SAFETY

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SIMULATION APPROACH FOR EDUCATION AND TRAINING IN EMERGENCY

SHOCK

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Summary

Content

This text delivers basic knowledge about clinical management of shock. The target group are 5th year medical students (or above).

Definition

Shock is a life-threatening condition. It is the clinical expression of the final phase of circulatory failure (CF). CF is characterized by an acute and inadequate oxygen delivery and/or increased consumption and/or inadequate oxygen utilization leading to cellular hypoxia and organ dysfunction. Effects of shock might be reversible in the early stages, however, delay in diagnosis and treatment can lead to multiorgan failure (MOF) and death.

Diagnosis

The diagnosis of shock is based on clinical signs and biochemical markers of **inadequate tissue perfusion** and **hemodynamic parameters**.

Inadequate tissue perfusion

Clinical signs of tissue hypoperfusion. Shock is associated with inadequate tissue perfusion on physical examination. The "windows" of the body to evaluate hypoperfusion are:

- ***Skin***: cold and clammy skin, acrocyanosis, slow capillary refill time (commonly defined as >2 seconds)
- ***Kidneys:*** decreased urine output (<0,5ml/kg/h)
- ***Brain:*** altered mental status (confusion, disorientation and/or reduced level of consciousness)
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Biochemical markers of tissue hypoperfusion. Elevated lactate levels (> 2mmol/L) indicate abnormal metabolism. Mixed venous oxygen saturation (SvO_2) or central venous oxygen saturation ($ScvO_2$) - if available - provides information about the balance between oxygen supply and demand. A low SvO_2 or $ScvO_2$ associated with elevated lactate suggests impairment of oxygen supply. A normal/high SvO_2 or $ScvO_2$ in this context is less relevant, because a high value does not confirm adequate oxygen supply. For example, if microcirculation is altered, oxygen extraction will be impaired and therefore local hypoperfusion is present. However, mixed venous oxygen saturation (SvO_2) or central venous oxygen saturation ($ScvO_2$) will remain normal or high.

Hemodynamic parameters

Arterial hypotension is defined as a systolic blood pressure (SBP) of < 90mmHg or a decrease of 40mmHg from baseline blood pressure or a mean arterial pressure (MAP) of < 65mmHg. Arterial

hypotension is generally present in shock states, but should not be required to define shock. Hypotension is usually accompanied by tachycardia, also in later shock states.

Pathophysiological classification and etiology of shock

From a very simplified point of view, the circulatory system is composed by three elements: The heart or the pump, the vascular system or the container and the circulating volume or the content. The pathophysiological classification of shock is based on which of these three elements is first affected *(Figure 1):* ***a) Hypovolemic shock*** is caused by internal or external fluid loss that leads to a decreased circulating volume, i.e. a reduced content. It can be haemorrhagic or non-haemorrhagic, **b) Cardiogenic shock** is caused by a pump failure ***c) Obstructive shock*** is caused by an impairment of the continuity of the vascular system, i.e. a problem with the container, resulting in an impaired flow of the content ***d) Distributive shock*** is caused by loss of vascular tone that results in maldistribution of blood flow, i.e. a too large container.

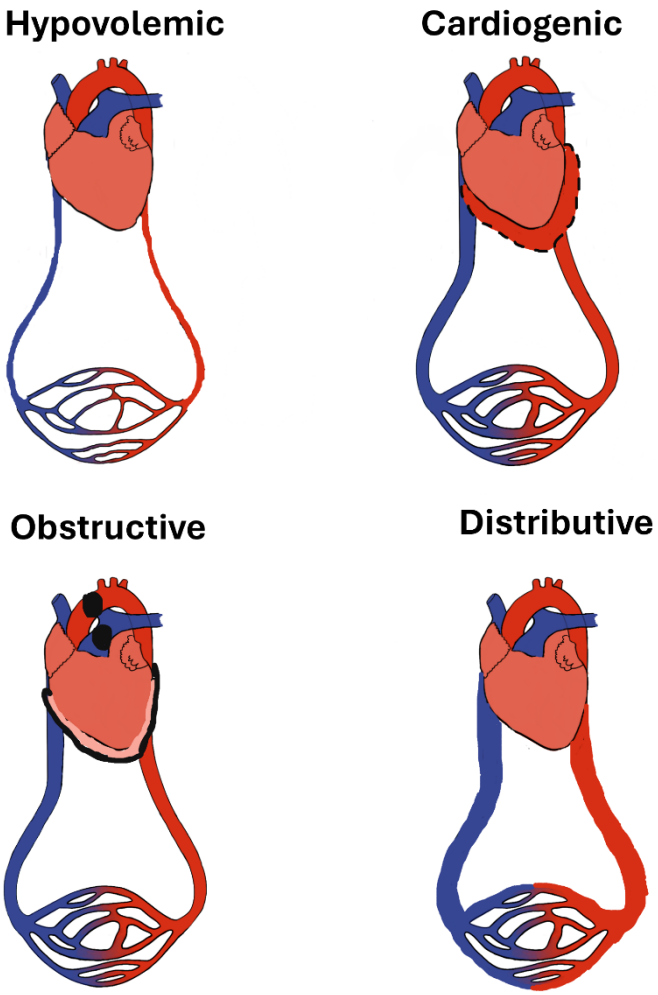

Figure 1: Pathophysiological classification of shock

From a hemodynamic point of view, hypovolemic, cardiogenic and obstructive shock are characterized by a low cardiac output (CO), with decreased stroke volume and increased systemic vascular resistance leading to inadequate oxygen transport. In contrast distributive shock presents, initially, with increased CO and decreased systemic vascular resistance and in the particular case of septic shock altered oxygen extraction.
The classification presented above is a very simplistic one. Shock can result from one mechanism or a combination of them. For example, a patient with a septic shock can also have a cardiogenic shock due to septic myocardial depression, and in this particular case, septic shock might present with low CO. A traumatic patient might have a haemorrhagic shock secondary to spleen rupture and an obstructive shock due to tension pneumothorax.

Main etiologies according to the type of shock are listed in *table 1*

Table 1: Etiology of types of shock

Type of shock	**Etiology**
Hypovolemic	- Internal or external bleeding - Gastrointestinal losses (vomiting, diarrhoea…) - Renal losses (medication induced diuresis, endocrine disorders…) - Skin losses (burns…)
Cardiogenic	- Cardiomyopathies (myocardial ischemia, infarction, myocarditis, Takotsubo syndrome…) - Cardiac arrythmia (ventricular tachycardia or high degree A-V block) - Mechanical (valvular disease)
Obstructive	- Cardiac tamponade - Hypertrophic obstructive cardiomyopathy (HOCM) - Pulmonary embolism - Tension pneumothorax - Pneumomediastinum - Supine hypotensive syndrome (inferior vena cava compression syndrome) in late pregnancy
Distributive	- Severe sepsis - Anaphylaxis - Spinal injury

Steps for the diagnosis and management of shock

Shock is a life-threatening clinical condition that requires acting fast to avoid permanent organ damage, multiple-organ failure and death. Early diagnosis and initial supportive therapy are crucial. Moreover, finding the etiology of shock is essential to start specific treatment and to stop further patient deterioration. We propose a 5-step actuation sequence.

Step 1: Recognition of shock

The first and most important step is to recognize the shock, especially in an early stage! The later the diagnosis is made, the later the therapy starts, the worse the outcome of shock might be.
As mentioned before, the diagnosis of shock is based on clinical signs and laboratory markers of inadequate tissue perfusion and hemodynamic parameters *(Figure 2)*. Although hypotension is usually present in shock, there may be clinical situations of shock without hypotension, specially at early stages. For instance, some acute aortic syndromes or acute heart failure may cause an oxygen delivery-consumption imbalance and still do not show with hypotension at their initial clinical presentation.

The challenge might be to recognize a shock at early stages, when clinical signs and symptoms have changed only discretely, especially when the hemodynamic is still compensated. For instance, the Advanced Trauma Life Support group show us with their classification of haemorrhagic shock that a patient may lose 15% of their blood and still have normal blood pressure and heart rate below 100 beats/minute. At that point clinical expression of shock may be subtle. It is only when the patient loses 30-40% of their blood that hemodynamics are clearly impaired (hypotension and heart rate above 120 beats/minute).

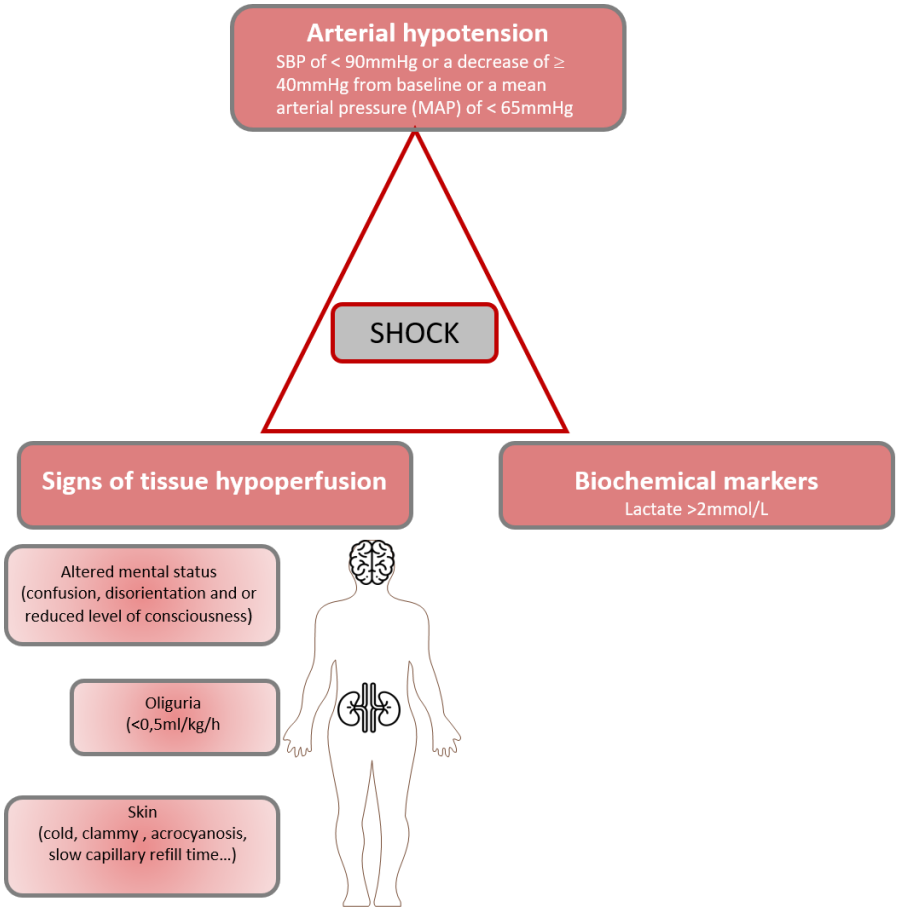

Figure 2: Symptoms of shock

Step 2: Start supportive therapy

Once shock is recognized, supportive treatment should start as soon as possible, even though the underlaying cause is unknown. The main goal of supportive therapy is to restore tissue perfusion by improving oxygen delivery (DO_2) and perfusion pressure. DO_2 depends on CO and oxygen arterial content (CaO_2) while perfusion pressure depends on CO and systemic vascular resistance (SVR) *(Figure 3)*

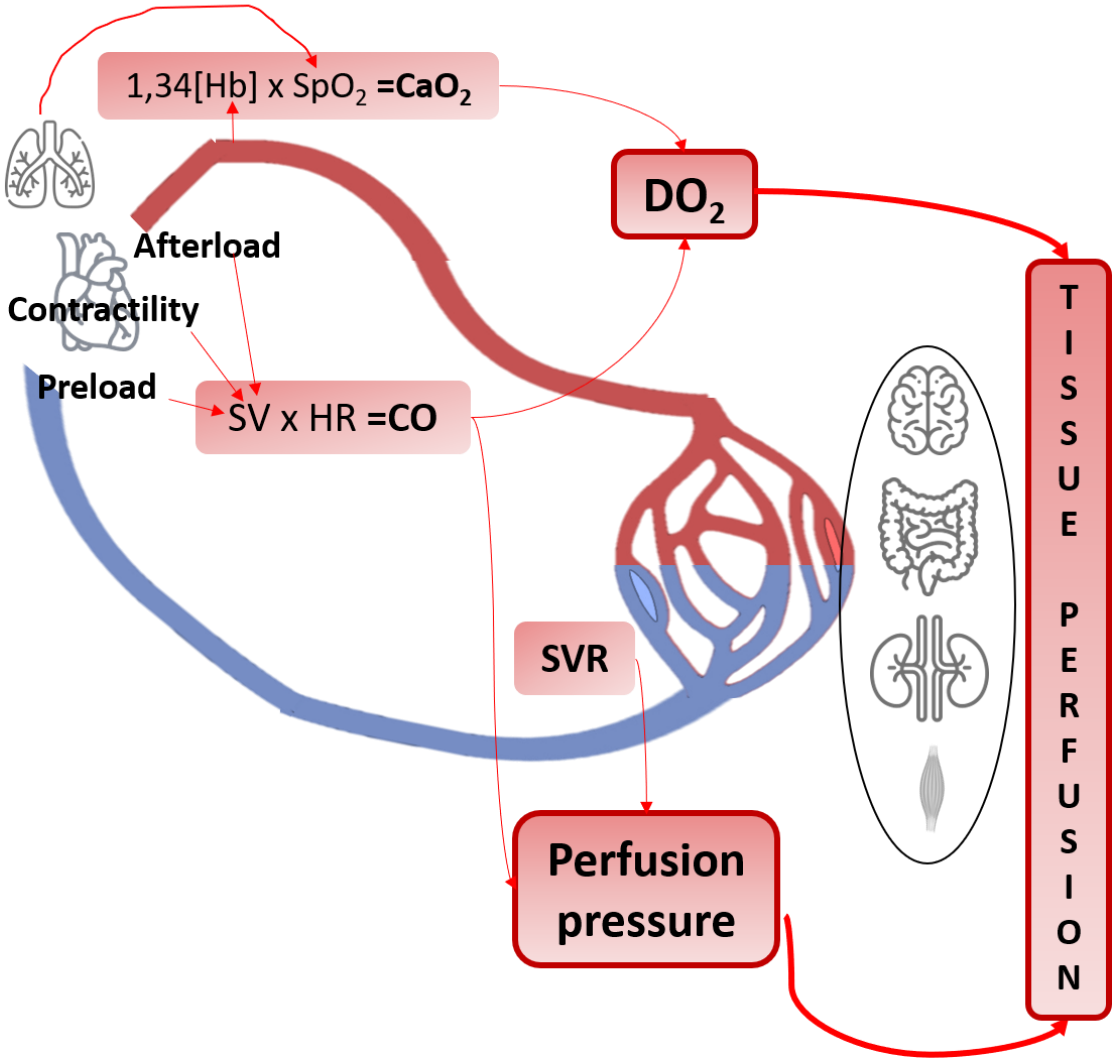

Figure 3: supportive therapy of a shock
Hb: Haemoglobin, SpO_2: Oxygen saturation, CaO_2: oxygen arterial content, DO_2: oxygen delivery, SV; stroke volume, HR: heart rate, CO: cardiac output, SVR: systemic vascular resistance

Respiratory support

The main goal of oxygen therapy is to increase oxygen saturation and thus oxygen delivery (DO_2). A careful respiratory assessment should be performed as soon as possible to determine the amount of oxygen and the way of administration that the patient needs. Respiratory support therapy goes from conventional oxygen therapy in spontaneous breathing to veno-venous ECMO *(Figure 4)*

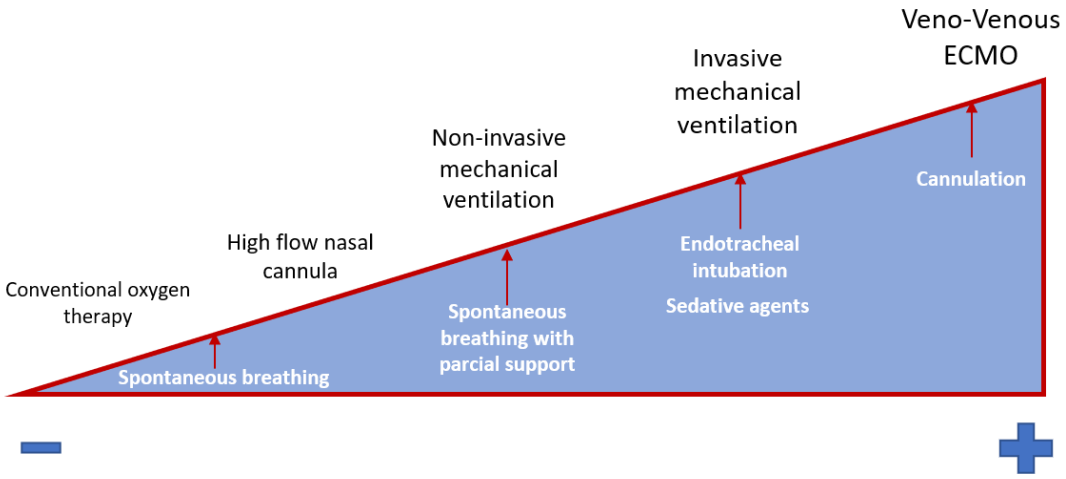

Figure 4: Steps to improve oxygenation

Hemodynamic support

The main goal of hemodynamic support is to maintain an optimal CO according to the requirements of the patient, adequate systemic vascular resistance (SVR) and therefore DO_2 as well as perfusion pressure. This goal can be achieved with fluid resuscitation, administration of vasopressors and/or ionotropic agents and/or vasodilators and instauration of mechanical support if necessary.

a) ***Fluid resuscitation.***

Fluid resuscitation has been a trendy topic, and it would probably continue this way. Then, the only irrefutable statement regarding fluid resuscitations is that there is not irrefutable statement regarding fluid resuscitation. The basics of fluid resuscitation is to improve oxygen delivery and blood pressure. First, fluid resuscitations would improve oxygen delivery by increasing preload and therefore stroke volume and CO. This improvement in CO would also have a positive impact on blood pressure. Once these basics are understood, the one size fits all concept must be buried and health care professionals should embrace the holistic concept that the type and amount of fluid may change according to the clinical status of the patient. Health care professionals should also have the same considerations about fluid administrations as they would have about other medical treatments. Fluid administration may have serious side effects on patients. For instance, some authors have described the fluid overload syndrome. On the other hand, a systematic restrictive fluid approach would miss life-threatening situations when fluid resuscitation is vital. There is also controversial evidence about what type of fluid to use. For instance, the eternal discussion about colloids

vs crystalloids remains on the table. Therefore, it seems like the how is more important than the what.

There have been some attempts to try to guide fluid administration. However, the perfect equations have not arrived yet. For instance, static parameters of preload (central venous pressure or pulmonary artery occlusion pressure) are not useful to predict fluid responsiveness or to evaluate the effect of fluid administration. Dynamic parameters of preload (pulse pressure variation, stroke volume variation, aortic flow variation or vena cava collapsibility or distensibility) are helpful to differentiate between preload dependent or independent patients. However, preload dependence does not imply that fluids should be administered. For example, a patient with a diagnose of septic shock and a distress respiratory syndrome may be hypotensive and preload-dependent but also hypoxemic, so fluid administration may aggravate this condition.

Dynamic parameters are preferred to static parameters, when they are available and applicable. Static parameter may be useful in early shock resuscitation with very low or very high values. Transfusion of red blood cells may be part of fluid resuscitation in the context of haemorrhagic shock to improve haemoglobin and as a result arterial oxygen content (CaO_2). Also, other blood products might be helpful like fresh frozen plasma or platelets to improve coagulation, when indicated.

b) Vasopressors

Vasopressors increase vascular systemic resistance and restore blood pressure. Here, the main goal is to achieve a proper perfusion pressure. However, the proper perfusion pressure value varies within the patient, depending on the organ, and among different patients.
Therefore, a target blood pressure during shock resuscitation should be individualized according to the patient and the clinical context. In general, an ***initial target of MAP of [3] 65mmHG is recommended***. Experts suggest to tolerate a lower threshold in patients with uncontrolled bleeding and a higher one in patients with medical history of hypertension and distributive shock.

Most of the guidelines consider ***norepinephrine*** the first-choice vasopressor. Other alternatives are ***dopamine***, but it has no advantages over norepinephrine and it induces more arrhythmias, and some authors may not recommend it in septic shock. ***Epinephrine*** is recommended as a second-line agent for severe cases, but it is the first-line choice in anaphylactic shock. ***Vasopressin*** and ***terlipressin*** have also been used as second-line vasopressors in the context of septic shock.

c) Inotropic agents

Inotropic agents increase contractility and thus increasing stroke volume and CO. Evaluation of cardiac function by means of echocardiography is essential when deciding if inotropic agents are the therapy of choice for a given patient.
Target CO during shock resuscitation is difficult to define, a predefined CO target for all patients is not a good strategy since the CO needed varies among patients and in a same patient over the time. Therefore, target CO should be individualized and integrated with other parameters.

Current guidelines give different recommendations for the use of inotropes and there is a lack of evidence of the beneficial effects of these agents in shock. ***Dobutamine*** is considered the inotropic agent of choice. Other alternatives are phosphodiesterase type III inhibitors (milrinone and enoximone) and calcium sensitizer (levosimendan). However, hey have strong limitations, because they can cause severe hypotension and have a long-half live.

d) Vasodilators

Vasodilators increase CO by decreasing ventricular afterload. Their use in shock is limited to specific circumstances (e.g. in cardiac shock) and their major drawback is the risk of hypotension.

Nitrates are the most common vasodilators used in this context.

e) Mechanical circulatory support

Temporary mechanical circulatory support may be a therapeutic alternative in patients with refractory shock, especially cardiogenic shock. Mechanical circulatory support requires specialist expertise for implantation and management, and it might be associated with severe complications.

The mechanical circulatory support devices available are: intra-aortic balloon pump (AIBP) and extracorporeal life support devices (Impella, TandemHeart and venous-arterial ECMO). They have very different active principles how the work.

Step 3: Identify the type and cause of shock

Once the diagnosis of shock is done and the initial support therapy has been started, the next step is the attempt to identify the type and its underlying cause. STEP 3 should be done simultaneously with STEP 2, resuscitation should continue while searching for the cause of shock.

The key elements for narrowing the diagnosis can be divided in patient related (medical history, current symptoms and clinical context, physical examination, vital signs) and workup results (laboratory, electrocardiogram, chest X-ray or pulmonary echography and echocardiography *(Figure 5 and table 2).*

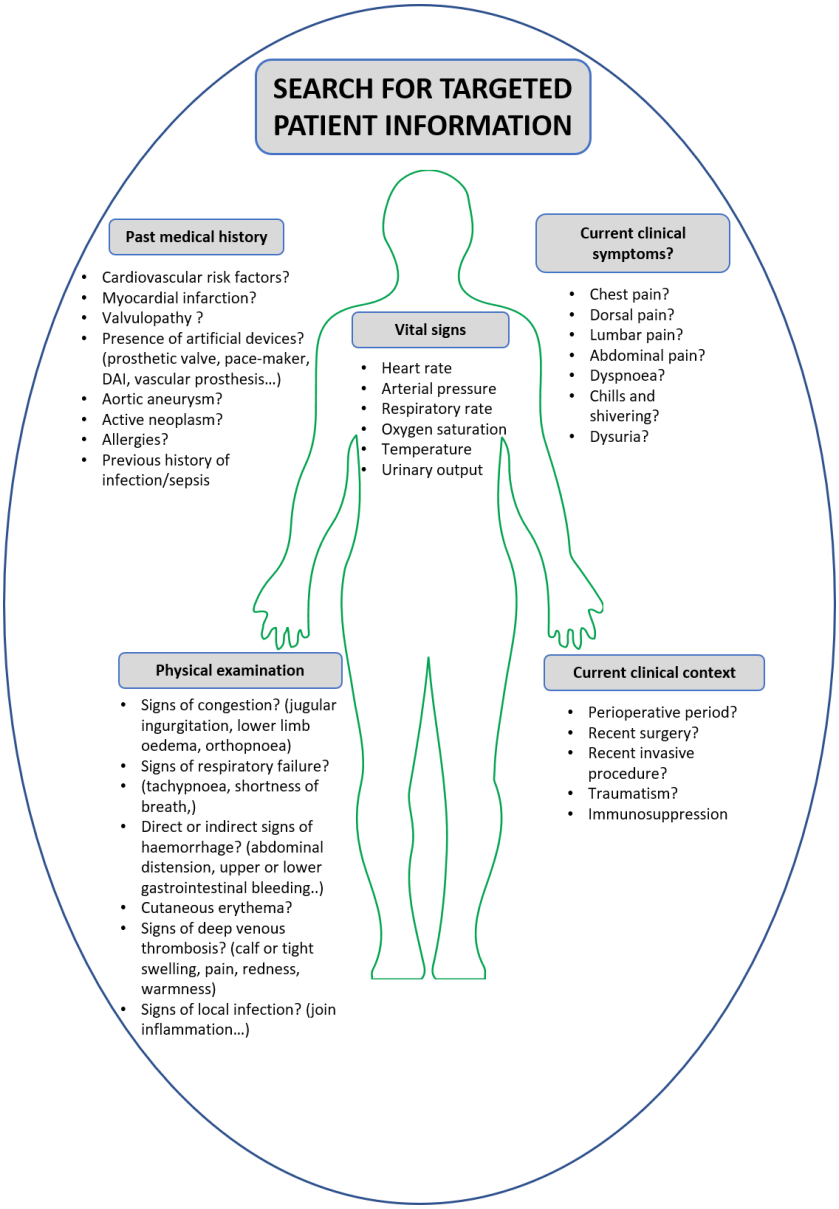

Figure 5: Patient information

Echocardiographic evaluation is proposed as the first-line evaluation modality in a clinical setting and should be performed as soon as possible in a patient with shock. It allows rapid differential diagnosis of the type of shock and might confirm its primary cause. It is a non-invasive and immediate technique that can be performed bedside, and the results will be obtain within minutes. Furthermore, it allows sequential evaluation once the initial treatment has started. As listed in table 2, echocardiography provides information about ventricular function and valve performance, allows estimation of CO and left ventricle filling pressures, discards or confirms the presence of pericardial effusion and cardiac tamponade, and provides dynamic parameters of preload responsiveness.

Table 2: Work-up for diagnosis of type of shock and etiology		
WORK-UP		**POSSIBLE UNDERLYING CAUSE**
Laboratory	***Hemogram***	Low Hb suggests haemorrhagic shock High white blood cells suggest infection Low white blood cell account suggest immunosuppression (Ei. Febrile neutropenia)
	Coagulation	Use to guide transfusion in haemorrhagic shock
	Creatinine and electrolytes	Kidney function
	Liver function test	High values may suggest acute liver disease (if chronic liver disease is excluded) or right ventricular failure
	Pancreatic function test	High values may suggest pancreatic disease
	C-reactive protein ***Procalcitonin***	High values suggest inflammatory process High values suggest infection
	Troponin	High values suggest myocardial ischemia Pulmonary thromboembolism might also present with elevated troponin values
	NT-ProBNP	High values suggest heart failure
	Lactate	High values suggest tissue hypoperfusion. Lactate value is useful for the diagnosis of shock and for evaluating patient response to therapy. Lactate values may not vary very quickly and may have some delay from the current clinical status. They can also be elevated with the use of adrenaline.
	D-Dimer	High values suggest pulmonary embolism
	SvO_2 or $ScvO_2$	A low SvO_2 or $ScvO_2$ suggests impairment of oxygen supply. A normal/high SvO_2 or $ScvO_2$ is less informative, because a high value does not confirm adequate oxygen supply. It can be used for evaluating response to therapy
12-lead electrocardiogram		Abnormal in myocardial ischemia, pulmonary thromboembolism and tachy- and bradyarrhythmia.
Chest X-ray	Chest X-ray may be replaced by pulmonary echocardiography	Confirms the diagnosis of acute respiratory distress syndrome that might accompany septic shock Confirms the diagnosis of pulmonary oedema that might suggest cardiogenic shock due to left ventricular failure Confirms the diagnosis of tension pneumothorax Confirms the diagnosis of pneumonia that might accompany septic shock
Echocardiography	***Evaluate left ventricular function***	Low left ventricular systolic function suggests cardiogenic shock. Left ventricular systolic disfunction may also be present in septic shock
	Evaluate right ventricular size and function	Low right ventricular systolic function suggests cardiogenic shock.

		Right ventricular disfunction may also be present in septic shock Low ventricular systolic function and dilated right ventricle might suggest pulmonary thromboembolism
	Evaluate pericardium	Pericardial effusion with cavity collapse and Doppler variations suggests obstructive shock due to pericardial tamponade. If pericardial tamponade is present try to discard rupture of free wall and aortic dissection type A
	Evaluate left ventricle outflow tract (LVOT)	Obstruction in the LVOT and systolic anterior movement of the mitral valve suggest obstructive shock, due hypertrophic obstructive myocardiopathy
	Evaluate aortic valve	Severe stenosis or regurgitation suggests cardiogenic shock. Aortic regurgitation might be acute due to endocarditis or aortic dissection
	Evaluate mitral valve	Severe stenosis or regurgitations suggests cardiogenic shock. Mitral regurgitation might be acute due to endocarditis, aortic dissection, papillary rupture caused by myocardial infarction
	Measure left ventricle outflow tract (LVOT) and* Velocity Time Integral *(VTI)	This measurement allows calculation of stroke volume and cardiac output
	IVC variations and dimensions	The result helps to identify the type of shock, e.g. a reduced mean systemic venous pressure and/or increased resistance to the venous return

Step 4: Management of the underlaying cause of shock

Some causes of shock have a specific management (Table 3). Once the cause is identified it must be corrected as soon as possible. Early identification and treatment of the underlying cause may improve the outcome.

Table 3: Management of the underlying cause of shock	
CAUSE OF SHOCK	**SPECIFIC MANAGEMENT**
Hemorrhagic shock	Stop the bleeding - haemorrhage control (Surgery and/or embolization) Transfusion (restoring haemoglobin and treating coagulopathy)
Anaphylactic shock	Stop possible causal agent Vasopressor of choice: adrenaline
Septic shock	Culture sample and afterwards start empirical broad-spectrum antibiotics Infection source control (surgery, abscess drainage…)
Myocardial infarction	Emergency myocardial revascularization (percutaneous or surgical). Surgical intervention might be the first choice if a mechanical complication

	is present (severe mitral regurgitation due to papillary muscle rupture, ventricular septal rupture, free wall rupture). Fibrinolysis might be an alternative if percutaneous or surgical revascularization cannot be performed within 120min from the diagnosis
Valvulopathy	Surgical or percutaneous repair
Tachyarrhythmia	Electrical cardioversion Pharmacological cardioversion
High degree AV block	Pacemaker (transcutaneous or temporary)
Pericardial tamponade	Pericardial drainage (except in the cases of free wall rupture or aortic dissection)
Pulmonary thromboembolism	Fibrinolysis Percutaneous mechanical thrombectomy (if fibrinolysis is contraindicated)
Tension pneumothorax	Thoracic drainage
Pneumomediastinum	Initially needle aspiration Surgical intervention in case of oesophageal or bowel rupture
Severe burns	Surgical intervention in a burn centre
Supine hypotensive syndrome (inferior vena cava compression syndrome) in late pregnancy	Positioning of the patient on her left side
Spinal injury	Immobilization and management in a spinal trauma Centre

Step 5: Monitoring

Unless the clinical situation is rapidly reversed:

- An ***arterial catheter*** should be inserted for monitoring continuous blood pressure and blood sampling.
- A ***central venous catheter*** should be inserted for the administration of vasoactive agents, $ScVO_2$ and central venous pressure measurement. Central venous pressure (CVP) is a static preload parameter and does not predict fluid responsiveness and should not be used to guide fluid resuscitation. CVP may be useful identifying the type of shock in extreme ranges and interpreted together with other variables. For example, a hypovolemic shock will present with a very low CVP value and an obstructive shock with a very high one.

As mentioned earlier, **echocardiography** allows orientation of the type and underlaying cause of shock. It also allows sequential evaluation of cardiac function, CO, left ventricle filling pressures and preload responsiveness. However, echocardiography it is operator-dependent, and it does not provide continuous monitoring.

Advanced hemodynamic monitoring may be needed in patients with severe or refractory shock to initial therapy and in complex conditions. **Continuous CO monitoring** might be useful to evaluate the response to fluids and/or inotropes in patients who are not responding to initial therapy. Pulmonary artery catheterization might be useful in patients with refractory shock and right ventricular disfunction or acute respiratory distress syndrome. Transpulmonary thermodilution might be useful in patients with severe shock and acute respiratory distress syndrome. When using less invasive devices to monitor CO, it is essential to confirm that they have been validated in the context of shock.

Step 6: Evaluation of response to therapy

We will assess the response to therapy based on the same clinical, laboratory and other tests conducted to identify the state of shock and diagnose the etiology of the shock. As mentioned above, the goal of supportive therapy is to restore tissue perfusion by improving oxygen delivery (DO_2) and perfusion pressure *(Figure 3).*
The evaluation of response to therapy is complex. We can use tissue perfusion parameters, such as lactate, SvO_2 or urine output /or DO_2 determinants such as CO. More than one variable should be used to evaluate therapy response and different values should be integrated together.

a) ***Lactate.*** As mentioned before, elevated blood lactate level in shock states reflects anaerobic metabolism secondary to tissue hypoxia. It may be useful for the diagnosis, but serial measurements can help us to evaluate the response to therapy. Changes in lactate values are slower that arterial pressure or CO, but a decrease in lactate level over a period of hours translates in effective therapy.

b) ***Urine output***. As for lactate values, improvement of urinary output is slower than other parameters such as arterial pressure or CO. It is also conditioned by the degree of renal impairment.

c) ***SvO_2 or $ScvO_2$***: Mixed venous oxygen saturation (SvO_2) or central venous oxygen saturation ($ScvO_2$) They are useful in assessing the balance between oxygen delivery and oxygen supply and in the interpretation of CO. A low SvO_2 / $ScvO_2$ suggests impairment of oxygen supply and elevated oxygen extraction ratio. Improvement in SvO_2 or $ScvO_2$, should be interpreted carefully, since a high value does not confirm adequate oxygen supply. A high value may be the consequence of altered oxygen extraction due to microvascular dysfunction.

d) **CO (and SV)**. It may be measured discontinuously with echocardiography or continuously with advanced monitoring techniques such as pulmonary or transpulmonary thermodilution. Less advanced monitoring techniques are available but not all of them are validated in shock states. CO advanced monitoring is not recommended in the early phase of shock, and it is reserved for refractory to initial therapy or severe shock. CO is useful to evaluate the response to fluid therapy and inotropes. Targeting a predefined CO value is not recommended. The CO needed will vary among patients and in the same patient over the time. Therefor we should aim a adapted CO value more than an absolute value.

e) **Arterial pressure**. Arterial pressure may increase in response to administration of vasopressors and fluids. Target value of arterial pressure have been discussed in section 4 (supportive therapy).

Figure 6 shows the 6 steps.

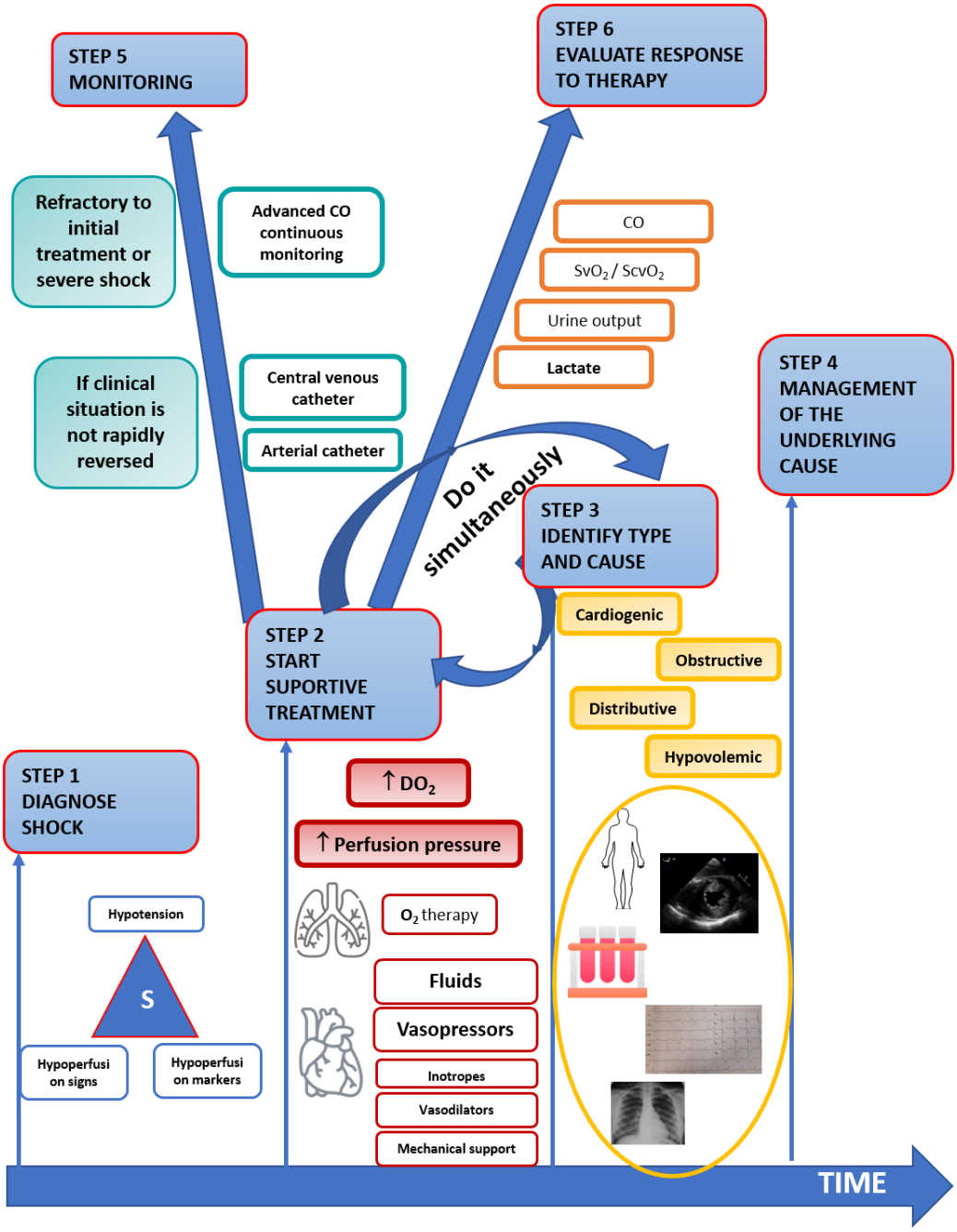

Figure 6: standardized approach for shock treatment

Student skills needed

Prerequisites

We suggest basic knowledge of haemodynamic physiology as well as cardiac and circulatory anatomy.

Diagnostic skills

The student must be able to:

- Perform a directed anamnesis and physical examination to diagnose shock and its underlying cause.

- Interpret the results of the initial work-up: laboratory, electrocardiogram, chest-X ray and echocardiogram.

Monitoring skills

The student must:

- Know the indications, contraindications, potential complications, and material needed and technique of insertion of an arterial catheter.

- Know the indications, contraindications, potential complications, and material needed and technique of insertion of a central venous catheter.

- Know the different devices available to CO monitoring and its indications, value interpretation, advantages, drawbacks, degree of invasiveness, contraindications and possible complications.

Therapy skills

The student must know:

- The different types of respiratory support and its indications.
- How to perform an endotracheal intubation.
- How to prescribe vasopressors, inotropes and vasodilators.
- The indications for mechanical circulatory support.
- The specific management of the different causes of shock.

Bibliography

You will find the bibliographical references of this chapter in the corresponding video in QR code format.