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Chapter 2

Shock

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Abstract

This chapter targets to provide information to medical students, residents, and nurses on emergency medicine. The concept and definitions of shock have been mentioned. An overview about shock has been given at the beginning of the chapter. Different categories of shock have been elaborated with comparison. Each type of shock has been discussed with its specificities and its management. Septic shock has been extensively discussed and different definitions and terminology have been used. Latest surviving sepsis campaign guidelines have been scribbled down. Hemorrhagic shock has been widely discussed too and a table provided for the differentiation of stages of hemorrhagic shock. Finally, the chapter is concluded with all the references used for accomplishing this chapter.

Keywords: hypovolemia, sepsis, distributive shock, obstructive shock, cardiogenic shock

1. Introduction

Shock is a life-threatening condition that leads to global tissue hypoperfusion and circulatory collapse. It can be reversible if detected, treated, and resuscitated early; otherwise it can cause multi-organ failure and death. Almost one million cases of shock are seen at the emergency department, annually in the USA [1].

Septic shock causes highest rates of mortality (40–60%), compared to other types of shock.

Identification of the cause of shock can be challenging [1].

There is no one specific vital sign that is diagnostic of shock [1].

Bedside ultrasound is a useful tool for diagnosing some types of shock. It can help to evaluate the volume status and cardiac contractility. It can detect tension pneumothorax and cardiac tamponade [2].

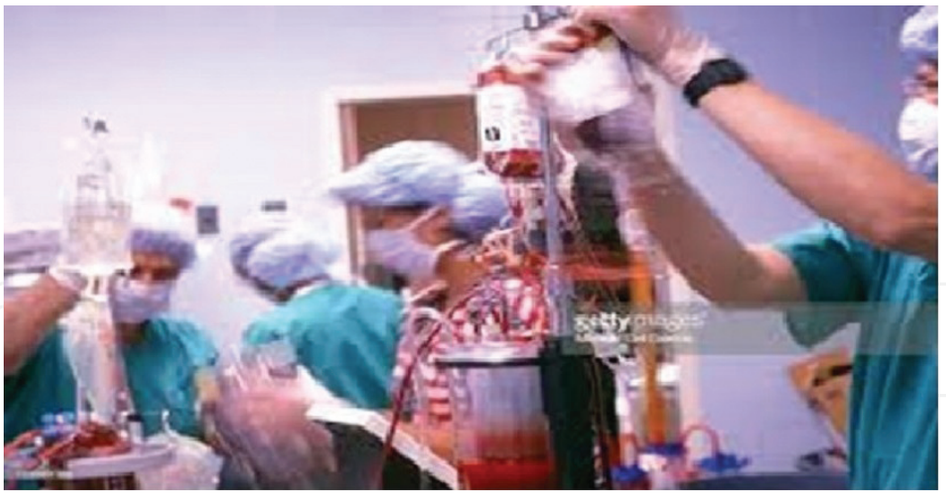

Figure 1. Management of shock [2].

Shock management requires securing airway, controlling breathing, and optimizing circulation to ensure adequate tissue perfusion [2] (**Figure 1**).

2. Pathophysiology

Shock is a state of circulatory insufficiency creating imbalance between oxygen delivery and demand to the tissues, resulting in end-organ dysfunction. At the cellular level, shock first affects the mitochondria. Majority of the aerobic energy comes from combustion of substrates (carbohydrates and fats) along with oxygen, forming carbon dioxide and water. But in shock there is cellular hypoxemia; the tissues enter in anaerobic state and accumulate lactic acid. Lactate starts building up in the blood and acidosis develops [3].

Measurement of serum lactic acid aids in detecting tissue hypoxemia. It is a reliable tool for predicting the outcome and prognosis [4] (**Figure 2**).

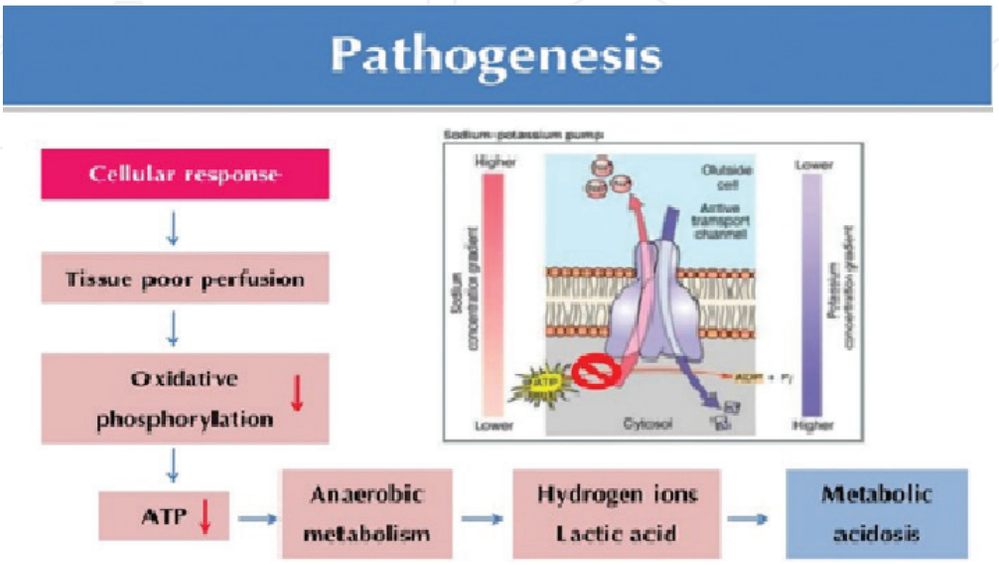

Figure 2. Pathophysiology of shock [2].

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3. Stages of shock

- Pre-shock: compensated phase where patient usually has normal blood pressure.
- Shock: the compensatory mechanism of the body is overwhelmed (almost 20–25% of blood volume is lost).
- End-organ failure: there is irreversible organ damage and death [4].

4. Recognition of shock

After the airway is secured and ventilation is optimized, the circulatory status must be evaluated. Tachycardia and cutaneous vasoconstriction may be early signs of shock. Respiratory rate and pulse pressure (difference between systolic pressure and diastolic pressure) are of significance, too [2].

Hypotension develops in late stages of shock, and its absence does not exclude shock [2].

Serum lactic acid and base deficit are good parameters for detecting the presence and severity of shock [2].

The cause of shock can be determined by taking good history, detailed physical examination, and ordering for necessary investigations (e.g., imaging, bedside ultrasound) [2].

5. Classification of different categories of shock

- Hemorrhagic shock (or hypovolemic shock)
- Non-hemorrhagic shock includes:

  a. Septic shock
  b. Cardiogenic shock
  c. Obstructive shock (tension pneumothorax, pericardial tamponade)
  d. Neurogenic shock
  e. Anaphylactic shock

Sometimes there is a combination or coexistence of more than one type of shock [2].

5.1. Hemorrhagic shock (hypovolemia)

There is loss in intravascular volume which decreases preload and diminishes the cardiac output. Sometimes despite fluid or blood replacement, inotropes and vasopressors might need to be added on [1].

In hemorrhagic shock, due to rapid drop in the blood volume, there is activation of the baroreceptors causing peripheral vasoconstriction and increased cardiac contractility and heart

rate. Initially, as a response to the blood loss, the body tries to compensate by increasing the pulse rate and diastolic pressure, causing the pulse pressure (difference between systolic and diastolic pressure) to narrow. As the volume deficit continues, the cardiac output drops followed by reduction in the blood pressure. Simultaneously there is renal vasoconstriction too, leading to tubular necrosis. There is impaired fuel delivery to all the vital organs including the brain, due to impaired hepatic glucose output and peripheral lipolysis. The most common cause of hemorrhage is trauma [2].

5.2. Management of hemorrhagic shock.

The main treatment is to stop bleeding and restore volume by administering fluids, including blood.

- Establish a secure patent airway and optimize breathing.
- Establish intravenous access for fluid resuscitation.
- Determine the level of conscious (by evaluating the eye, verbal, and best motor response) (Glasgow Coma score, GCS).
- Expose the patient from head to toe to look for injuries or deformities, bearing in mind to avoid hypothermia.

Patient may need to have orogastric tube in place to relieve gastric distension and urinary catheter to monitor output. Patient may need central venous access for measuring central venous pressure, fluid resuscitation, and blood sampling. Imaging studies would aid to the diagnosis of the source of hemorrhage. Initial resuscitation should start with 1–2 L of fluid bolus in adults and 20 ml/kg in pediatric patients. Blood transfusion might be needed too. Balanced resuscitation must be the target in hemorrhagic shock management, which means balancing organ perfusion with risks of rebleeding and accepting lower than normal blood pressure [2].

Target goal of resuscitation is to maintain a urinary output of 0.5 ml/kg/hr. in adults and 1 ml/kg/hr. in pediatric patients [2].

In severe bleeding, uncross-matched blood can be used. Consider O negative blood in women of childbearing age. Vasopressors would usually worsen the hypoperfusion, so it must be considered only if resuscitation by fluids and blood fails. Bedside ultrasound scanning (E-FAST) can help in detecting hemorrhage in intra-abdominal, pleural, pericardial, and pelvic cavities. Knowing the base deficit is important as it can help to distinguish trivial bleeding from significant blood loss. Base deficit is the amount of base needed to be added in 1 L of blood to normalize the pH. Normal base deficit is more positive than −2 mEq/L. It can become negative early in hemorrhage when the pH and blood pressure remain normal [2].

During hemorrhage, tissues suffer academia and accumulation of lactic acid which can be treated if resuscitated promptly and adequately. Treatment is with fluids, blood, and control of hemorrhage [2].

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5.2.1. Stages of hemorrhagic shock

Stage 1: Loss of approximately 750ml of blood, with no significant change in physiological parameters,

Stage 2: loss of 750-1500ml of blood, with mild changes in the vital signs,

Stage 3: loss of 1500ml- 2000ml of blood, affecting the normal body physiology, requiring urgent attention,

Stage 4: loss of >2000ml of blood resulting in severe disturbance of body functions, calling for urgent replacement of blood to restore normal functions.

See **Figure 3**.

5.3. Effects of hemorrhage

There are some factors that can alter the outcome in patients with shock. Factors like:

- Extremes of age.
- Site and severity of injury.
- Time lapse (from injury to resuscitation).
- Comorbidities and medications.

Class	I	II	III	IV
Blood loss (ml)	≤750	750-1500	1500-2000	≥ 2000
Blood loss (% blood volume)	≤15%	15-30%	30-40%	≥40%
Pulse rate	<100	>100	>120	≥ 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Capillary refill test	Normal	Positive	Positive	Positive
Respiratory rate	14-20	20-30	30-40	>35
Urine output (ml/hr)	≥ 30	20-30	5-15	Negligible
CNS-mental status	Slightly anxious	Mildly anxious	Anxious and confused	Confused, lethargic
Fluid replacement (3:1 rule)	Crystalloid	Crystalloid	Crystalloid + Blood	Crystalloid + Blood

Figure 3. Stages of hemorrhagic shock [2].

Hemostasis and balanced fluid therapy (including blood) should be started as soon as signs of hemorrhage are suspected [2].

As mentioned in the table above, patients in classes I and II have good compensatory mechanism to overcome the blood loss and may just need crystalloid infusion, while patients in classes III and IV have lost significant amount of blood and need to have blood transfusion [2].

Soft tissue injuries and fractures compromise the hemodynamics of patients with trauma, e.g., 1500 ml blood can be lost in femur fractures [2].

The cytokines released during tissue injury increase permeability of tissues. Fluid shifts and volume depletion in the intravascular compartment cause hypovolemia [2].

6. Septic shock

It is the most common form of distributive shock. The body's defense system is overwhelmed by infection leading to life-threatening organ dysfunction. In resuscitating septic shock, few effects should be considered: hypovolemia, cardiovascular depression, and systemic inflammation. Besides, there is capillary leak, which causes intravascular volume loss. The combined interaction of chemical mediators, inflammation, and disturbed metabolism causes heart injury during septic shock. There is also capillary leak in the lungs, presenting as acute respiratory distress syndrome (ARDS) [2].

Common causative organisms are pneumococcus, methicillin-resistant *Staphylococcus aureus* (MRSA), *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, etc. [4].

Thus, the early use of antibiotics is advised. Ensuring good oxygenation and ventilation, the use of fluids and vasopressors is the main pillar of treatment in septic shock [1].

The use of parenteral steroids is controversial and can only be considered in patients who are on chronic steroid therapy [1].

Definitions and criteria for septic shock:

Systemic inflammatory response syndrome (SIRS) includes (**Figure 4**):

Two or more of the following:

- Temperature > 38°C or < 36°C.
- Heart rate > 90 beats/min.
- Respiratory rate > 20 breaths/min or $paCO_2$ < 32 mmHg.
- White blood cell count >12,000 or < 4000 or > 10% band neutrophilia [5].

Severe sepsis:

SIRS with suspected infection associated with organ dysfunction [5].

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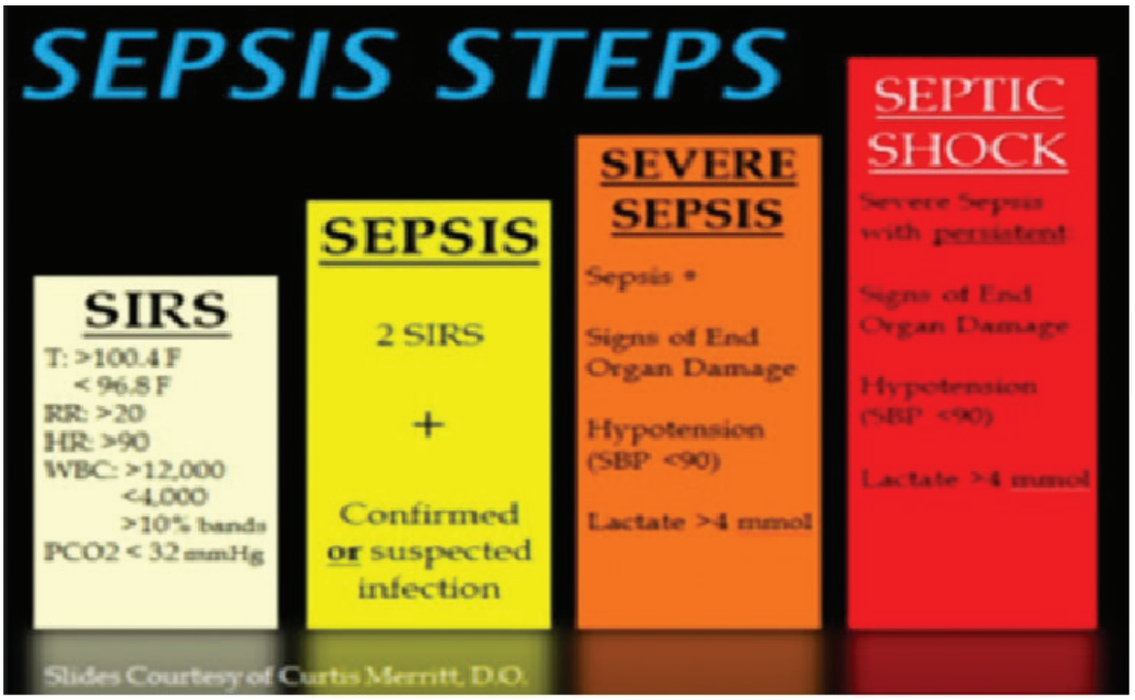

Figure 4. Steps of sepsis [6].

Septic shock:

SIRS with suspected infection and hypotension, despite adequate fluid resuscitation [5].

Surviving Sepsis Campaign has altered the approach to managing sepsis and septic shock, worldwide. It has helped to deliver early timely care to critically ill patients in as less than 6 h [5].

The latest **Surviving Sepsis Campaign Bundles** are as follows:

To be completed 3 h of time of presentation:

1. Measure lactate level.
2. Obtain blood cultures prior to administration of antibiotics.
3. Administer broad spectrum antibiotics.
4. Administer 30 ml/kg crystalloid for hypotension or lactate ≥4 mmol/L [5].

To be completed within 6 h of time of presentation:

5. Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥65 mmHg.
6. In the event of persistent hypotension after initial fluid administration (MAP <65 mm Hg) or if initial lactate was ≥4 mmol/L, reassess volume status, tissue perfusion, and document findings.
7. Remeasure lactate if initial lactate is elevated [5].

Document reassessment of volume status and tissue perfusion with either:

- Repeat focused exam (after initial fluid resuscitation) including vital signs, cardiopulmonary examination, and skin findings.

Or any two of the following:

- Measure CVP.
- Measure $ScvO_2$.
- Bedside cardiovascular ultrasound.
- Dynamic assessment of fluid responsiveness with passive leg raise or fluid challenge [5].

7. Cardiogenic shock

Cardiogenic shock results when more than 40% of the myocardium is damaged by necrosis from ischemia, inflammation, and toxins. There is decreased cardiac output due to pump failure such as cardiomyopathy, myocardial infarction, valvular insufficiency, and arrhythmias. This shock can further persist and eventually lead to cardiac arrest. Usually patients in cardiogenic shock look ill, drowsy, sweaty, and pale and can have tachycardia with weak pulse and hypotensive. The urine output would be deceased to less than 0.5 ml/kg/h, and serum lactic acid would be as high as 4 mmol/L, indicating circulatory insufficiency. Left ventricular dysfunction can be detected by echo early in the course of cardiogenic shock. Patients with severe left ventricle dysfunction are more liable to develop shock than those with mild to moderate dysfunction. Serial cardiac markers and bedside echo for such cases are worth doing as they can aid in diagnosis and effective management. Patient needs to be monitored closely and vital signs recorded frequently. It is worth to have an arterial line in place for accurate blood pressure readings. Monitoring urine output, base deficit, and serum lactic acid is important for the assessment of resuscitation in all patients who are in shock [4] (**Figure 5**).

Management of cardiogenic shock includes:

1. Improve the work of breathing by adequate oxygenation and ventilation.
2. Initiation of vasopressors or inotropes, e.g., norepinephrine (0.5 μg/min or dobutamine (5μg/kg/min).
3. Treating arrhythmias.
4. Aspirin and heparin (if indicated).
5. Treatment of the cause, e.g., thrombolysis or angioplasty (e.g., myocardial infarction). In refractory cases of cardiogenic shock, intra-aortic balloon pump can be used.

Emergency reperfusion procedure (thrombolysis/PTCA) is not superior to medical management in cardiogenic shock, secondary to myocardial infarction. There is no reduction in the mortality rate [1].

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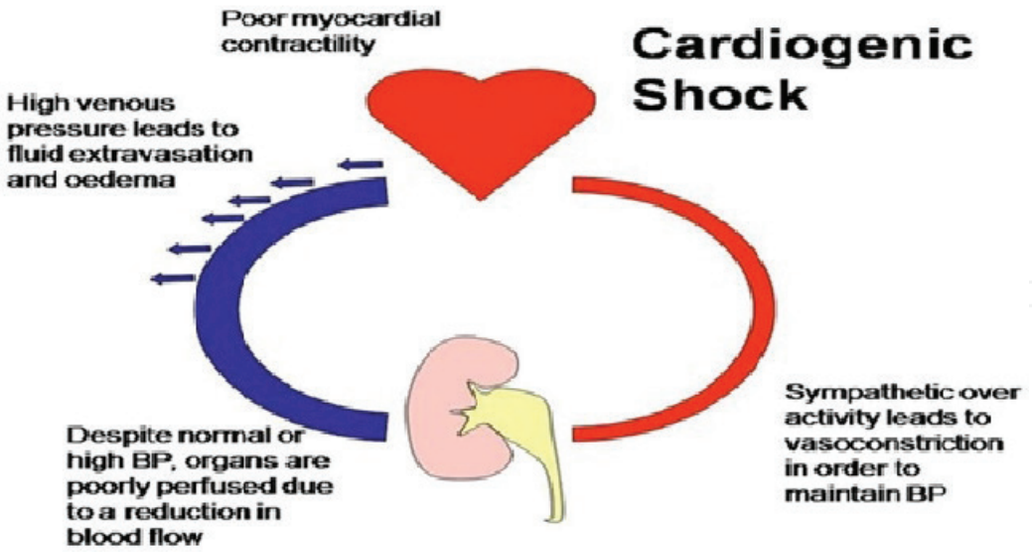

Figure 5. Pathophysiology of cardiogenic shock [7].

8. Obstructive shock

It is usually due to extra cardiac etiologies which result in poor right ventricle output. Causes are:

- Pulmonary: right ventricular failure from pulmonary embolism or severe pulmonary hypertension, as the heart cannot generate enough pressure to overcome the high pulmonary vascular resistance.
- Mechanical: there is a reduction in venous return to the right atrium and inadequate right ventricle filling. Causes: tension pneumothorax, tamponade, constrictive pericarditis, and restrictive cardiomyopathy [4].

Bedside ultrasound would be of absolute benefit in diagnosing obstructive shock [2].

9. Neurogenic shock

It is characterized by hypotension due to severe brainstem or spinal cord injury resulting in autonomic system disruption. Trauma to the cervical or upper thoracic spine leads to sympathetic chain injury resulting in vasodilation.

Good fluid resuscitation and vasopressors would help to manage this type of shock [1].

10. Anaphylactic shock

Immunoglobulin E mediated response due to insect stings, food, and drugs. Cardinal feature is circulatory collapse associated with bronchospasm and increase airway resistance. It can be associated with skin manifestations of wheals and hyperemia. There can also be vomiting and

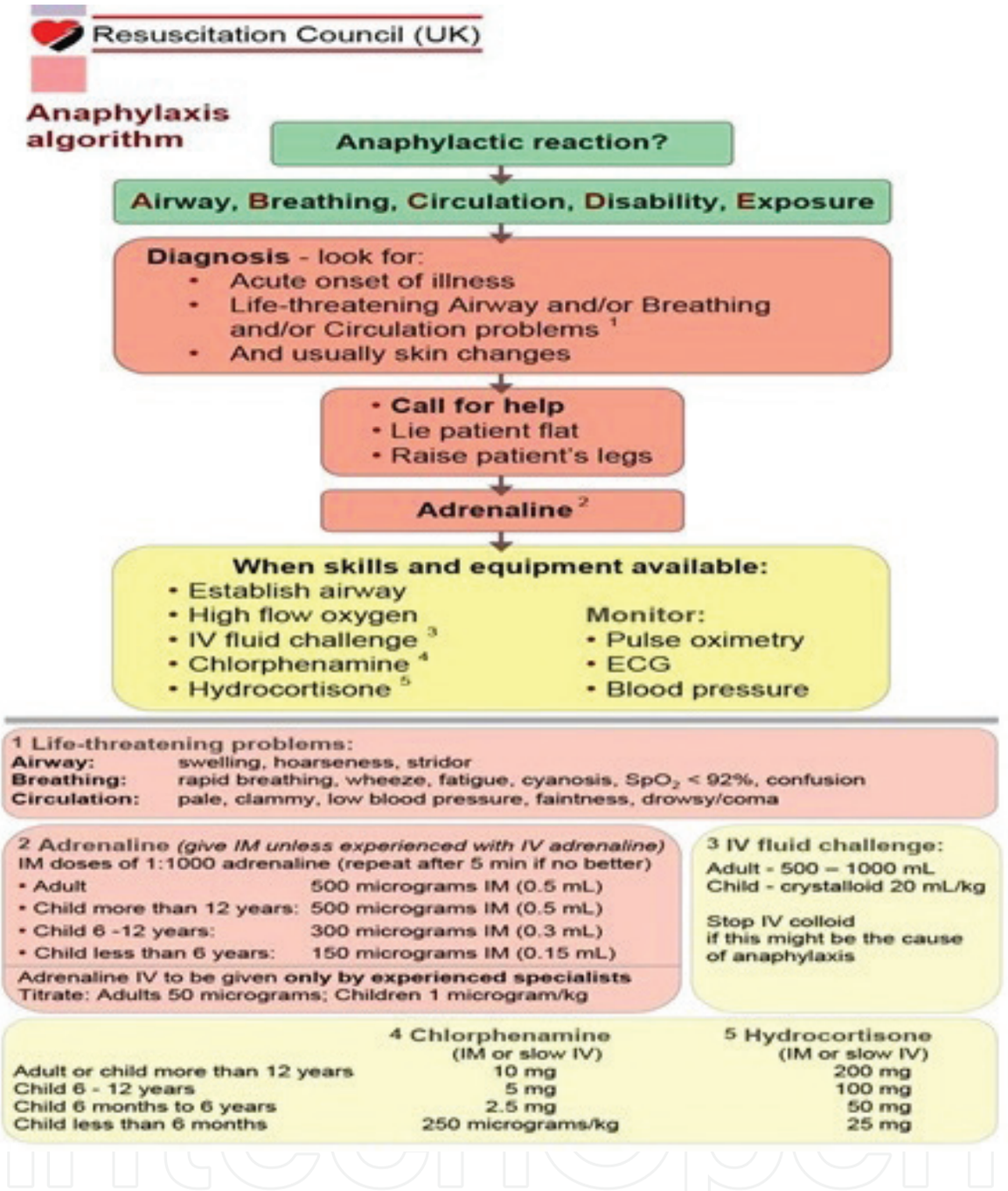

Figure 6. Anaphylaxis management algorithm [8].

diarrhea, too. Management includes stabilizing the patient; maintaining the airway patency; effective breathing; prompt use of epinephrine, fluid resuscitation, parenteral steroids, and antihistamine; and the use of bronchodilators, if necessary [3] (**Figure 6**).

11. Special considerations

11.1. Advanced age

Trauma in elderly patients needs special attention as cardiac compliance decreases with age. The heart cannot compensate for blood loss as efficiently as in youngsters. There is a decrease

in catecholamine production resulting in manifestation of tachycardia, when in shock. Elderly patients are mostly on multiple medications. Beta blockers worsen hypotension caused by trauma and also mask the tachycardia, which is an early sign of shock. Volume resuscitation must be strictly monitored as most patients have decreased cardiac contractility and can easily go into volume overload [2].

11.2. Athletes

They may not manifest any signs of shock due to their unusual compensatory mechanism, despite significant amount of blood loss [2].

11.3. Pregnancy

Pregnant patients would present with signs of shock only after huge volume of blood is lost, due to their physiological maternal hypervolemia [2].

12. Conclusion

Shock is a state of global tissue hypoperfusion. After initial resuscitation, detailed physical examination is important to determine the cause of shock. Patients in shock have to be kept on monitored bed. Urine output and central venous pressure would need to be monitored in such patients [2].

In trauma patients, hypovolemia is the main reason for shock. Control of hemorrhage and blood replacement are necessary. Hypovolemia can also develop due to gastroenteritis, heat stroke, febrile illness, etc. Septic shock needs early administration of antibiotics, after drawing a full septic workup. Cardiogenic shock needs to be treated meticulously and monitored closely. Inotropes and fluids have to be administered, cautiously. Neurogenic shock needs good vasopressor support. Obstructive shock whether it is tension pneumothorax or pericardial tamponade, both, need decompression. Pulmonary embolism needs to be treated with anticoagulants. Anaphylactic shock can be managed by administration of parenteral epinephrine, crystalloids, steroids, and antihistamines [3].

Management of shock is often complicated especially in extremes of age, pregnancy, and patients with multiple comorbidities [2].

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