. The type of shock must be evaluated by reviewing the history of the disease process.

In cardiogenic shock, the patient may have a history of cardiac disease, poor cardiac function, congestive heart failure, myocardial ischemia, or valvular heart disease. In hypovolemic shock, there is usually a history of blood loss, trauma, fluid losses, dehydration, third spacing, or other fluid losses. Distributive shock is usually associated with exposure to an infectious or allergic agent, neurologic events, or a reaction to various immunologic substances. In obstructive shock, there may be a history of trauma or a process that leads to a mechanical obstruction of cardiac filling, such as cardiac tamponade.

Early recognition of hypotension and hypoperfusion is essential in prevention
and treatment of all types of shock. Hypoperfusion may be the trigger for much of the end-organ dysfunction and cytokine activation. In adults, a drop in systolic blood pressure of more than 40 mm Hg constitutes significant hypotension. Hypoperfusion may be present in the absence of significant hypotension if microcirculatory factors are activated. Shock is usually recognized as hypotension characterized by hypoperfusion abnormalities.

General symptoms are illustrated in Table 4 and are a guide for rapid evaluation and treatment. *Hypovolemic Shock.* Hypovolemic shock occurs when there is a depletion of fluid in the intravascular space as a result of hemorrhage, vomiting, diarrhea, dehydration, capillary leak, or a combination of these. Capillary leak is common with the activation of the systemic inflammatory response. The typical clinical presentation is a patient with hypotension, skin vasoconstriction and collapsed jugular veins. The key haemodynamic findings are a decreased cardiac output, high SVR and low filling pressures. Haemoglobin can initially be maintained in haemorrhage. The echocardiographic and echodoppler profile is one of decreased right-sided filling, decreased stroke volume, and decreased aortic diameter.

Cardiogenic shock. Cardiogenic shock is due to a failure of the cardiac pump, related either to impaired contractile function or to valvular dysfunction. In addition, it can be global, affecting both right and left sides, or predominantly left or right sided. It is
important to make a precise diagnosis of the cause of cardiogenic shock, as therapy may differ accordingly.

The typical clinical presentation is a patient with hypotension, skin vasoconstriction and dilated jugular veins.

Diagnosis: the haemodynamic defi nition of cardiogenic shock is based on the finding of a decreased and inadequate cardiac index (CI lower than 2.2l/min/m^2 and
low SvO_2) in the presence of adequate preload and accompanied by signs of hypoperfusion (decreased blood pressure or elevated lactate levels). In this defi nition, it is important to evaluate cardiac output, and especially to determine that it is inadequate for metabolic needs (signs of inadequate tissue perfusion): indeed patients with chronic heart failure may present signs of decreased systolic function and dilated ventricules, but cardiac output can be preserved.

Evaluation of ventricular preload (pressure or echo measurements) is essential to rule out hypovolaemic shock, but also to differentiate right from left side dysfunction. Intravascular pressures can either be invasively measured (central venous and pulmonary artery catheters) or estimated by echocardiography. Intravascular volumes can be estimated by echocardiography and transpulmonary thermodilution. Of note, the latter cannot differentiate left and right ventricular volumes.

Distributive Shock. Distributive shock is a complex syndrome characterized by profound cardiovascular derangements, associating a decreased vascular tone, myocardial depression, blood fl ow redistribution between organs and microcirculatory alterations. In addition, hypovolaemia is frequent in its early stages (fluid losses and venous blood pooling in the splanchnic area). The typical clinical presentation is a patient with hypotension, skin vasodilation, and acrocyanosis or mottled skin.

The most common cause of distributive shock is septic shock. Other forms of distributive shock are anaphylactic shock, acute adrenal insufficiency, and neurogenic shock. The primary problems are the development of shunts and capillary leak. In distributive shock, there is activation of SIRS and a breakdown of cellular function in the septic process. The hemodynamic profile is characterized by a normal or increased cardiac output with a low SVR and low-to-normal left ventricular filling pressure. The echocardiographic profile is one of low stroke volume and an increase in aortic diameter.

Obstructive Shock. Direct mechanical obstruction to cardiac filling is the keystone of obstructive shock. In obstructive shock, there is depression of the ability to fill the right side of the heart, which may be the result of a fluid

collection around the heart, cardiac tamponade, or a massive increase in intrathoracic pressure. In cardiac tamponade, the pressure in the right side of the heart, the pulmonary artery, and the
left side of the heart equilibrate in diastole. The typical clinical presentation is a patient with hypotension, skin vasoconstriction and dilated jugular veins. Pulsus paradoxus is frequent.

The typical haemodynamic presentation is a low cardiac output, high SVR, high fi lling pressures (right in PE, left in aortic dissection, bilateral in tamponade) and pulmonary hypertension in PE.

Another form of obstructive shock is tension pneumothorax, in which there is increased intrathoracic pressure with hypotension, resulting from decreased preload.

Echocardiography is extremely helpful to diagnose obstructive shock and its cause.

8. DIAGNOSIS

General laboratory tests should include measurement of blood lactate level (usually secondary to anaerobic metabolism), which is a marker for poor oxygen delivery or use6 and serum bicarbonate level (a decrease in this level is a marker for metabolic acidosis). There can also be an elevation in blood glucose level and changes in the level of several electrolytes: zinc, magnesium, and calcium; these should be measured. Alterations in renal parameters commonly include an elevation in creatinine and blood urea nitrogen (BUN) levels and changes in urine electrolyte levels. Liver parameters are also affected by shock states; alterations occur in all liver enzyme levels. ABG analysis is one of the most important laboratory tests because it measures the baseline oxygen delivery and utilization, which is the basic problem in shock. The most common findings are hypoxia, metabolic acidosis, and an elevation in $PaCO_2$.

Coagulation. The coagulation cascade may be affected by the shock syndrome through activation of SIRS with evidence of disseminated intravascular coagulation (DIC), an increase in fibrin split products, and a fall in fibrinogen and antithrombin III levels. Coagulation factors are also affected by liver failure, with increases in prothrombin time (PT) and activated partial thomboplastin time (APTT).

Hematologic Parameters. In septic or infectious shock, the WBC count can be either high or low. In other forms of shock, bone marrow suppression may lead to decreased production of all hematologic cells. Platelet counts may fall or platelets may not function normally in several forms of shock. Erythropoietin levels also decrease in shock.

Renal Parameters. Oliguria and renal insufficiency are important markers for shock because the kidney is very sensitive to hypoperfusion and cytokine effects. Oliguria may be caused by direct renal injury by cytokines, prerenal volume problems, or postrenal problems. In all critically ill patients, a urine output of less than 0.5 mL/kg
per hour is defined as oliguria.

Echocardiography. The addition of echocardiography in the ICU has added greatly to our ability to diagnose and manage various forms of shock. Formal echocardiography requires special training for both the transthoracic and esophageal forms. Over the past few years, an esophageal Doppler echocardiographic probe has been developed that is easy to use and gives data on aortic artery diameter, stroke volume, and cardiac output in real time.

9. HYPOVOLEMIC SHOCK

Hypovolaemic shock is the most common source of shock; it often can easily be reversed, if detected early and provided its cause can be corrected.

Pathophysiology of hypovolaemic shock: Hypovolaemic shock is due to a profound reduction in blood volume. Hypovolaemia is associated with an initial decrease in ventricular preload, but the almost immediate increase in endogenous catecholamines limits the decrease in stroke volume (by increasing contractility), while cardiac output is maintained by the compensatory increase in heart rate. The profound vasoconstriction also helps to redistribute blood volume from the peripheral to the central compartment, helping to maintain cardiac output and blood pressure. This venous constriction limits the decrease in preload by decreasing the amount of blood stored in large venous capacitance beds (mostly splanchnic veins, but also arm and leg veins). It also affects blood fl ow distribution, so that the limited amount of blood is redirected to the most vital organs. Blood fl ow to splanchnic organs, kidneys and the skin is markedly decreased, while brain and heart circulations are somewhat preserved. If hypovolaemia persists, these compensatory mechanisms are not suffi cient and cardiac output and blood pressure decrease.

These compensatory mechanisms are clearly vital and benefi cial in the short term, but unless the hypovolaemia is corrected they will begin to cause deleterious effects. The decrease in kidney perfusion may lead to renal damage and eventually acute tubular necrosis. Myocardial oxygen balance is threatened by the combination of tachycardia, increased contractility, and increased catecholamine levels in the context of decreased coronary perfusion (due to hypotension). This may lead to myocardial ischaemia and even to MI in patients with coronary lesions. The decrease in splanchnic perfusion may alter

the gut defences and integrity, with the possibility of promoting translocation of bacteria and bacterial toxins, especially during the reperfusion phase.

Causes of hypovolaemic shock. There are many causes, ranging from bleeding (trauma, digestive haemorrhage or other causes of blood losses), to decreased plasma volume (dehydration, most commonly due to diarrhoea or vomiting) or tissue damage and fluid loss such as burns. Children are particularly sensitive to dehydration.

Clinical features. Presentation; tachycardia, skin vasoconstriction and acrocyanosis. Hypotension often occurs, but blood pressure can initially be preserved even with large fl uid losses, especially in the young. Signs of tissue hypoperfusion are frequent (alteration in mental state, oliguria). Signs of dehydration or anaemia may be encountered, but these may sometimes be missing.

Key haemodynamic fi ndings. Hypovolaemic shock is characterized by a decreased cardiac output, high SVR and low filling pressures. In patients on mechanical ventilation, large respiratory variations in pulse pressure and stroke volume can be observed. Venous O_2 saturation (mixed venous and central venous) is typically decreased. Lactic acidosis is frequent.

Haemoglobin levels can initially be maintained in haemorrhage and decrease only after initiation of fl uid resuscitation or when shock is prolonged (volume mobilization from the extravascular compartment).

Hemorrhagic shock

Hemorrhage is the most common cause of shock in trauma patients. The trauma patient's response to blood loss is made more complex by fluid shifts among the fluid compartments in the body, particularly in the extracellular fluid compartment. Soft tissue injury, even without severe hemorrhage, can result in shifts of fluid to the extracellular compartment. The response to blood loss must be considered in the context of these fluid shifts. Also consider the changes associated with severe, prolonged shock and the pathophysiologic results of resuscitation and reperfusion.

Definition of Hemorrhage. Hemorrhage is an acute loss of circulating blood volume. Although it can vary considerably, normal adult blood volume is approximately 7 % of body weight. For example, a 70-kg male has a circulating blood volume of approximately 5 L. The blood volume of obese adults is estimated based on their ideal body weight, because calculation based on actual weight can result in significant overestimation. The blood volume for a child is calculated as 8 % to 9 % of body weight (70–80 mL/kg).

Physiologic Classification. The physiologic effects of hemorrhage are divided into four classes, based on clinical signs, which are useful for estimating the percentage of acute blood loss. The clinical signs represent a continuum of ongoing hemorrhage and serve only to guide initial therapy.

Subsequent volume replacement is determined by the patient's response to therapy. The following classification system is useful in emphasizing the early signs and pathophysiology of the shock state:

• *Class I hemorrhage* is exemplified by the condition of an individual who has donated 1 unit of blood.

• *Class II hemorrhage* is uncomplicated hemorrhage for which crystalloid fluid resuscitation is required.

• *Class III hemorrhage* is a complicated hemor- rhagic state in which at least crystalloid infusion is required and perhaps also blood replacement.

• *Class IV hemorrhage* is considered a preterminal event; unless aggressive measures are taken, the patient will die within minutes. Blood transfusion is required.

Class I Hemorrhage: <15 % Blood Volume Loss

The clinical symptoms of volume loss with class I hemorrhage are minimal. In uncomplicated situations, minimal tachycardia occurs. No measurable changes occur in blood pressure, pulse pressure, or respiratory rate. For otherwise healthy patients, this amount of blood loss does not require replacement, because transcapillary refill and other compensatory mechanisms will restore blood volume within 24 hours, usually without the need for blood transfusion.

Class II Hemorrhage: 15 % to 30 % Blood Volume Loss

Clinical signs of class II hemorrhage include tachycardia, tachypnea, and decreased pulse pressure. The latter sign is related primarily to a rise in diastolic blood pressure due to an increase in circulating catecholamines, which produce an increase in peripheral vascular tone and resistance. Systolic pressure changes minimally in early hemorrhagic shock; therefore, it is important to evaluate pulse pressure rather than systolic pressure. Other pertinent clinical findings associated with this amount of blood loss include subtle central nervous system (CNS) changes, such as anxiety, fear, and hostility. Despite the significant blood loss and cardiovascular changes, urinary output is only mildly affected. The measured urine flow is usually 20 to 30 mL/hour in an adult with class II hemorrhage. Accompanying fluid losses can exaggerate the clinical manifestations of class II hemorrhage. Some patients in this category may eventually require blood transfusion, but most are stabilized initially with crystalloid solutions.

Class III Hemorrhage: 30 % to 40 % Blood Volume Loss

Patients with class III hemorrhage typically present with the classic signs of inadequate perfusion, including marked tachycardia and tachypnea, significant changes in mental status, and a measurable fall in systolic blood pressure. In an uncomplicated case, this is the least amount of blood loss that consistently causes a drop in systolic blood pressure. The priority of initial management is to stop the hemorrhage, by emergency operation or embolization, if necessary. Most patients in this category will require packed red blood cells (pRBCs) and blood products to reverse the shock state.

Class IV Hemorrhage: >40% Blood Volume Loss

The degree of exsanguination with class IV hemorrhage is immediately life-threatening. Symptoms include marked tachycardia, a significant decrease in systolic blood pressure, and a very narrow pulse pressure or unmeasurable diastolic blood pressure. (Bradycardia may develop preterminally.) Urinary output is negligible, and mental status is markedly depressed. The skin is cold and pale. Patients with class IV hemorrhage frequently require rapid transfusion and immediate surgical intervention. These decisions are based on the patient's response to the initial management techniques described in this chapter.

Confounding factors. The physiologic classification system is helpful, but the following factors may confound and profoundly alter the classic hemodynamic response to the acute loss of circulating blood volume; all individuals involved in the initial assessment and resuscitation of injured patients must promptly recognize them:

- Patient age
- Severity of injury, particularly the type and anatomic location of injury
- Time lapse between injury and initiation of treatment
- Prehospital fluid therapy
- Medications used for chronic conditions

It is dangerous to wait until a trauma patient fits a precise physiologic classification of shock before initiating appropriate volume restoration. Initiate hemorrhage control and balanced fluid resuscitation when early signs and symptoms of blood loss are apparent or suspected – not when the blood pressure is falling or absent. Stop the bleeding.

Fluid changes secondary to soft-tissue injury. Major soft-tissue injuries and fractures compromise the hemodynamic status of injured patients in two ways: First, blood is lost into the site of injury, particularly in major fractures. For example, a fractured tibia or humerus can result in the loss of up to 750 mL of blood. Twice that amount, 1500 mL, is commonly associated with femur fractures, and several liters of blood can accumulate in a retroperitoneal hematoma associated with a pelvic fracture. Obese patients are at risk for extensive blood loss into soft tissues, even in the absence of fractures. Elderly patients are also at risk because of fragile skin and subcutaneous tissues that injures more readily and tamponades less effectively, in addition to inelastic blood vessels that do not spasm and thrombose when injured or transected. Second, edema that occurs in injured soft tissues constitutes another source of fluid loss. The degree of this additional volume loss is related to the magnitude of the soft-tissue injury. Tissue injury results in activation of a systemic inflammatory response and production and release of multiple cytokines. Many of these locally active substances have profound effects on the vascular endothelium, resulting in increased permeability. Tissue edema is the result of shifts in fluid primarily from the plasma into the extravascular, or extracellular,

space as a result of alterations in endothelial permeability. Such shifts produce an additional depletion in intravascular volume.

Initial management of hemorrhagic shock. The diagnosis and treatment of shock must occur almost simultaneously. For most trauma patients, clinicians begin treatment as if the patient has hemorrhagic shock, unless a different cause of shock is clearly evident. The basic management principle is to stop the bleeding and replace the volume loss.

Physical examination. The physical examination is focused on diagnosing immediately life-threatening injuries and assessing the **ABCDE**s. Baseline observations are important to assess the patient's response to therapy, and repeated measurements of vital signs, urinary output, and level of consciousness are essential. A more detailed exam- ination of the patient follows as the situation permits.

Airway and **B**reathing. Establishing a patent airway with adequate ventilation and oxygenation is the first priority. Provide supplementary oxygen to maintain oxygen saturation at greater than 95 %.

Circulation: Hemorrhage Control. Priorities for managing circulation include controlling obvious hemorrhage, obtaining adequate intravenous access, and assessing tissue perfusion. Bleeding from external wounds in the extremities usually can be controlled by direct pressure to the bleeding site, although massive blood loss from an extremity may require a tourniquet. A sheet or pelvic binder may be used to control bleeding from pelvic fractures. Surgical or angioembolization may be required to control internal hemorrhage. The priority is to stop the bleeding, not to calculate the volume of fluid lost.

Disability: Neurological Examination. A brief neurological examination will determine the patient's level of consciousness, which is useful in assessing cerebral perfusion. Alterations in CNS function in patients who have hypovolemic shock do not necessarily imply direct intracranial injury and may reflect inadequate perfusion. Repeat neurological evaluation after restoring perfusion and oxygenation.

Exposure: Complete Examination. After addressing lifesaving priorities, completely undress the patient and carefully examine him or her from head to toe to search for additional injuries. When exposing a patient, it is essential to prevent hypothermia, a condition that can exacerbate blood loss by contributing to coagulopathy and worsening acidosis. To prevent hypothermia, always use fluid warmers and external passive and active warming techniques.

Gastric Dilation: Decompression. Gastric dilation often occurs in trauma patients, especially in children. This condition can cause unexplained hypotension or cardiac dysrhythmia, usually bradycardia from excessive vagal stimulation. In unconscious patients, gastric distention increases the risk of aspiration of gastric contents, a potentially fatal complication. Consider decompressing the stomach by inserting a nasal or oral tube and attaching it to

suction. Be aware that proper positioning of the tube does not eliminate the risk of aspiration.

Urinary Catheterization. Bladder catheterization allows clinicians to assess the urine for hematuria, which can identify the genitourinary system as a source of blood loss. Monitoring urine output also allows for continuous evaluation of renal perfusion. Blood at the urethral meatus or perineal hematoma/bruising may indicate urethral injury and contraindicates the insertion of a transurethral catheter before radiographic confirmation of an intact urethra.

Vascular access (Intra Venous line). Obtain access to the vascular system promptly. This measure is best accomplished by inserting two largecaliber (minimum of 18-gauge in an adult) peripheral intravenous catheters. The rate of flow is proportional to the fourth power of the radius of the cannula and inversely related to its length, as described in Poiseuille's law. Hence, short, large-caliber peripheral intravenous lines are preferred for the rapid infusion of fluid, rather than longer, thinner catheters. Use fluid warmers and rapid infusion pumps in the presence of massive hemorrhage and severe hypotension. The most desirable sites for peripheral, percutaneous intravenous lines in adults are the forearms and antecubital veins. This can be challenging in the young, very old, obese patients, and intravenous drug users. If peripheral access cannot be obtained, consider placement of an intraosseous needle for temporary access. If circumstances prevent the use of peripheral veins, clinicians may initiate large-caliber, central venous (i.e., femoral, jugular, or subclavian vein) access.

The clinician's experience and skill are critical determinants in selecting the most appropriate procedure or route for establishing vascular access. Intraosseous access with specially designed equipment is possible in all age groups. This access may be used in the hospital until intravenous access is obtained and is discontinued when it is no longer necessary. As intravenous lines are started, draw blood samples for type and cross-match, appropriate laboratory analyses, toxicology studies, and pregnancy testing for all females of childbearing age. Blood gas analysis also may be performed at this time. A chest x-ray must be obtained after attempts at inserting a subclavian or internal jugular line to document the position of the line and evaluate for a pneumothorax or hemothorax. In emergency situations, central venous access is frequently not accomplished under tightly controlled or completely sterile conditions. Therefore, these lines should be changed in a more controlled environment as soon as the patient's condition permits.

Treatment

Initial Fluid Therapy. The amount of fluid and blood required for resuscitation is difficult to predict on initial evaluation of a patient. Administer an initial, warmed fluid bolus of isotonic fluid. The usual dose is 1 liter for adults and 20 mL/kg for pediatric patients weighing less than 40 kilograms.

Absolute volumes of resuscitation fluid should be based on patient response to fluid administration, keeping in mind that this initial fluid amount includes any fluid given in the prehospital setting. Assess the patient's response to fluid resuscitation and identify evidence of adequate end-organ perfusion and tissue oxygenation. Observe the patient's response during this initial fluid administration and base further therapeutic and diagnostic decisions on this response. Persistent infusion of large volumes of fluid and blood in an attempt to achieve a normal blood pressure is not a substitute for definitive control of bleeding.

Table outlines general guidelines for establishing the amount of fluid and blood likely required during resuscitation. If the amount of fluid required to restore or maintain adequate organ perfusion and tissue oxygenation greatly exceeds these estimates, carefully reassess the situation and search for unrecognized injuries and other causes of shock.

The goal of resuscitation is to restore organ perfusion and tissue oxygenation, which is accomplished with administering crystalloid solution and blood products to replace lost intravascular volume. However, if the patient's blood pressure increases rapidly before the hemorrhage has been definitively controlled, more bleeding can occur.

For this reason, administering excessive crystalloid solution can be harmful. Fluid resuscitation and avoidance of hypotension are important principles in the initial management of patients with blunt trauma, particularly those with traumatic brain injury. In penetrating trauma with hemorrhage, delaying aggressive fluid resuscitation until definitive control of hemorrhage is achieved may prevent additional bleeding; a careful, balanced approach with frequent reevaluation is required. Balancing the goal of organ perfusion and tissue oxygenation with the avoidance of rebleeding by accepting a lower-than-normal blood pressure has been termed "controlled resuscitation," "balanced resuscitation," "hypotensive resuscitation," and "permissive hypotension." Such a resuscitation strategy may be a bridge to, but is not a substitute for, definitive surgical control of bleeding.

Early resuscitation with blood and blood products must be considered in patients with evidence of class III and IV hemorrhage. Early administration of blood products at a low ratio of packed red blood cells to plasma and platelets can prevent the development of coagulopathy and thrombocytopenia.

The initial resuscitative effort is to attempt to correct the absolute and relative hypovolemia by refilling the vascular tree. There is good evidence that early goal directed aggressive volume resuscitation improves outcomes in hypovolemic shock. Because resuscitation efforts are not always black and white, treatment must be individualized for patient condition and need.

The following are broad guidelines for the treatment of hypovolemic shock:

- Targeting O_2 delivery to vital organs rather than a specific BP or heart rate
- This usually requires MAP of 60-70 mmHg
- Monitor mental status, skin color, blood gases, hemoglobin, urine output, electrolytes and Lactic Acid levels
- PA catheter is not necessarily a mainstay but can offer a more detailed view of patient condition. If PA catheter not available, try using CVP monitoring for baseline.

Fluid Replacement Choices

Conventional *"crystalloids"* include both balanced salt solutions (BSS) and hypotonic salt solutions. Balanced salt solutions include such fluids as 0.9% NaCl (Normal Saline), and Ringer's Lactate solutions. These solutions are characterized by having an electrolyte composition or calculated osmolality approximating that of plasma (isotonic). Balanced salt solutions distribute approximately ¾ or their volume to the extravascular space with ¼ of the volume remaining in the intravascular space.

Colloid solutions are solutions of proteins, starches, dextrans, and gelatins containing molecules sufficiently large enough so that they do not normally cross capillary membranes. Under normal conditions most of the administered volume remains in the intravascular space (unless tissue is damaged and then it can cross membranes). Once colloids have leaked into the interstitium, they must be removed by the lymphatic system or they will exert a reverse pressure gradient, drawing water from the vascular space. The removal of colloids from the interstitium is typically much slower than that of crystalloids.

Blood Component Therapy. •Packed Red Blood Cells – Red cell transfusions initially may be achieved with uncross-matched type O red cells. If a patient's blood type has been determined, ABO and Rh specific red cells can be used. Every effort should be made to establish the blood type of a patient prior to transfusion to preserve type O red cell availability and accurately determine the patient's blood type.

- Platelets – Platelet transfusion therapy after massive transfusion is an accepted intervention in the presence of micro-vascular bleeding prior to documentation of thrombocytopenia. The platelet transfusion dose recommended is 1 unit per 10 kg body weight for platelet counts <50,000 or when platelet dysfunction is suspected.
- Plasma – Plasma (FFP) transfusion therapy should be instituted after laboratory confirmation of coagulation factor deficiencies. The recommended dose is 10-15 mL/kg body weight for PT/PTT>1.5 normal range.
- Cryoprecipitate – Cryoprecipitate therapy should be instituted for the correction of laboratory evidence of hypo-fibrinogenemia (fibrinogen

<100 mg/dl). Dosing will depend on the degree of hypo-fibrinogenemia and the patient's weight. For an average size adult, 6-unit pool for fibrinogen levels between 50 – 100 mg/dl and 12-unit pool for fibrinogen levels <50 mg/dl.

Re-establishing Circulation

In addition to fluid resuscitative measures, the use of vasopressors may be required to assist with the restoration of circulation. Vasopressors are agents that cause constriction of blood vessels, leading to an increase in blood pressure. Some vasopressors are also positive inotropes (capable of increasing contractility of the heart) and/or positive chronotropes (capable of increasing heart rate). The hemodynamic effects of most vasopressors occurs secondary to their interactions with receptors in the heart and vascular system. The following vasopressors should be considered to improve circulatory efforts in hypovolemic shock:

Norepinephrine: Norepinephrine is one of the principal neurotransmitters chemical substances involved in the transmission of nerve impulses in the sympathetic nervous system. It is released from nerve cells, and is indicated for the treatment of acute hypotension resulting from conditions such as spinal anesthesia, myocardial infarction, septicemia, blood transfusions, and drug reactions. This agent is also used adjunctively in the treatment of cardiac arrest and profound hypotension. Norepinephrine is a potent alpha adrenoceptor agonist and is therefore a strong vasoconstrictor, increasing systolic and diastolic blood pressures. In addition, Norepinephrine stimulates beta 1 cells so it increases both heart rate and contractility.

Epinephrine: Epinephrine is another neurotransmitter in the sympathetic nervous system, but it is not released from nerve cells; rather, epinephrine is a hormone secreted by the adrenal medulla. Epinephrine is used intravenously during advanced cardiac life support and may also be used to treat other conditions, including anaphylactic shock and acute, severe asthma unresponsive to normal treatment. Because Epinephrine is a potent alpha and beta adrenoceptor agonist, it is also a powerful vasoconstrictor with both positive inotropic, and chronotropic effects. Epinephrine causes increased heart rate, increased force of contraction, an increase in cardiac output, and increased systolic blood pressure. The vasoconstrictive effects of epinephrine become more apparent as the dose is increased.

Dopamine: Dopamine, a precursor of norepinephrine and epinephrine, is also a neurotransmitter. Dopamine is found in both the central and peripheral nervous systems and is released from nerve cells. Dopamine is indicated in the treatment of shock due to myocardial infarction, trauma, septicemia, open-heart surgery, renal failure, and chronic cardiac decompensation. The effects of Dopamine are complex and dose dependent. Dopamine directly stimulates dopaminergic receptors, alpha and beta adrenoceptors, and it indirectly causes the release of endogenous norepinephrine. At low doses (l to 5mcg/kg/minute),

dopamine directly stimulates dopaminergic receptors on arteries in the kidneys, abdomen, heart, and brain and causes vasodilatation. At these doses, urine output may increase, but blood pressure and heart rate are usually not affected. As the dose is increased (5 to 10 mcg/kg/min), dopamine stimulates beta 1 adrenoceptors, resulting in positive inotropic and chronotropic effects, which increases myocardial contractility, and heart rate, which results in, enhanced cardiac output. At higher doses (greater than 10 mcg/kg/min), dopamine exerts effects primarily alpha-receptors, and extensive vasoconstriction causes blood pressure to increase.

Vasopressin: Vasopressin is a unique vasopressor for two reasons. First, its principal use is for a condition unrelated to its vasopressor properties. Vasopressin is an antidiuretic hormone indicated to inhibit diuresis in patients with diabetes insipidus. However, at higher doses, vasopressin causes vasoconstriction. Because there is a fair amount of evidence to support its effectiveness as a vasopressor, vasopressin is now considered as an alternative to epinephrine for the treatment of adult shock-refractory ventricular fibrillation during advanced cardiac life support. Vasopressin is a distinctive vasopressor also because its vasoconstrictive effects do not result from its interaction with adrenoceptors; rather, vasoconstriction arises from vasopressin's actions on vasopressin receptors. Vasopressin receptors are classified as V-1 and V-2 receptors. V-1 receptors are located on arterial smooth muscle, and V-2 receptors are found in renal tubules. It is Vasopressin's interaction with V-1 receptors that is responsible for its potent vasopressor effects.

Prevent Hypothermia

Hypothermia must be prevented and reversed if a pa- tient is hypothermic on arrival to the hospital. The use of blood warmers in the ED is critical, even if cumbersome. The most efficient way to prevent hypothermia in any patient receiving massive resuscitation of crystalloid and blood is to heat the fluid to 39°C (102.2°F) before infusing it. This can be accomplished by storing crystalloids in a warmer or infusing them through intravenous fluid warmers. Blood products cannot be stored in a warmer, but they can be heated by passage through intravenous fluid warmers.

Massive transfusion

A small subset of patients with shock will require massive transfusion, most often defined as > 10 units of pRBCs within the first 24 hours of admission or more than 4 units in 1 hour. Early administration of pRBCs, plasma, and platelets in a balanced ratio to minimize excessive crystalloid administration may improve patient survival. This approach has been termed “balanced,” “hemostatic,” or “damagecontrol” resuscitation. Simultaneous efforts to rapidly control bleeding and reduce the detrimental effects of

coagulopathy, hypothermia, and acidosis in these patients are extremely important. A MTP that includes the immediate availability of all blood components should be in place to provide optimal resuscitation for these patients, because extensive resources are required to provide these large quantities of blood. Appropriate administration of blood products has been shown to improve outcome in this patient population. Identification of the small subset of patients that benefit from this can be a challenge and several scores have been developed to assist the clinician in making the decision to initiate the MTP. None have been shown to be completely accurate.

Coagulopathy

Severe injury and hemorrhage result in the consumption of coagulation factors and early coagulopathy. Such coagulopathy is present in up to 30 % of severely injured patients on admission, in the absence of preexisting anticoagulant use. Massive fluid resuscitation with the resultant dilution of platelets and clotting factors, as well as the adverse effect of hypothermia on platelet aggregation and the clotting cascade, contributes to coagulopathy in injured patients. Prothrombin time, partial thromboplastin time, and platelet count are valuable baseline studies to obtain in the first hour, especially in patients with a history of coagulation disorders or who take medications that alter coagulation. These studies may also be useful in caring for patients whose bleeding history is unavailable. Point-of-care testing is available in many EDs. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) can be helpful in determining the clotting deficiency and appropriate blood components to correct the deficiency. Some jurisdictions administer *tranexamic acid* in the prehospital setting to severely injured patients in response to recent studies that demonstrated improved survival when this drug is administered within 3 hours of injury.

The first dose is usually given over 10 minutes and is administered in the field; the follow-up dose of 1 gram is given over 8 hours. In patients who do not require massive transfusion, the use of platelets, cryoprecipitate, and fresh-frozen plasma should be guided by coagulation studies, along with fibrinogen levels and balanced resuscitation principles.

Of note, many newer anticoagulant and antiplatelet agents cannot be detected by conventional testing of PT, PTT, INR, and platelet count. Some of the oral anticoagulants have no reversal agents. Patients with major brain injury are particularly prone to coagulation abnormalities. Coagulation parameters need to be closely monitored in these patients; early administration of plasma or clotting factors and/ or platelets improves survival if they are on known anticoagulants or antiplatelet agents.

Calcium administration. Most patients receiving blood transfusions do not need calcium supplements. When necessary, calcium administration should be

guided by measurement of ionized calcium. Excessive, supplemental calcium can be harmful.

Monitoring

The goal of resuscitation is to restore organ perfusion and tissue oxygenation. This state is identified by appropriate urinary output, CNS function, skin color, and return of pulse and blood pressure toward normal. Monitoring the response to resuscitation is best accomplished for some patients in an environment where sophisticated techniques are used. For elderly patients and patients with non-hemorrhagic causes of shock, consider early transfer to an intensive care unit or trauma center.

Recognition of other problems. When a patient fails to respond to therapy, causes may include one or more of the following: undiagnosed bleeding, cardiac tamponade, tension pneumothorax, ventilatory problems, unrecognized fluid loss, acute gastric distention, myocardial infarction, diabetic acidosis, hypoadrenalism, or neurogenic shock. Constant reevaluation, especially when a patient's condition deviates from expected patterns, is the key to recognizing and treating such problems as early as possible.

10. CARDIOGENIC SHOCK

Cardiogenic shock is defined as a life-threatening condition characterized by insufficient delivery of oxygenated blood to cells and tissues secondary to decreased contractility, thus resulting in decreased cardiac output.

Etiology. Cardiogenic shock occurs primarily as a result of severe left and/or right ventricular dysfunction that results in inadequate cardiac pumping. The most common cause is myocardial infarction; other causes include cardiomyopathy, ventricular rupture, and congenital heart defects.

Pathogenesis. Decreased contractility results in decreased cardiac output, which causes decreased tissue perfusion that is inadequate to meet basal metabolic requirements.

The sympathetic nervous system and renin-angiotensin system are stimulated as compensatory mechanisms to increase cardiac output. The net result of the activation of compensatory mechanisms is to increase myocardial oxygen demand. This may precipitate further myocardial damage, resulting in a progressive decline in cardiac output.

Clinical manifestations.

- Pain syndrom
- Early signs include increased heart rate with maintenance of blood pressure even though cardiac output has decreased. As compensatory

mechanisms fail, the systolic blood pressure falls and the diastolic pressure increases, causing a narrowed pulse pressure.

- Other signs include increased tachycardia; cool, clammy skin; rapid, deep respirations; pulmonary edema (crackles); an S_3 gallop; decreased urine output; and impaired level of consciousness.

Symptoms and signs. Often associated with anterior MI. Beside typical retrosternal pain, patients have symptoms and signs of severe tissue/ organ hypoperfusion (cyanosis, cold and clammy skin, slow capillary refi lling, mental changes, obtundation, oliguria) and pulmonary congestion (dyspnoea, tachypnoea, orthopnoea). Haemodynamic characteristics include tachycardia, hypotension, low cardiac output, elevated cardiac filling pressures, increased SVR and low mixed venous oxygen saturation (SvO_2).

It is the result of decreased stroke volume, which cannot be compensated by arterial vasoconstriction. The combination of low CO and systemic vasoconstriction results in severe tissue hypoperfusion and decreased coronary artery blood flow, which further worsen the cardiac performance. In some patients with cardiogenic shock, systemic arterial vasodilatation is present, most probably because of the systemic inflammatory response with cytokine release and elevated NO level, which provokes further systemic and coronary hypoperfusion and directly decreases myocardial contractility.

The differential diagnosis of cardiogenic shock includes AMI or ischaemia associated with severe aortic stenosis. Haemorrhage during thrombolytic treatment can result in haemorrhagic shock, and septic shock can develop in patients with multiple intravascular lines. Recognition is important as these diagnoses need a different management approach.

Diagnosis. The diagnosis and aetiology should be confirmed as soon as possible. Routine investigations include ECG, CXR and laboratory tests, which should include blood gas analysis, lactate and troponin measurement. Early TTE is mandatory for aetiological diagnosis and initial haemodynamic assessment in every patients with suspected cardiogenic shock. It enables rapid and reliable assessment of morphology, global and regional systolic function of the left and right ventricle, LV diastolic function, valvular morphology and function, and recognition of cardiac tamponade, PE and dissection of the proximal aorta. It is especially helpful for early diagnosis of mechanical complications of AMI. Low LVEF and severity of MR on echo examination are important predictors for poor outcome. Invasive haemodynamic measurements using an intra-arterial catheter and Swan Ganz pulmonary catheter allow haemodynamic diagnosis and continuous monitoring of haemodynamic variables, evaluation of the therapeutic effects and precise titration of the drugs. Invasive arterial pressure monitoring is mandatory in patients treated with vasopressors or vasodilators and patients with severe hypotension. The routine and early use of echocardiography significantly decreases the use of a

pulmonary artery catheter; it is still useful and necessary in patients with unstable haemodynamics, those who do not respond to the treatment and those with progressive pulmonary congestion or hypotension.

The acute coronary angiography can be performed in cardiogenic shock due to AMI, allowing confirmation of the diagnosis and immediate interventional treatment.

Treatment. Echocardiography and invasive monitoring are the pillars of diagnosis. The primary goal of treatment is removing the cardiac causes of the shock. This includes the earliest possible coronary reperfusion in avute coronary syndrome by means of percutaneous coronary intervention (PCI) with the insertion of stents (bare metal stent, BMS; drug-eluting stent, DES), surgical or other interventional treatment of mechanical causes and structural heart disease, and surgical or interventional ablation, and pacemaker therapy. In addition to this, symptomatic treatment is undertaken with the aim of improving end organ perfusion, microcirculation, and cellular oxygen utilization. This includes not just catecholamines such as dobutamine, norepinephrine, and epinephrine, vasodilators, calcium sensitizers, phosphodiesterase enzyme inhibitors PDE3, antiarrhythmic drugs, and more, but also mechanical circulatory support such as intra-aortic balloon counterpulsation, surgical and percutaneous interventional implantable ventricular support systems, and extracorporeal membrane oxygenation (ECMO).

11. DISTRIBUTIVE SHOCK

Distributive shock is a state of relative hypovolemia resulting from pathological redistribution of the absolute intravascular volume and is the most frequent form of shock. The cause is either a loss of regulation of vascular tone, with volume being shifted within the vascular system, and/or disordered permeability of the vascular system with shifting of intravascular volume into the interstitium. The three subtypes are septic, anaphylactic/anaphylactoid, and neurogenic shock.

Anaphylactic shock

Anaphylaxis is an acute systemic reaction involving symptoms of an immediate-type allergic reaction that can comprise the whole organism and potentially be fatal.

The incidence of anaphylactic reactions is diffi cult to quantify (no consensus on defi nition); however, a recent symposium suggested that any ‘allergic reaction that is rapid in onset and may cause death’ is, by defi nition, anaphylactic.

Pathophysiology.
Allergy to a substance occurs via a number of steps.
1. On fi rst exposure to an allergen, IgE antibodies specific to that allergen are produced by B cells.
2. These antibodies bind to mast cells and basophils and cause them to be 'sensitized'.
3. Further exposure to the allergen causes cross-linking of bound IgE, resulting in degranulation.

Mast cell degranulation leads to the release of a number of vasoactive mediators including histamine, prostaglandins, tryptase, platelet-activating factor and leukotrienes. The combined effect of these mediators is to produce vasodilatation, increased vascular permeability, oedema and, in cases of anaphylaxis, smooth muscle spasm in the respiratory and gastro-intestinal tracts. Chemotactic mediators are also released, attracting eosinophils to the site of infl ammation.

Anaphylactoid-type reactions cause mast cell degranulation with release of mediators, as above, but do not involve IgE cross-linkage and so do not require prior exposure to the causative agent. Clinical features and management are identical to those of anaphylactic reactions.

Anaphylactoid shock is caused by physical, chemical, or osmotic hypersensitivity reactions that are IgE-independent. Mediators are released from mast cells and basophilic granulocytes independently of any antigen–antibody reaction or presensitization. Typical triggers are X-ray contrast media.
Symptoms and signs. The clinical presentation varies greatly from one individual to another according to the dose and site of entry of the antigen and the degree of sensitization. Initially, skin manifestations, abdominal symptoms, or respiratory symptoms may be prominent. Anaphylactic reactions may resolve spontaneously or may progress despite appropriate therapy. In anaphylaxis with fatal outcome, thromboembolic events are seen as often as arrhythmias and ventricular dysfunction.
Anaphylaxis is difficult to recognize in the unstable critically ill patient. The presenting features may be attributed to the critical illness. The intubated patient provides a diagnostic challenge as early features of anaphylaxis are likely to go unnoticed. Thus a high level of clinical suspicion must be maintained. It must be remembered that anaphylactic reactions, although typically rapid in onset, may occasionally develop over a number of hours.
Cutaneous: flushing, angioedema, urticaria.
Respiratory: cough, hoarseness, stridor, bronchospasm, if ventilated a sudden rise in airway pressures may be noted.
*Cardiovascular: p*alpitations, tachycardia, arrhythmias, hypotension, collapse.
Differential diagnosis. Other conditions which should be considered when faced with the above symptoms and signs include: acute asthma

attack/bronchospasm, tension pneumothorax, myocardial infarction, airway obstruction, acute hypovolaemia.

Treatment. Patients with severe anaphylactic reactions require constant monitoring, as late reactions including arrhythmias, myocardial ischemia, and respiratory failure may manifest as late as 12 hours after the initial event. In terms of drug treatment, for anaphylactic shock especially the administration of epinephrine (plus norepinephrine, if necessary) and forced fluid replacement are required. In patients with bronchospasm, β-sympathomimetics and, as secondline treatment, glucocorticoids are indicated (as they are in patients with delayed progressive symptoms). Histamine antagonists suppress the histaminergic effects. Treatment for anaphylactoid shock is the same as for anaphylactic shock.

12. OBSTRUCTIVE SHOCK

Obstructive shock is a condition caused by the obstruction of the great vessels or the heart itself. Although the symptoms resemble those of cardiogenic shock, obstructive shock needs to be clearly distinguished from the latter because it is treated quite differently.

Pulmonary embolism is the occlusion of pulmonary arteries by thrombi that originate elsewhere, typically in the large veins of the legs or pelvis. Risk factors for pulmonary embolism are conditions that impair venous return, conditions that cause endothelial injury or dysfunction, and underlying hypercoagulable states. Symptoms of pulmonary embolism are nonspecific and include dyspnea, pleuritic chest pain, and, in more severe cases, light-headedness, presyncope, syncope, or cardiorespiratory arrest. Signs are also nonspecific and may include tachypnea, tachycardia, and in more severe cases, hypotension. Diagnosis of pulmonary embolism is most commonly accomplished with CT angiography, although ventilation/perfusion scanning is sometimes required. Pulmonary embolism treatment is with anticoagulants and, sometimes, clot dissolution with systemic or catheter-directed thrombolysis or catheter or surgical removal. When anticoagulation is contraindicated, an inferior vena caval filter should be placed. Preventive measures include anticoagulants and/or mechanical compression devices that are applied to the legs in hospitalized patients.

Cardiac Tamponade. Cardiac tamponade is accumulation of blood in the pericardial sac of sufficient volume and pressure to impair cardiac filling. Patients typically have hypotension, muffled heart tones, and distended neck veins. Diagnosis is made clinically and often with bedside echocardiography. Treatment is immediate pericardiocentesis or pericardiotomy.

Although cardiac tamponade is most commonly encountered in patients with penetrating thoracic trauma, it can result from blunt injury to the thorax.

Tachycardia, muffled heart sounds, and dilated, engorged neck veins with hypotension and insufficient response to fluid therapy suggest cardiac tamponade.

However, the absence of these classic findings does not exclude the presence of this condition. Tension pneumothorax can mimic cardiac tamponade, with findings of distended neck veins and hypotension in both. However, absent breath sounds and hyperresonant percussion are not present with tamponade. Echocardiography may be useful in diagnosing tamponade and valve rupture, but it is often not practical or immediately available in the ED. FAST performed in the ED can identify pericardial fluid, which suggests cardiac tamponade as the cause of shock. Cardiac tamponade is best managed by formal operative intervention, as pericardiocentesis is at best only a temporizing maneuver.

Tension Pneumothorax. Tension pneumothorax is accumulation of air in the pleural space under pressure, compressing the lungs and decreasing venous return to the heart.

Tension pneumothorax is a true surgical emergency that requires immediate diagnosis and treatment. It develops when air enters the pleural space, but a flapvalve mechanism prevents its escape. Intrapleural pressure rises, causing total lung collapse and a shift of the mediastinum to the opposite side, with subsequent impairment of venous return and a fall in cardiac output. Spontaneously breathing patients often manifest extreme tachypnea and air hunger, while mechanically ventilated patients more often manifest hemodynamic collapse. The presence of acute respiratory distress, subcutaneous emphysema, absent unilateral breath sounds, hyperresonance to percussion, and tracheal shift supports the diagnosis of tension pneumothorax and warrants immediate thoracic decompression without waiting for x-ray confirmation of the diagnosis. Needle or finger decompression of tension pneumothorax temporarily relieves this lifethreatening condition. Follow this procedure by placing a chest tube using appropriate sterile technique.

SUMMARY

1. Shock is an abnormality of the circulatory system that results in inadequate organ perfusion and tissue oxygenation.

2. Hemorrhage is the cause of shock in most trauma patients. Treatment of these patients requires immediate hemorrhage control and fluid or blood replacement. Stop the bleeding.

3. Diagnosis and treatment of shock must occur almost simultaneously.

4. Initial assessment of a patient in shock requires careful physical examination, looking for signs of tension pneumothorax, cardiac tamponade, and other causes of shock.

5. Management of hemorrhagic shock includes rapid hemostasis and balanced resuscitation with crystalloids and blood.

6. The classes of hemorrhage and response to interventions serve as a guide to resuscitation.

7. The primary treatment in anaphylactic shock is epinephrine, fluids, and oxygen.

8. Abnormalities in macrovascular, microvascular, endothelial, and mitochondrial function all contribute to the haemodynamic changes and organ failures seen in septic patients.

9. Special considerations in diagnosis and treatment of shock include differences in the response to shock in extremes of age, athleticism, pregnancy, hypothermia, and presence of some medications and pacemakers/ICDs. Avoid the pitfall of equating blood pressure with cardiac output.

TEST QUESTIONS FOR SELF-CONTROL

1. 5 minutes after cephazoic injection, the sick boy lost consciousness, there was swelling of the whole body, polymorphic rash, blood pressure – 30/0 mm Hg, heart rate – 120 / min. The use of which drug in this case is pathogenetically determined?

a) Norepinephrine;
b) Calcium chloride;
c) With prednisolone;
d) Adrenaline;
e) Diphenhydramine.

2. A 52-year-old sick man was hospitalized in the intensive care unit with complaints of intense chest pain, severe shortness of breath, weakness, and palpitations. Consciousness is darkened. The skin is pale, moist, cold. Blood pressure – 60/40 mm Hg, heart rate – 116 / min. Oliguria. ECG: elevation of segment ST I, II, avL, V2-V5, QS V2-V5. Which drug in the case of such a complication is most appropriate to administer to the patient?

a) Bisoprolol;
b) Furosemide;
c) Nifedipine;
d) Amiodarone;
e) Dobutamine.

3. You may see a slight increase in diastolic blood pressure early in shock because

a) Stroke volume has increased;
b) Concurent brain injury is present;
c) Left ventricular contractions have strengthened;
d) There is an increase in vascular tone.

4. Baroreceptors monitor blood pressure and help us to respond to shock. These are found in the:

a) Abdominal aorta;

b) Brain and ventricular muscle tissue;

c) Carotid arteries and aortic arch;

d) Brain and spinal cord.

5. Which of the following is true for vital signs in the shock patient?

a) The pulse will increase and th respirations will increase;

b) The pulse will increase and th respirations will decrease;

c) The pulse will decrease and th respirations will decrease;

d) The pulse will decrease and th respirations will increase.

6. Conceptually, the goal of shock resuscitation is to:

a) Let the blood pressure drop because elevating it may cause bleeding;

b) Get the blood pressure back to normal;

c) Try to restore some perfusion without elevating the BP too much;

d) Resuscitate until the pulse oximeter reads at least 94 %.

7. Shock from anaphylaxis is a result of which type of shock?

a) Hypovolemic shock;

b) Cardiogenic shock;

c) Distributive;

d) Obstructive.

8. Which of the following might lead you to consider obstructive shock over hypovolemic shock?

a) Widening pulse pressure;

b) Distended neck veins;

c) Suspicion of significant closed head injury;

d) Indications of abdominal injury.

9. Which of the following injuries would traditionally create an obstructive shock situation?

a) Acute abdominal bleeding;

b) Increase intracranial pressure;

c) Profound hemothorax;

d) Tension pneumothorax.

10. The primary difference between distributive shock and hypovolemic shock is that

a) Blood volume remains constant in distributive shock;

b) Hypovolemic shock results in decreased perfusion while distributive does not;

c) Distributive shock is most commonly caused by trauma;

d) Hypovolemic shock results in decreased vascular tone.

Keys: 1 D, 2 E, 3 D, 4 C, 5 A, 6 C, 7 D, 8 B, 9 D, 10 A.

SITUATIONAL TASKS

1. A 79-year-old woman with a history of coronary artery disease who underwent a coronary artery bypass graft (CABG) surgery in 2000 is brought to the emergency department (ED) by her family for 2 days of worsening shortness of breath. For the past 2 days, she has not gotten out of bed and is confused. She does not have chest pain, fevers, or cough. Her temperature is 36.7 °C, blood pressure (BP) is 85/50 mm Hg, heart rate (HR) is 125 beats per minute, and respiratory rate (RR) is 26 breaths per minute. On examination, she is unable to follow commands and is oriented only to name. The cardiovascular examination reveals tachycardia with no murmurs. Her lungs have rales bilaterally at the bases. The abdomen is soft, nontender, and nondistended. Lower extremities have 2+ edema to the knee bilaterally. Which of the following is the most likely diagnosis?

a) Hypovolemic shock;
b) Neurogenic shock;
c) Cardiogenic shock;
d) Anaphylactic shock;
e) Septic shock.

2. A 19-year-old man was struck by a motor vehicle while crossing the street. In the ED, he is awake, alert, and oriented, but complaining of severe right-leg pain. His temperature is 36.9°C, BP is 85/50 mm Hg, HR is 125 beats per minute, and RR is 24 breaths per minute. You confirm that his airway is patent, breath sounds are equal bilaterally, and his abdomen is soft and nontender. His right leg is shorter than his left leg, slightly angulated, and swollen in his anterior thigh area. There is no open wound.
Which of the following is the most likely diagnosis?

a) Hypovolemic shock;
b) Neurogenic shock;
c) Cardiogenic shock;
d) Anaphylactic shock;
e) Septic shock.

3. An 85-year-old man is rambling incoherently and not eating at his nursing home. Records indicate that he has a past medical history of hypertension, diabetes, dementia, and benign prostatic hypertrophy. On arrival to the ED, the patient is combative and oriented only to name. His temperature is 38.3 °C rectally, BP is 85/50 mm Hg, HR is 125 beats per minute, RR is 22 breaths per minute, and blood sugar is 154 mg/dL. He appears uncomfortable and cachectic. His lungs are clear to auscultation, with scant crackles at the bases, and his abdomen is soft, nontender, and nondistended. He has a Foley catheter

in place draining cloudy, white urine. He has no peripheral edema. Which of the following is the most likely diagnosis?

a) Hypovolemic shock;
b) Neurogenic shock;
c) Cardiogenic shock;
d) Anaphylactic shock;
e) Septic shock.

4. A 34-year-old woman with no known medical problems is having a sushi dinner with her husband. Halfway through dinner, she begins scratching her arms and her husband notices that her face is flushed. The itching intensifies and she begins to feel chest pain, shortness of breath, and dizziness. On arrival to the ED, she can barely talk. Her temperature is 37.7 °C, BP is 85/50 mm Hg, HR is 125 beats per minute, and RR is 26 breaths per minute, and oxygen saturation is 91 % on room air. Which of the
following is the most likely diagnosis?

a) Hypovolemic shock;
b) Neurogenic shock;
c) Cardiogenic shock;
d) Anaphylactic shock;
e) Septic shock.

5. A 25-year-old man fell off his surfboard and landed on rocks. He was pulled from the water by lifeguards and brought to the ED in full cervical and spinal immobilization. He is alert and oriented to person, place, and time. He is complaining of weakness in all of his extremities. His temperature is 36.6 °C, BP is 85/50 mm Hg, HR is 60 beats per minute, RR is 20 breaths per minute, and oxygen saturation is 98% on room air. On examination, he has no external signs of head injury. His heart is bradycardic without murmurs. The lungs are clear to auscultation and the abdomen is soft and nontender. He has grossly normal peripheral sensation, but no motor strength in all four extremities. Which of the following is the most likely diagnosis?

a) Hypovolemic shock
b) Neurogenic shock
c) Cardiogenic shock
d) Anaphylactic shock
e) Septic shock

Standards of answers: 1 C, 2 A, 3 E, 4 D, 5 B.

REFERENCES

1. Baker, S. Y., Tarkowski, A. F., Falk, J. L. (2020) Shock Overview. In: Shiber J., Weingart S. (eds) Emergency Department Critical Care. Springer, Cham. https://doi.org/10.1007/978-3-030-28794-8_1
2. Macdonald, G. (2008), Harrison's Internal Medicine, 17th edition. - by A. S. Fauci, D. L. Kasper, D. L. Longo, E. Braunwald, S. L. Hauser, J. L. Jameson and J. Loscalzo. Internal Medicine Journal, 38: 932-932. https://doi.org/10.1111/j.1445-5994.2008.01837.x
3. Waldmann, C., Soni, N., Rhodes, A. (2008) Oxford desk reference: critical care. Oxford: Oxford University Press. DOI:10.1093/med/9780199229581.001.0001
4. Kasper, D. L., Braunwald, E., Fauci, A. S., Hauser, S. L., Longo, D. L., & Jameson, J. L. (Eds.). (2005). Harrison's manual of medicine. New York: McGraw-Hill.
5. Standl, T., Annecke, T., Cascorbi, I., Heller, A. R., Sabashnikov, A., & Teske, W. (2018). The Nomenclature, Definition and Distinction of Types of Shock. Deutsches Arzteblatt international, 115(45), 757–768. https://doi.org/10.3238/arztebl.2018.0757
6. Rosh, A.J., & Barclay-Buchanan, C.J.(Eds.), (2016). Emergency Medicine: PreTest® Self-Assessment and Review, 4e. McGraw Hill. https://accessemergencymedicine.mhmedical.com/content.aspx?bookid=1888&sectionid=136157953
7. Ring, J., Beyer, K., Biedermann, T. et al. (2021) Guideline (S2k) on acute therapy and management of anaphylaxis: 2021 update. Allergo J Int**:** 30, 1–25. https://doi.org/10.1007/s40629-020-00158-y
8. Thompson, J.P., Moppett, I.K., Aitkenhead, A.R. (2019) Textbook of Aneathesia. 7th edition. Elsevier, p. 960.
9. Anesthesiology and Intensive Care. (2010) Edited by F. Glumcher. Medicine Publishing, Kyiv, p. 310.
10. The ESC Textbook of Intensive and Acute Cardiovascular Care (Second Edition) (2015). Edited by M. Tubaro, P. Vranckx - Oxford University Press. DOI: 10.1093/med/9780199687039.001.0001
11. Order №1269 of the Ministry of Health of Ukraine "On approval and implementation of medical and technological documents for standardization of emergency medical care" from 05.06.2019 https://moz.gov.ua/article/ ministry-mandates/nakaz-moz-ukraini-vid-05062019--1269-pro-zatverdzhennja-ta-vprovadzhennja-mediko-tehnologichnih-dokumentiv-zi-standartizacii-ekstrenoi-medichnoi-dopomogi

12. Ji, J., & Brown, D. L. (2018). Distributive shock. In Cardiac Intensive Care (pp. 208-215.e4). Elsevier. https://doi.org/10.1016/B978-0-323-52993-8.00021-7
13. Dave, S., & Cho, J. J. (2021). Neurogenic Shock. In *StatPearls*. StatPearls Publishing. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459361/
14. Pich, H., & Heller, A. R. (2015). Obstruktiver Schock [Obstructive shock]. Der Anaesthesist, 64(5), 403–419. https://doi.org/10.1007/s00101-015-0031-9
15. McEvoy, M., Furse, C., Sobol, J., & Louro, J. (2017-07). Obstructive Shock. In Advanced Perioperative Crisis Management. Oxford, UK: Oxford University Press. Retrieved 30 Nov. 2021, from https://oxfordmedicine.com/view/10.1093/med/9780190226459.001.0001/med-9780190226459-chapter-12.
16. Litell, J., & Shapiro, N. (2016-04). Pathophysiology of septic shock. In Oxford Textbook of Critical Care. Oxford, UK: Oxford University Press. Retrieved 1 Dec. 2021, from https://oxfordmedicine.com/view/10.1093/med/9780199600830.001.0001/med-9780199600830-chapter-297.

FOR NOTES

Навчальне видання

Матвєєнко Марія Сергіївна
Белозьоров Ігор Вікторович
Баранова Надія Вікторівна

ШОК

Методичні рекомендації до практичних занять для здобувачів вищої медичної освіти 5-го року навчання з дисципліни «Екстрена та невідкладна медична допомога»

(Англ. мовою)

Комп'ютерне верстання *Н. О. Ваніна*
Макет обкладинки *І. М. Дончик*

Формат 60 × 84/16. Ум. друк. арк. 3,1. Наклад 100 пр. Зам. № 328/21.

Видавець і виготовлювач
Харківський національний університет імені В. Н. Каразіна,
61022, Харків, майдан Свободи, 4.
Свідоцтво суб'єкта видавничої справи ДК № 3367 від 13.01.2009

Видавництво ХНУ імені В. Н. Каразіна
Тел. 705-24-32