Inclusive Educational Content

ADVANCED LIFE SUPPORT

SaveLife: Reorganizing Basic/Advanced Life Support Training Through the Use of Innovative Digital Materials

Erasmus+ Project
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SaveLife

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TURKISH NATIONAL AGENCY

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"SaveLife: Reorganizing Basic/Advanced Life Support Training Through the Use of Innovative Digital Materials"

SaveLife Project

The “Save Life: Reorganizing Basic/Advanced Life Support Training Through the Use of Innovative Digital Materials” (SaveLife) project is a pioneering initiative funded by the European Union within the Erasmus+ KA220 program. This project aims to revolutionize the training methods for Basic and Advanced Life Support (B/ALS) by providing medical staff with up-to-date, open, and free access to high-quality B/ALS training materials. These materials can be used as booster training or as a complement to in situ training when deemed necessary by trainers.

Basic and Advanced Life Support training is essential for medical professionals to effectively handle cardiac arrest situations. BLS training is typically introduced early in medical education and is further developed through ALS training. This knowledge is crucial for physicians to deal with cardiac emergencies, and the SaveLife Project aims to ensure that these skills are kept current and accessible.

The SaveLife Project primarily targets university staff, medical students, nurses, paramedics, and general practitioners who work or will work in high-risk areas. By focusing on these groups, the project aims to make a significant and lasting impact on B/ALS training across Europe.

The SaveLife Project stands out due to its innovative approach: Interactive Digital Tools, Increased Accessibility, Diverse Consortium.

Chapter 1. Respiratory Arrest Due to Anaphylaxis

Nataliia Pavliukovych, Oleksii Godovanets

Chapter 1. Respiratory Arrest Due to Anaphylaxis

Anaphylaxis is a critical and sometimes fatal allergic reaction that manifests swiftly and can impact several bodily systems. It is marked by an abrupt onset, swift progression, and generally encompasses symptoms associated with the airway, respiration, and circulation. These symptoms frequently occur alongside dermatological or mucosal alterations, including erythema, urticaria, or angioedema.

Anaphylaxis is a critical and sometimes fatal allergic reaction that manifests swiftly.

Common triggers of anaphylaxis encompass different allergens, including specific foods (e.g., peanuts, milk, shellfish), pharmaceuticals (e.g., antibiotics, NSAIDs), and insect bites. The fundamental mechanism is frequently IgE-mediated, indicating that allergen exposure initiates a series of immunological responses including mast cells and basophils. This results in the production of inflammatory mediators such as histamine, which induces tissue edema, smooth muscle contraction (resulting in bronchoconstriction and respiratory distress), and may lead to cardiovascular collapse.

Timely recognition and treatment of anaphylaxis is essential. The "ABCDE" method is advised for evaluation:

Assess the airway for any obstructions or edema.

Evaluate the respiratory rate and effort, and auscultate for wheezing or stridor.

Circulation: Assess pulse rate, blood pressure, and capillary refill to evaluate circulatory status.

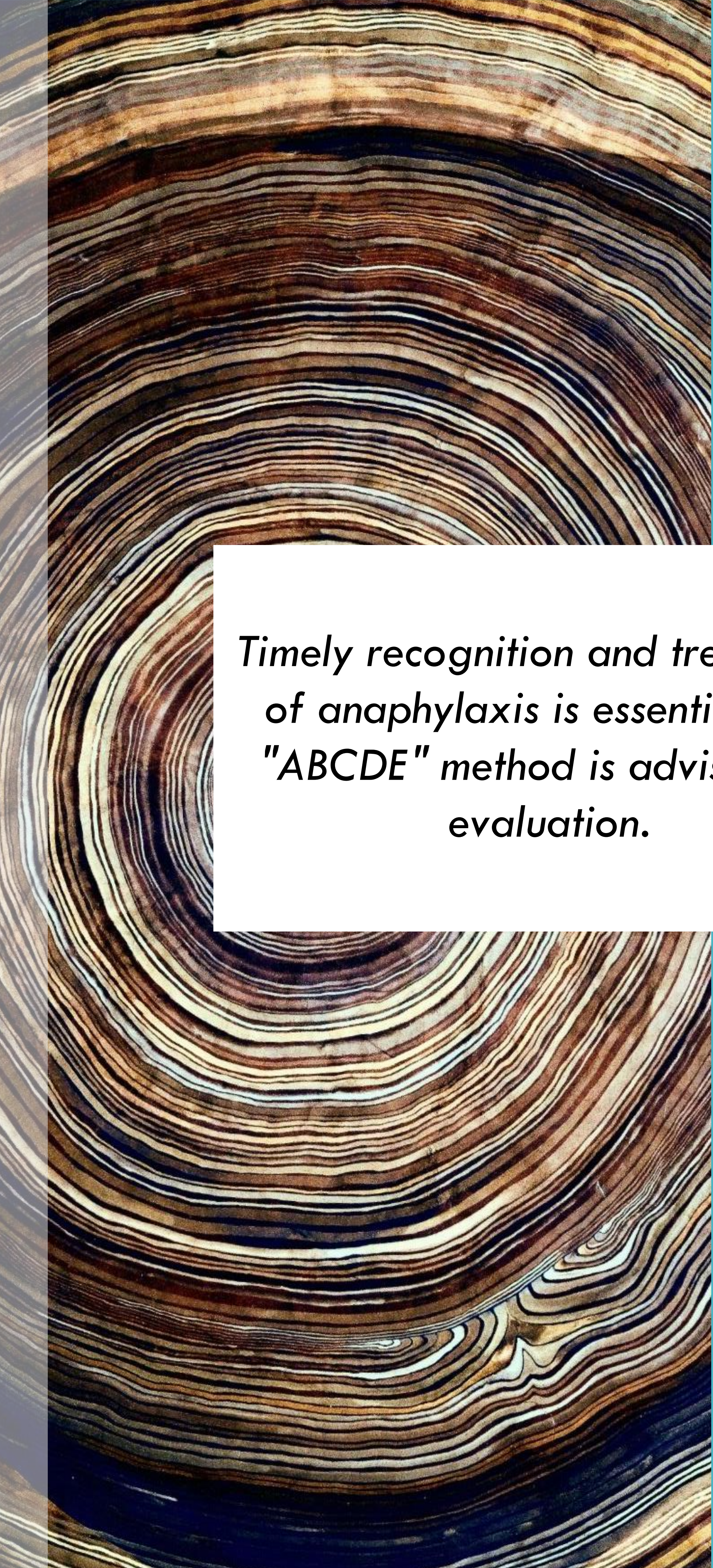
Disability: Assess the degree of consciousness and neurological function.

Assessment: Inspect the skin for indications of allergic reactions such as urticaria, erythema, or angioedema.

The symptoms of anaphylaxis can differ significantly, from minor discomfort to potentially fatal circumstances. These may encompass:

- **Airway**: Edema of the tongue and pharynx, dysphagia, dysphonia.
- **Respiration**: Dyspnea, wheezing, coughing, tachypnea.
- **Circulation**: Diminished or accelerated pulse, hypotension, vertigo, syncope, and in extreme instances, cardiac arrest.
- **Dermatological manifestations**: Urticaria, pruritus, erythema.
- **Gastrointestinal**: Nausea, emesis, abdominal spasms, diarrhea.

It is crucial to distinguish anaphylaxis from other illnesses that may replicate its symptoms, like asthma attacks, panic attacks, or vasovagal syncope.



Timely recognition and treatment of anaphylaxis is essential. The "ABCDE" method is advised for evaluation.

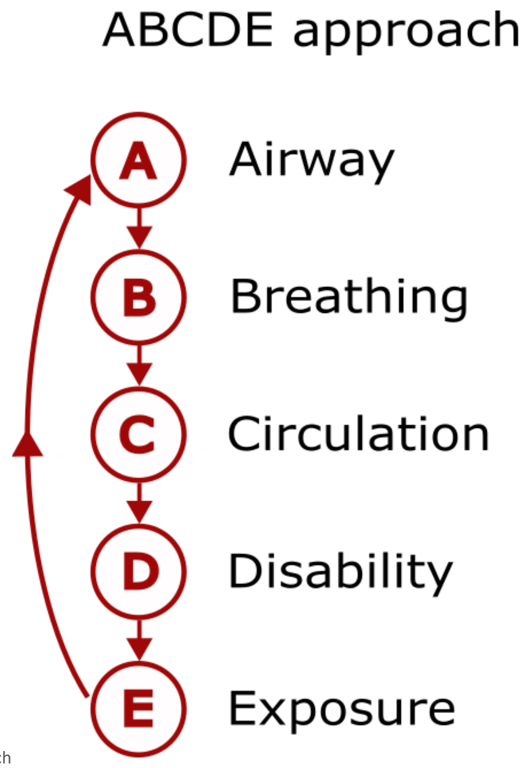


Figure. The ABCDE approach

The management of anaphylaxis necessitates prompt intervention. Adrenaline (epinephrine) is the primary treatment and should be injected intramuscularly, typically in the mid-outer thigh. Appropriate patient posture is essential. Individuals with hypotension should assume a supine position with elevated legs, but those encountering respiratory distress may choose a semi-recumbent posture. If the trigger is identifiable and can be safely extracted (for instance, a stinger), it should be executed without delay. Ongoing surveillance of vital signs, encompassing oxygen saturation, blood pressure, and heart rate, is essential to evaluate the patient's reaction to treatment.

In instances of respiratory arrest, supplementary measures such as oxygen delivery, airway stabilization using techniques like the head tilt-chin lift maneuver, and ventilatory assistance may be necessary. Artificial airways and suctioning may be required to ensure airway patency.

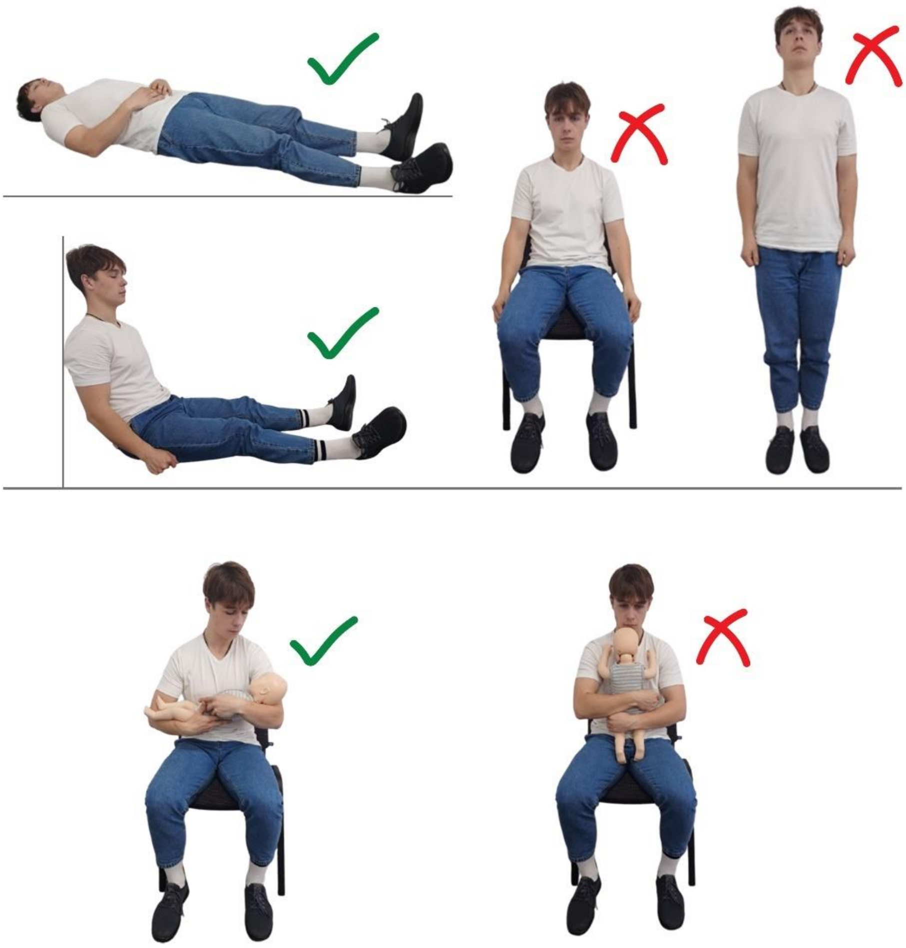
Adrenaline is fundamental to anaphylaxis treatment because it activates both alpha and beta-adrenergic receptors. This intervention mitigates the consequences of anaphylaxis by elevating blood pressure, diminishing angioedema, and facilitating bronchodilation. In critical instances, intramuscular adrenaline may be injected at 5-minute intervals until symptom alleviation occurs.

Adrenaline is fundamental to anaphylaxis treatment because it activates both alpha and beta-adrenergic receptors.

Antihistamines may reduce cutaneous symptoms, and corticosteroids can manage severe or prolonged reactions; nonetheless, these are adjuvant therapies and should not postpone the delivery of adrenaline.

Figure. Adrenaline administration in case of anaphylaxis

Figure. The key steps for the initial treatment of anaphylaxis



Healthcare personnel must possess a comprehensive grasp of the signs and symptoms, swift recognition, and timely management of anaphylaxis to enhance patient outcomes. The guidelines underscore the necessity of prompt adrenaline delivery, suitable patient placement, and airway treatment to rapidly stabilize the patient.

Anaphylaxis is a critical and sometimes fatal allergic reaction that manifests swiftly.

Chapter 2.
Management of STEMI

Gergana Ivanova

Chapter 2. Management of STEMI

Chest pain and perhaps signs of permanent heart damage can be symptoms of acute coronary syndromes (ACS), a group of conditions caused by a sudden reduction in blood flow to the heart muscle. Reduced blood supply to the heart, known as myocardial ischemia, occurs when the coronary arteries are narrowed or blocked. Myocardial infarction with ST-segment elevation (STEMI) is the most severe form of acute coronary syndrome (ACS) and is characterized by a complete blockage of a coronary artery. A myocardial infarction occurs when this blockage prevents oxygen from reaching a section of the heart muscle.

Patients suffering with acute coronary syndrome often report severe chest discomfort, which can be described as a tightening, burning, or constriction of the chest muscles. Symptoms may radiate to other parts of the body, such as the jaw, neck, back, shoulders, and arms (particularly the left arm). Lightheadedness, nausea, vomiting, dyspnea, diaphoresis, or a sense of impending doom are some of the associated symptoms.

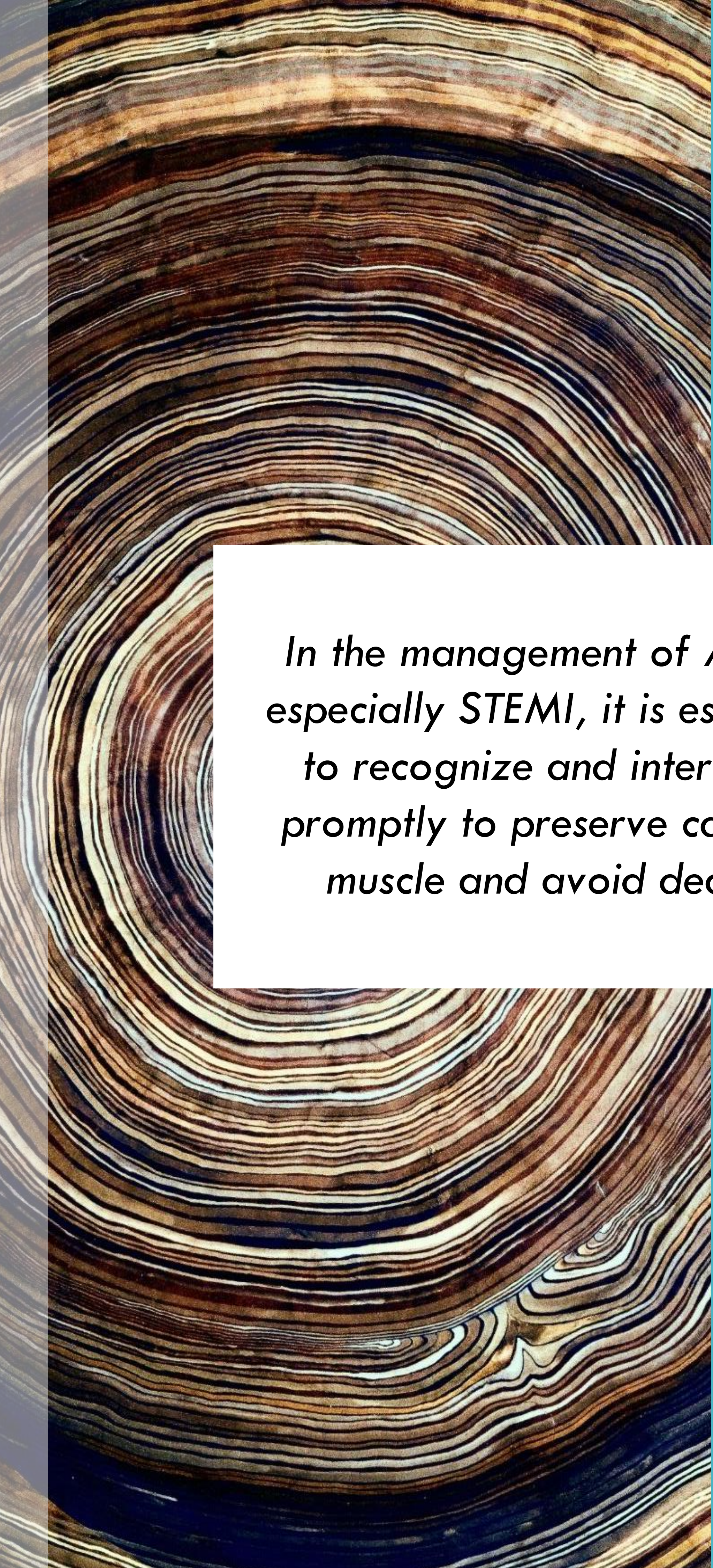
In the management of ACS, especially STEMI, it is essential to recognize and intervene promptly to preserve cardiac muscle and avoid death. As part of the initial assessment, the patient's symptoms are carefully reviewed, with the use of the **OPQRST** memory aid, in order to identify the specific type of chest pain.

- How long has it been since you first felt the pain? **O**nset
- How did you become hurt in the first place? What were you doing when it started? **P**rovocation and **P**alliation
- Identify the type of pain you're experiencing. **Q**uality
- Does the discomfort spread to other areas? **R**adiation
- In what exact spot does the pain originate? **S**ite
- To what extent has the pain been there for a certain amount of time? **T**ime course

A 12-lead electrocardiogram (ECG) is crucial and must be acquired within 10 minutes of the patient's arrival. The ECG assists in detecting specific alterations, such as ST-segment elevation, which signify a STEMI.

The foundation of STEMI management is prompt reperfusion therapy to reestablish blood flow to the compromised coronary artery. Percutaneous coronary intervention (PCI) is the favored reperfusion technique that entails the insertion of a catheter equipped with a balloon tip into the occluded artery. The balloon is inflated to dilate the artery, and a stent (a tiny mesh tube) is frequently inserted to maintain arterial patency. PCI is most efficacious when conducted within 12 hours of symptom onset, and optimally within 90 minutes for patients with STEMI.

Myocardial infarction with ST-segment elevation (STEMI) is the most severe form of acute coronary syndrome (ACS).



In the management of ACS, especially STEMI, it is essential to recognize and intervene promptly to preserve cardiac muscle and avoid death.

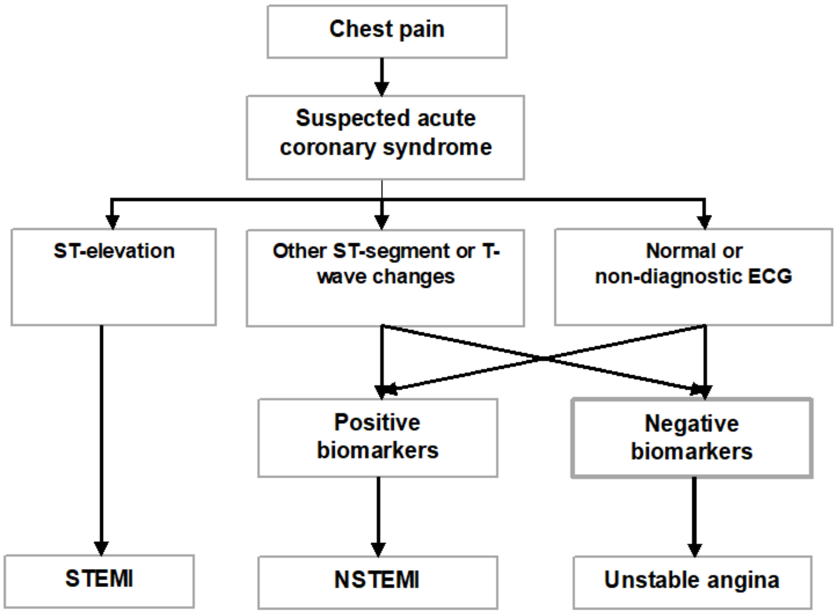


Figure. 12-Lead ECG differential diagnosis of ACS

When early percutaneous coronary intervention is impractical, fibrinolytic therapy, commonly referred to as thrombolytic medicine administration, serves as an alternate reperfusion method. Fibrinolytics function by lysing the thrombus blocking the coronary artery. This medication is especially beneficial in cases where PCI cannot be executed within 120 minutes of diagnosis and is most efficacious when given within 12 hours of symptom onset.

The care of ACS and STEMI requires a combination of reperfusion therapy and other adjuvant therapies. To prevent future thrombus formation, aspirin is given as an antiplatelet medication. By widening the coronary arteries, nitroglycerin is given to patients suffering from angina. When a patient's oxygen saturation drops below 90%, they are given oxygen therapy. The use of anticoagulant and antiplatelet drugs helps to maintain blood circulation and reduces the likelihood of blood clots. Severe pain is treated with painkillers such as Morphine 2 mg I.V. with attention to breathing and consciousness.

The care of ACS and STEMI requires a combination of reperfusion therapy and other adjuvant therapies.

Patients with STEMI face an increased risk of life-threatening consequences, such as cardiac arrest. In the event of cardiac arrest, high-quality cardiopulmonary resuscitation (CPR) and immediate defibrillation are crucial. Defibrillation is essential for shockable arrhythmias such as ventricular fibrillation, wherein an electrical shock is administered to reinstate a normal cardiac beat. Cardiopulmonary resuscitation must be conducted with chest compressions at a frequency of 100-120 compressions per minute, and it is essential to minimize interruptions in compressions to ensure sufficient blood circulation. In the absence of spontaneous circulation, advanced life support (ALS) protocols are implemented, encompassing continuous vital sign monitoring, sophisticated airway management, and intravenous pharmacotherapy to enhance heart function.

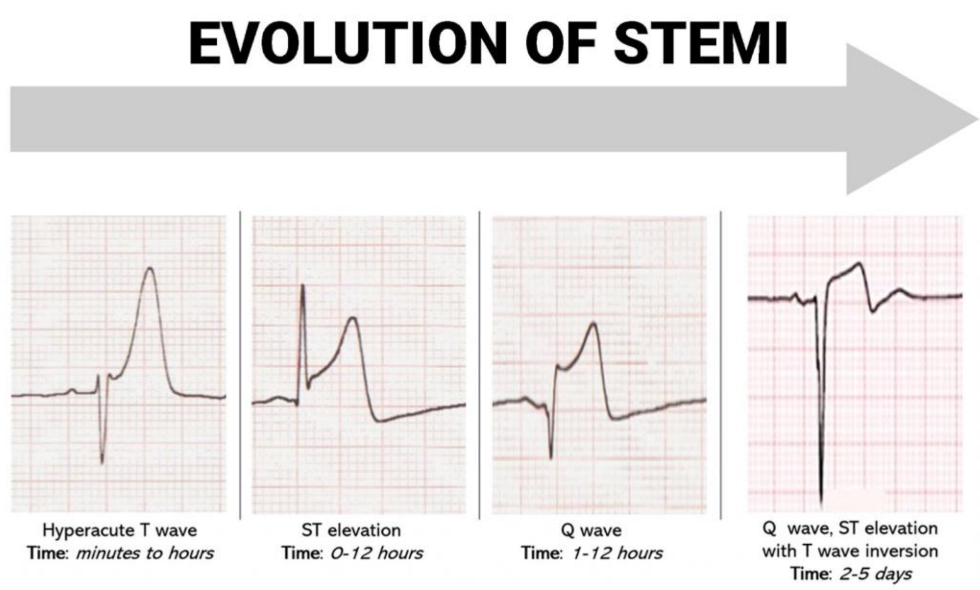


Figure. High-probability ECG features of MI

Figure. mCPR device

Subsequent to the initial stabilization, continuous monitoring and care are required. This encompasses serial ECGs to evaluate the progression of ischemia and infarction, together with cardiac biomarkers, such as troponin, to verify myocardial damage. Long-term management frequently necessitates pharmacological interventions to regulate risk factors such as hypercholesterolemia, hypertension, and diabetes mellitus. Lifestyle adjustments, such as smoking cessation, adherence to a heart-healthy diet, frequent physical activity, and effective stress management, are essential for reducing recurring occurrences and enhancing overall cardiovascular health.

The primary objective in the management of acute coronary syndromes, including STEMI, is prompt diagnosis, immediate reperfusion therapy to reestablish blood flow, and thorough supportive care to enhance patient outcomes and reduce sequelae.

Myocardial infarction with ST-segment elevation (STEMI) is the most severe form of acute coronary syndrome (ACS).

Chapter 3.
Targeted Temperature Management

Margarita Petrova Atanasova

Chapter 3. Targeted Temperature Management

Achieving restoration of spontaneous circulation after it has abruptly stopped is simply the initial step. There is a risk of permanent harm to their brain and other vital organs due to the lack of oxygen. One method that has been found to aid in these patients' recoveries is Targeted Temperature Management (TTM). It entails gradually rewarming the body after cooling it to a certain temperature. Controlled hypothermia shields the brain and other organs from potential harm due to hypoxia during cardiac arrest.

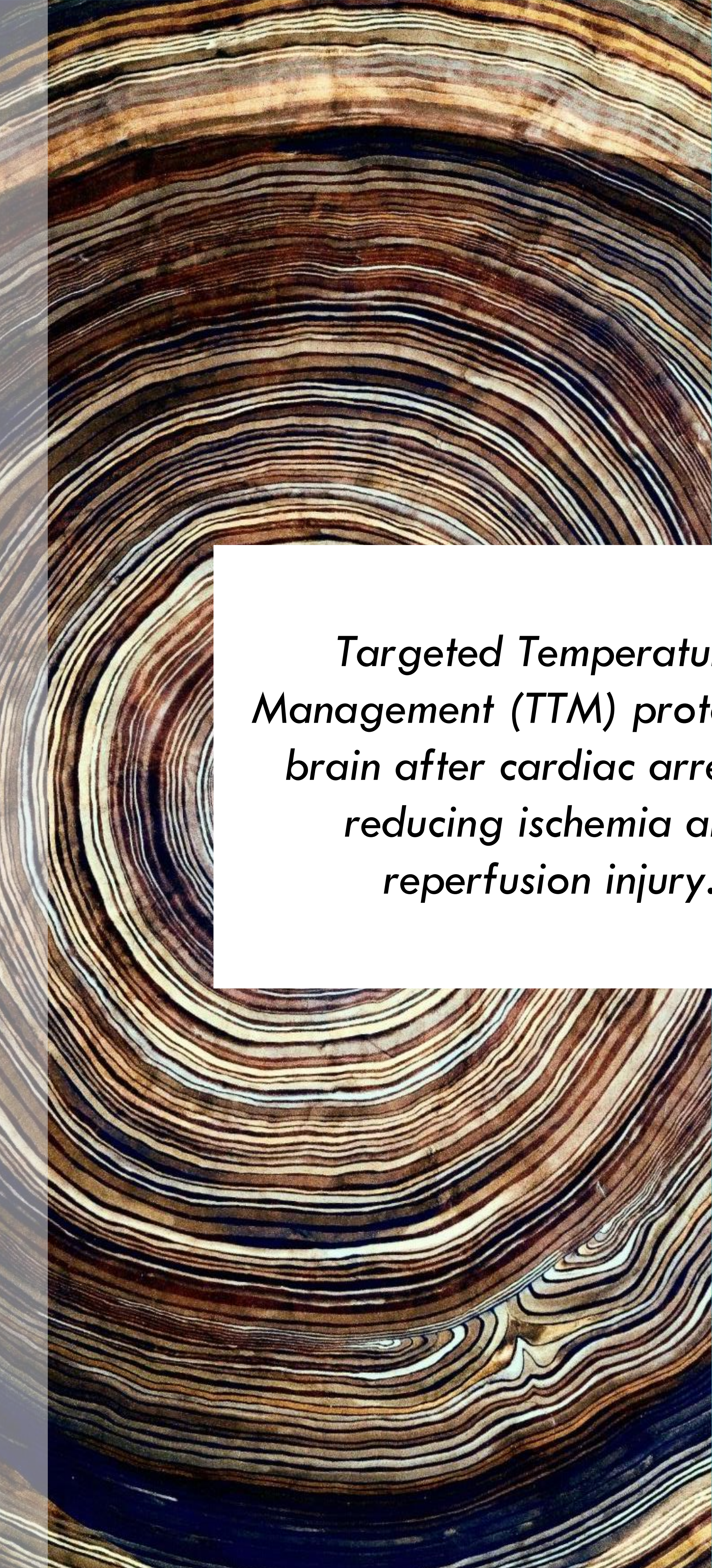
What Makes TTM Significant?

Targeted Temperature Management (TTM) protects the brain after cardiac arrest by reducing ischemia and reperfusion injury. It lowers metabolic demands of the heart, aids oxygen conservation, and suppresses excitotoxicity from excess neurotransmitters. TTM also reduces inflammation and oxidative stress by limiting cytokine and free radical production, preserves the blood-brain barrier, and ultimately improves survival and neurological outcomes.

How Does TTM Help People?

After a cardiac arrest, adults who are still unconscious but have been resuscitated can be given TTM. It works best when administered as soon as the heart begins to beat again. Post-resuscitation care is crucial for patients after cardiac arrest. The European Resuscitation Council (ERC) recommends therapeutic temperature management (TTM) to reduce neurological damage and improve outcomes, particularly in comatose survivors. TTM may be reconsidered if a patient rapidly regains consciousness after ROSC or remains hemodynamically unstable. It may also be unsuitable in cases of severe coagulopathy, non-cardiac arrest coma (e.g., drug overdose, stroke), terminal illness, poor prognosis, or uncontrolled infections.

One method that has been found to aid patients' recoveries is Targeted Temperature Management (TTM).



Targeted Temperature Management (TTM) protects the brain after cardiac arrest by reducing ischemia and reperfusion injury.

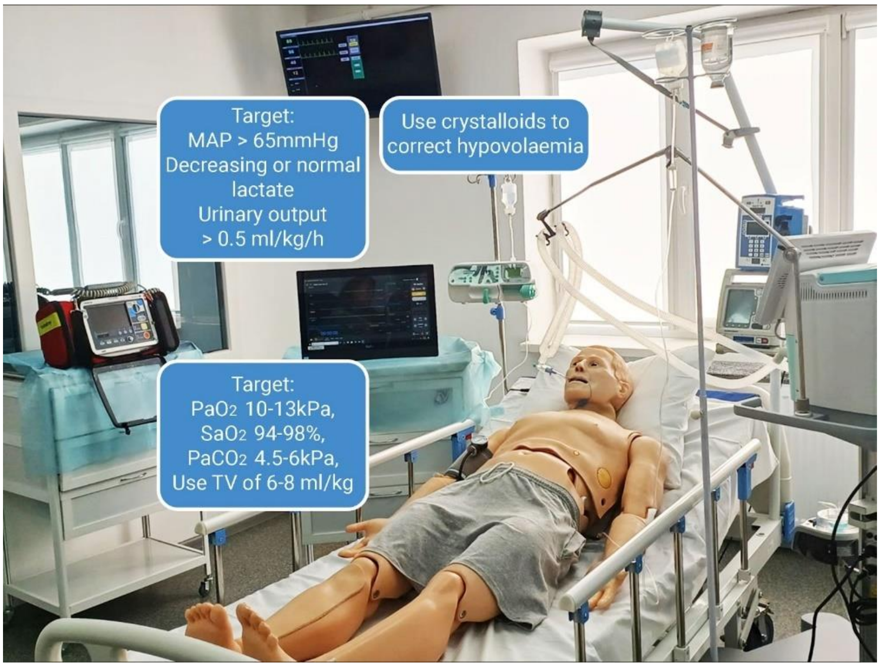


Figure. Oxygenation, ventilation and hemodynamic support

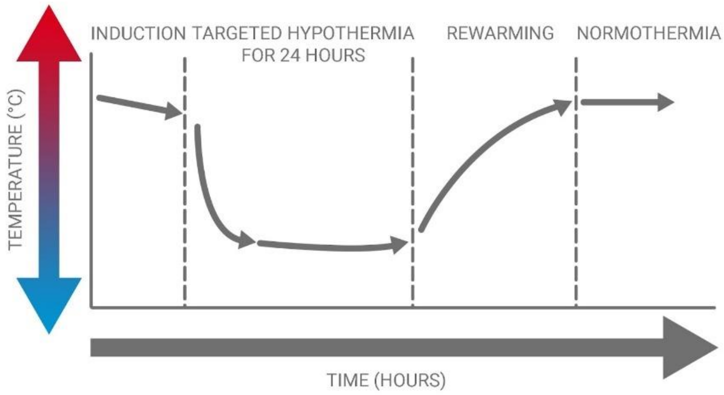
How Does TTM Operate?

Therapeutic Temperature Management (TTM) involves lowering core body temperature to 32°C–36°C after ROSC in comatose adults, ideally within 60 minutes. TTM is maintained for at least 24 hours, followed by gradual rewarming (0.25°C to 0.5°C per hour). Continuous monitoring ensures temperature consistency, and fever prevention is key due to its link with poor outcomes. Hypothermia is induced through surface or intravascular cooling methods, though prehospital cold IV fluids are not recommended. Rebound hyperthermia post-TTM increases risk. Close monitoring during rewarming is critical to avoid complications.

What Dangers Could Occur?

Shivering during TTM raises metabolic demand, counteracting its benefits. Bradycardia and hypotension may occur due to cooling, while arrhythmia can arise, requiring continuous monitoring. Electrolyte imbalances, particularly in potassium and magnesium, are common and must be closely watched. Infections and coagulopathies are risks due to cooling's effects on immunity and coagulation. Rebound hyperthermia during rewarming may reverse neuroprotection, and TTM can alter drug metabolism, necessitating dose adjustments to maintain therapeutic efficacy.

Figure. Targeted temperature management



What Follows Next?

The prognosis for a patient's recovery following a cardiac arrest remains uncertain, even when using TTM. To determine the severity of brain damage, doctors conduct a battery of procedures, including imaging scans and neurological examinations. Recovery from TTM can be difficult and time-consuming, but it is worth it to increase the likelihood of survival and a positive neurological outcome.

Essentially, TTM is a sophisticated technique that is essential for the recovery of individuals who have experienced a cardiac arrest. It provides an opportunity to lessen harm and increase the likelihood of a substantial recovery by regulating core body temperature.

One method that has been found to aid patients' recoveries is Targeted Temperature Management (TTM).

Chapter 4.

Management of Stable and Unstable SVT in Pediatric Patients

Nataliia Bogutska

Chapter 4. Management of Stable and Unstable SVT in Pediatric Patients

A frequent cardiac rhythm issue in children, supraventricular tachycardia (SVT) causes the heart to beat excessively fast. The atria, the upper chambers of the heart, are the sites of origin and abrupt cessation of this rapid heart rate. Although SVT episodes usually do not pose a life-threatening threat, they can still cause significant distress and, if not addressed, can lead to serious problems.

Investigating the Mechanisms of SVT in Pediatric Patients

Electrical impulses traverse the heart, setting in motion the coordinated contraction of its muscles, which in turn regulates the heart's rhythm. When SVT occurs, the heart's electrical signals are interrupted, causing the heartbeat to be fast and irregular. The re-entry phenomenon, in which the electrical signals become stuck in a loop and persistently stimulate the heart to beat faster than normal, is the most prevalent cause of SVT in children.

In re-entry SVT, there are primarily two varieties:

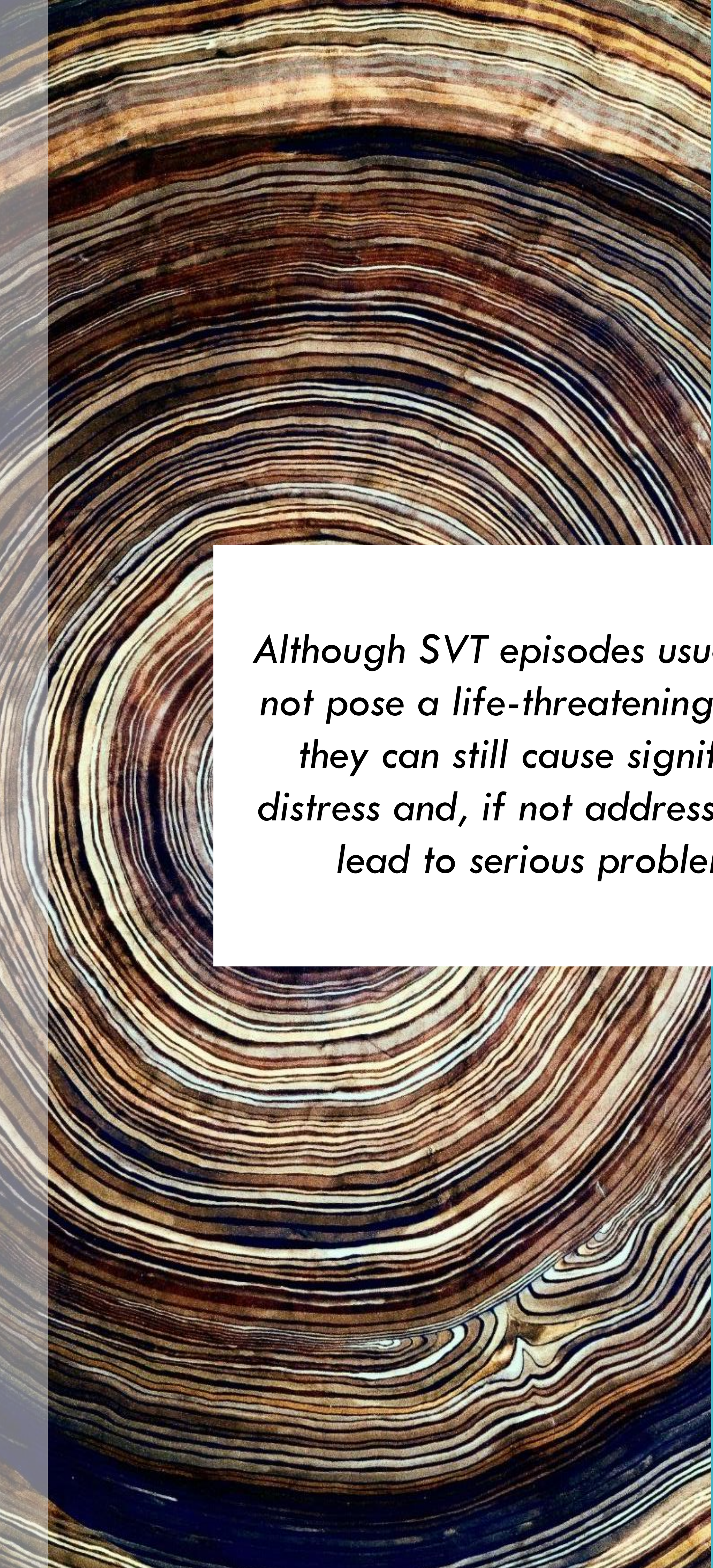
- The first kind is atrioventricular re-entry tachycardia (AVRT), which is characterized by the presence of an additional cardiac electrical circuit and is seen in conditions like Wolff-Parkinson-White syndrome. Infants are more likely to experience AVRT.
- The second kind, known as atrioventricular nodal re-entry tachycardia (AVNRT), affects the normal electrical link between the atrium and ventricles. Older kids and teens are more likely to have AVNRT.

Identifying the Warning Signals

Depending on the child's age and the intensity of the event, the symptoms of SVT can differ. There are a number of mild symptoms that infants may have, including pale complexion, irritability, lethargy, poor feeding, vomiting, and cyanosis. In addition to these symptoms, they may exhibit tachypnea, an enlarged liver, and profuse sweating, all of which are indicators of heart failure.

Palpitations, a feeling of a beating heart, pressure in the neck or chest, lethargy, vertigo, and difficulty breathing are all possible manifestations in older children and teenagers. Fainting (syncope) is a symptom that may indicate an elevated risk of abrupt cardiac arrest in extreme situations.

A frequent cardiac rhythm issue in children, supraventricular tachycardia (SVT) causes the heart to beat excessively fast.



Although SVT episodes usually do not pose a life-threatening threat, they can still cause significant distress and, if not addressed, can lead to serious problems.

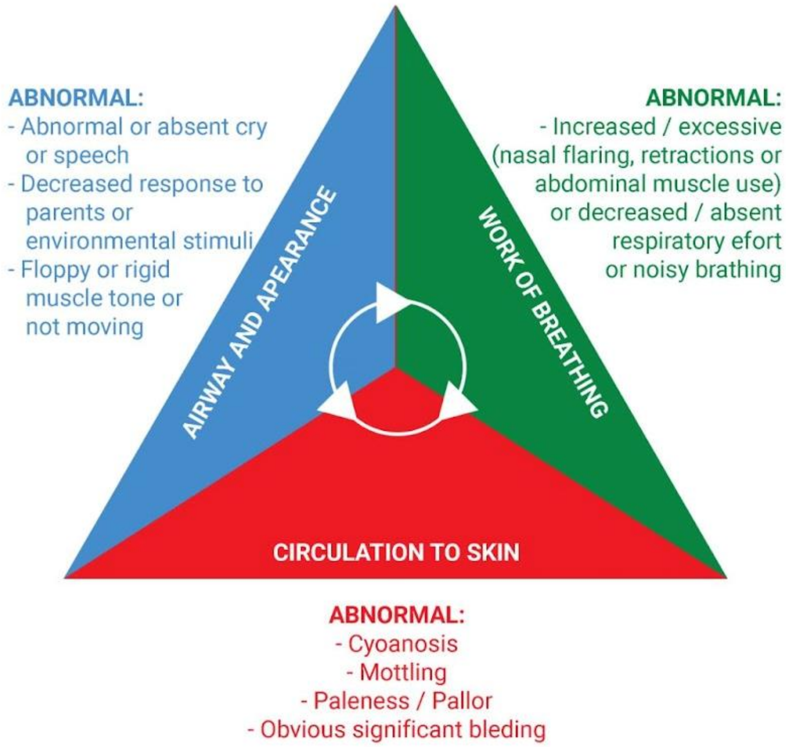


Figure. Paediatric Assessment Triangle

Timely Detection is Critical

Since a fast heart rate might impair the heart's capacity to efficiently deliver blood to the body, early detection of SVT is vital. Complications including heart failure or cardiac muscle injury (cardiomyopathy) may result from the decreased oxygen delivery to the organs and tissues caused by the lower blood flow.

An electrocardiogram (ECG), which captures the heart's electrical activity, is used to diagnose SVT by doctors. In SVT, the electrocardiogram (ECG) usually reveals a regular, fast heart rate and a narrow QRS complex, which is a unique pattern of electrical activity. Distinguishing SVT from sinus tachycardia, a natural rise in heart rate brought on by things like exertion, fever, or stress, is crucial. The heart rate changes depending on the child's degree of activity, and sinus tachycardia typically has a slow start and end.

Pediatric SVT Treatment Alternatives

The severity of a child's SVT episode and their general health determine the course of treatment. The use of vagal procedures to reduce the heart rate is an option for clinicians when the infant is stable, with good blood pressure and no indications of distress. These moves work by activating the vagus nerve, which aids in controlling the heart rate. The Valsalva technique, in which an older child bears down as if having a bowel movement, or the application of a cold stimulus to an infant's face are both examples of vagal manoeuvres.

Adenosine is frequently used as a first-line treatment after vagal techniques have failed. The fast heart rate can be temporarily alleviated by administering adenosine intravenously (IV), which blocks the electrical signals responsible for the condition. Because of this break, the heart is able to return to its regular rhythm.

If a child is showing symptoms of poor perfusion, such as low blood pressure, changed mental status, or cardiac failure, synchronized cardioversion treatment must be administered immediately. A regulated electrical shock is administered to the heart as part of this operation to restore the rhythm.

Figure. Vagal manoeuvre with application of a bag filled with ice and cold water over the child's face

Persistent Care and Supervision

Children may need continuous monitoring and follow-up treatment following an episode of SVT. Medication to reduce the likelihood of future attacks or more testing to diagnose cardiac problems may be part of the plan. When treated properly and followed up with care, children with SVT can go on to lead active, normal lives.

Caregivers and parents should know the symptoms of SVT and know how to get their child to the hospital quickly if they think he or she is having an episode. The prognosis for children suffering from SVT is typically rather favourable when caught early and treated properly.

A frequent cardiac rhythm issue in children, supraventricular tachycardia (SVT) causes the heart to beat excessively fast.

Chapter 5.
Management of Hypovolemic Shock in Paediatric Patients

Inna Horbatiuk

Chapter 5. Management of Hypovolemic Shock in Paediatric Patients

It is vital to examine pediatric patients quickly and accurately in order to treat hypovolemic shock promptly because it is a serious condition. Reduced tissue perfusion and oxygen delivery are symptoms of the illness, which is marked by an inadequate volume of blood circulating in the body. A lack of fluids due to burns, vomiting, diarrhea, or extreme dehydration can trigger this condition. In order to properly manage shock in children, rapid identification and classification of the condition is crucial. The following is a synopsis of the signs, symptoms, and treatment options for pediatric hypovolemic shock.

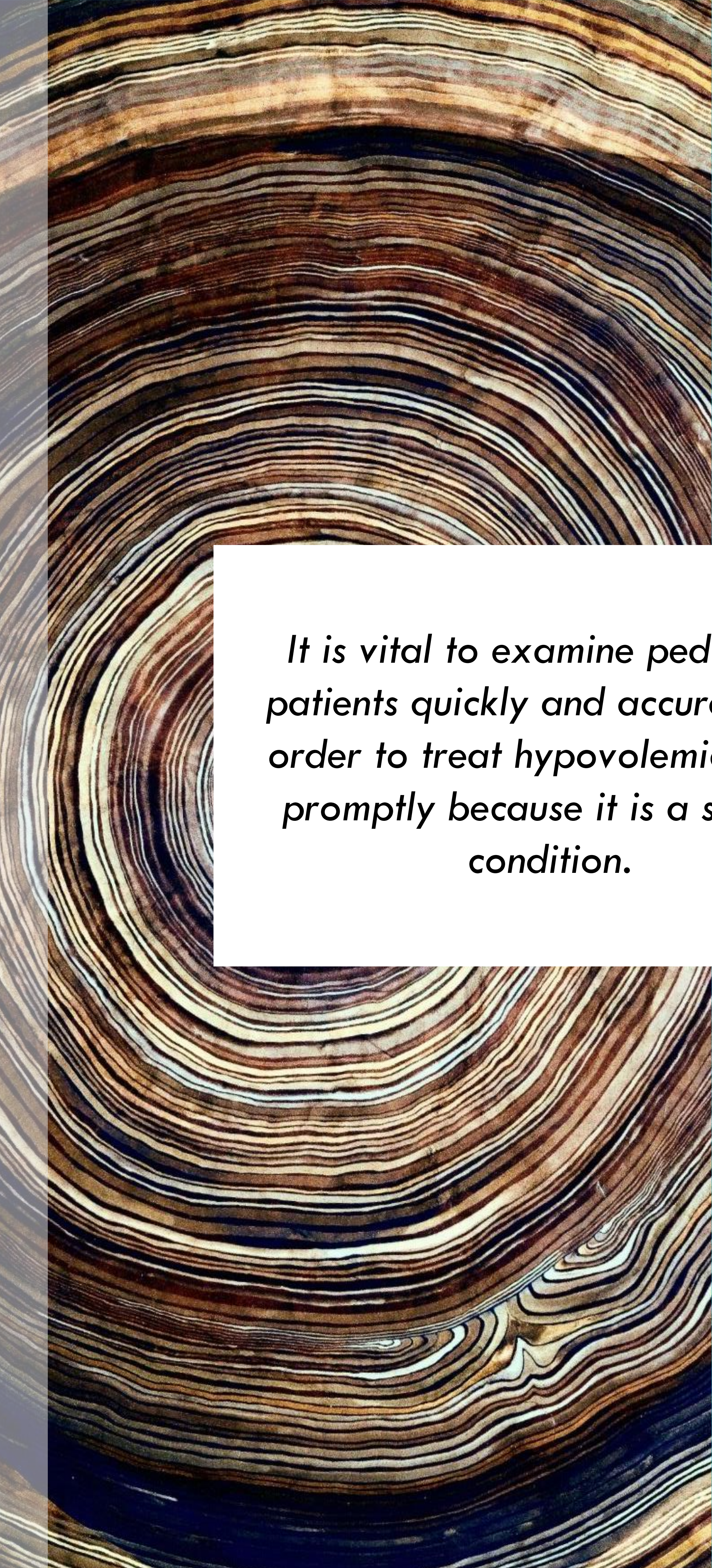
In order to properly manage shock in children, rapid identification and classification of the condition is crucial.

Identification of Critical Childhood Illnesses

The most critically sick children brought to hospitals can be quickly identified with the use of a triage system. Rapid evaluation of appearance, respiration, and circulation can be achieved with the use of tools like the Pediatric Assessment Triangle (PAT), which can reveal conditions that require urgent care. A decrease in cerebral perfusion, a symptom of shock, might manifest physically as changes in appearance like weak crying, poor muscle tone, or an unfocused look. Tachypnea without respiratory distress and other breathing-related symptoms can emerge in reaction to metabolic acidosis. Delays in capillary filling and weak pulses are common symptoms of poor circulation that can be detected prior to taking a blood pressure reading.

As soon as problems emerge, the ABCDE method can be used as a systematic evaluation tool:

- **A (Airway)**: Assist in keeping the airway open and secure.
- **B (Breathing)**: Measure oxygenation, tidal volume, respiratory rate, and effort of breathing (bruising, retractions, etc.). While diagnosing hypoxia, techniques such as capnography and thoracic ultrasound might be helpful, as the condition is not often accompanied by obvious symptoms.
- **C (Circulation)**: Check the patient's vitals, including heart rate, blood pressure, urine output, renal function, and awareness. Keep in mind that CRT isn't very sensitive, so it's best to stick to other forms of monitoring (such blood pressure, cardiac ultrasonography, and lactate measures) instead.
- **D (Disability)**: Make that the patient is conscious by administering tests like the Glasgow Coma Scale or the Alert-Verbal-Pain-Unresponsive (AVPU) score. If the GCS score is 8 or below or the AVPU score is P or lower, it means that the state of consciousness is severely disturbed.
- **E (Exposure)**: With the patient's dignity maintained and heat loss minimized, expose them fully for a complete physical examination.



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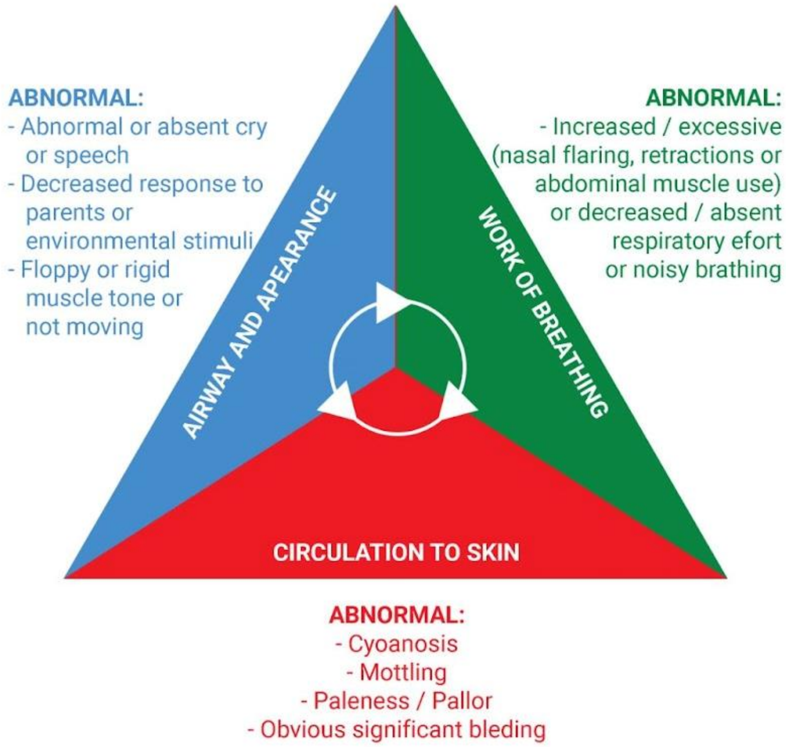


Figure. Paediatric Assessment Triangle

Assessing Hypovolemic Shock in Pediatric Patients

There are several potential causes of shock in children, including hypovolemic (inadequate blood volume), distributive (wrong blood distribution), cardiogenic (impaired cardiac contractility), and obstructive shocks (blood flow obstruction). According to the World Health Organization (WHO), a combination of cold extremities, a weak and fast pulse, and a CRT longer than three seconds indicates significant compromised circulation. These symptoms, along with a weak and rapid pulse, indicate shock.

Key signs of hypovolemic shock include rapid heart rate, normal systolic blood pressure with narrow pulse pressure, slow or nonexistent peripheral pulses, pale skin, delayed capillary refill, and decreased urine output. Compensated shock is characterized by moderate symptoms including pallor of the skin and rapid heart rate; uncompensated shock is characterized by low blood pressure, acidosis, and diminished mental status; and irreversible shock is characterized by organ failure despite treatment.

Stages and Pathophysiology

An increase in thirst and a decrease in renal fluid conservation are the first lines of defense against hypovolemic shock, which occurs when blood volume is inadequate to sustain tissue perfusion. Untreated, these processes can lead to tachycardia, which is characterized by a rapid heart rate, higher systemic vascular resistance, which is necessary to reroute blood to important organs, and improved cardiac contractility, which is necessary to sustain stroke volume.

We classify mild hypovolemia (3-5%), moderate hypovolemia (6-9%), and severe hypovolemia (≥10%) in children who do not have bleeding according to their weight loss. Based on the proportion of blood loss, there are four classes of hemorrhagic shock. Class II shock, which occurs in patients with 15- 30% blood loss, is followed by all patients with 30-40% and >40% shock. At this point, major clinical signs begin.

Laboratory and Clinical Evaluation

Quick identification of clinical symptoms such as sunken eyes, low skin turgor, lethargy, or altered mental status is essential when assessing hypovolemia and dehydration. A urine dipstick, serum electrolytes, blood lactate, and fasting blood glucose are all necessary laboratory tests to diagnose hypovolemic shock or severe dehydration. Further imaging studies, such as radiography of the abdomen and chest, may be required in cases when the patient does not show enough improvement after the first round of fluid treatment.

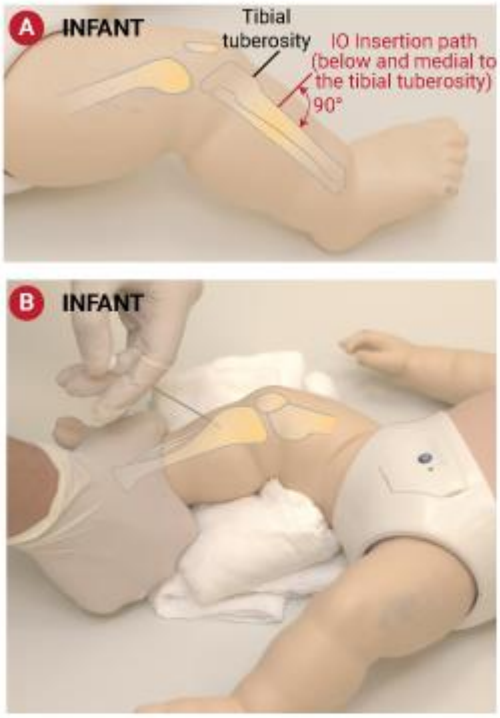


Figure. Intraosseous (IO) access (A. General landmarks for IO insertion in the leg of an infant B. Technique for immobilising the leg while twisting the IO needle into the leg of an infant)

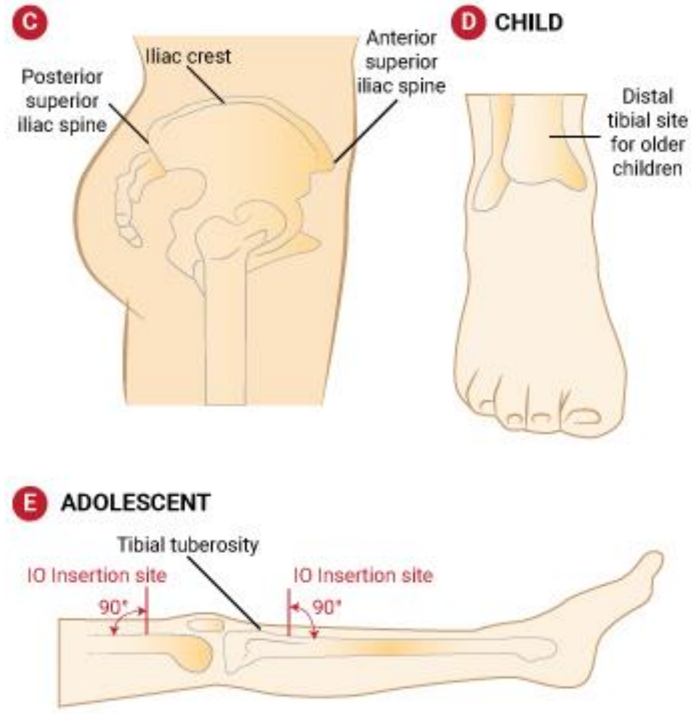
Non-Hemorrhagic Hypovolemic Shock Management

When treating children with hypovolemic non-hemorrhagic shock, it is essential to administer fluids quickly, preferably during the first one or two hours. Fluid boluses are best administered with a balanced crystalloid solution, however plain saline can do. The usual starting dose is 10 milliliters per kilogram over 5 to 10 minutes, although as much as 40 to 60 milliliters per kilogram may be required in the first hour. Avoiding fluid-related problems or overdosing the child requires close monitoring of clinical reaction following each bolus.

For quick fluid administration, intraosseous (IO) access can be a good alternative to intravascular (IV) access if it is difficult. Adequate analgesia, the use of needles of the correct size, and constant monitoring for problems like extravasation allow for IO access to be accomplished.

If fluid therapy doesn't work, it's best to start vasoactive medications and respiratory support right away. As a first-line vasopressor, noradrenaline or adrenaline are recommended; as an inodilator, dobutamine or milrinone could be used. When none of these drugs are available, dopamine is considered.

Figure. Intraosseous (IO) access (C. Location for IO insertion in the iliac crest D. Location for IO insertion in the distal tibia E. Locations for IO insertion in the proximal tibia and distal femur in older children)



Post-Resuscitation Care and New Perspectives

The danger of post-resuscitation shock is significant even after hypovolemic shock has been successfully resuscitated. This disorder, which manifests heart failure and symptoms similar to sepsis, is the result of systemic ischemia-reperfusion damage. Stabilizing circulation and perfusion are the goal of intensive hemodynamic support, which involves the use of vasopressors and inotropes. Patients in critical care who have a good neurological prognosis may require mechanical support, such as extracorporeal membrane oxygenation (ECMO).

Emerging topics such as the management of vasoplegia in post-resuscitation shock with arginine-vasopressin are the subject of continuing investigation. Given the susceptibility of the developing brain to ischemia damage, these approaches seek to accomplish "hormonal healing" by joining forces with therapies such as hydrocortisone and tailored temperature control.

In order to properly manage shock in children, rapid identification and classification of the condition is crucial.

Chapter 6. Tension Pneumothorax in Adult Patients

Ruslan Knut, Iryna Kozlovska

Chapter 6. Tension Pneumothorax in Adult Patients

Tension pneumothorax is a severe medical condition where air accumulates in the pleural space, the area surrounding the lungs. This air buildup increases pressure on the lung, causing it to collapse and compromising the heart's ability to function correctly. In some cases, this can lead to death. The increased pressure squeezes the lungs, heart, and major blood vessels, which reduces blood flow returning to the heart and diminishes cardiac output. Essentially, the heart struggles to pump effectively. Tension pneumothorax is a reversible cause of cardiac arrest, often grouped with other critical conditions like tamponade, thrombosis, and toxins, and it demands immediate treatment to prevent circulatory collapse.

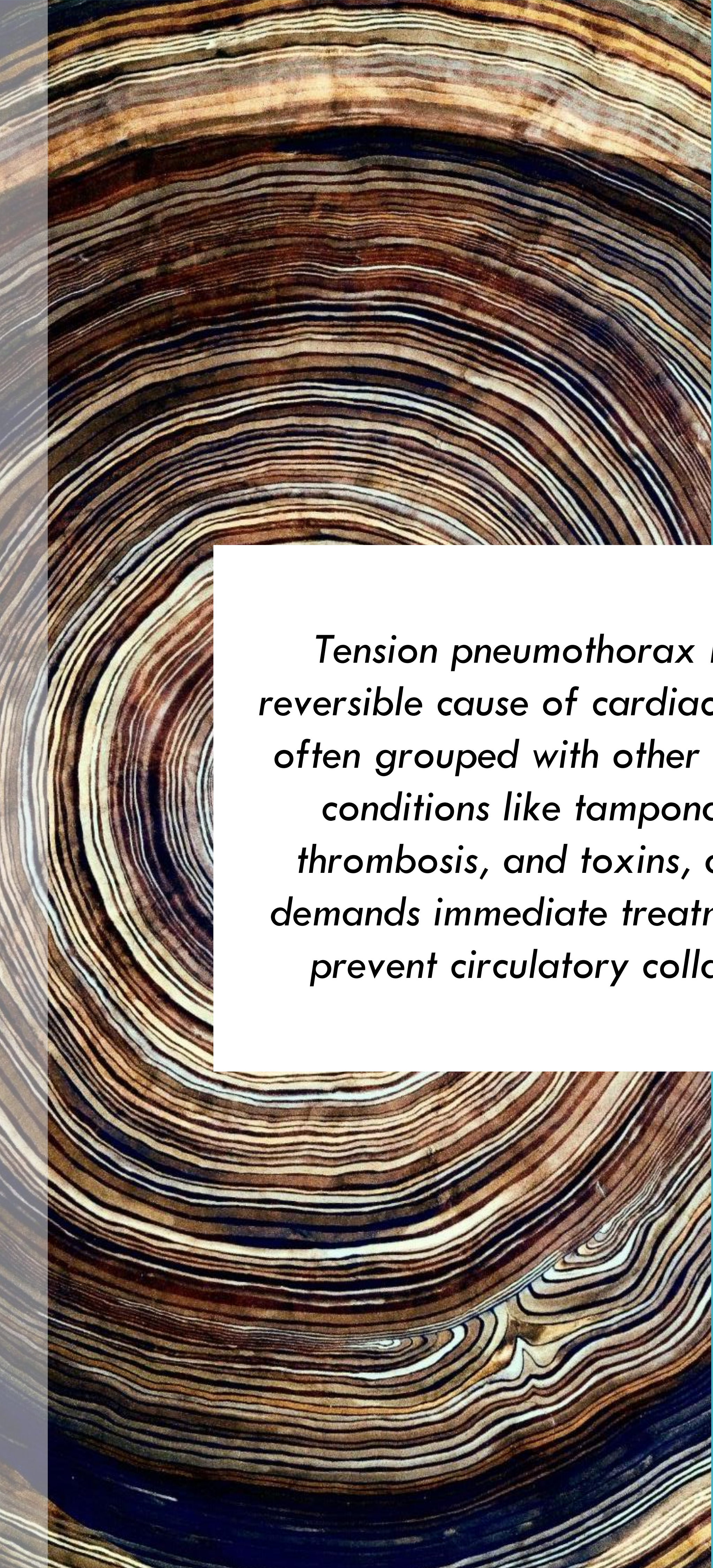
Origins and Contributing Factors

This condition can arise from various factors. One of the most common is mechanical ventilation, especially in patients who already have some form of lung damage. Chest injuries, whether from accidents or medical procedures like inserting central lines, can also lead to tension pneumothorax. In essence, any lung injury that creates an opening but prevents it from sealing properly can cause air to build up in the pleural space. Additionally, abnormalities in the chest wall can sometimes act like one-way valves, permitting air to enter the pleural space but not escape. In rare instances, tension pneumothorax may develop without any clear cause.

Risk Factors

Several factors increase the risk of developing this condition. Trauma to the chest can rupture the pleura or cause the lung to collapse. People with lung conditions such as COPD, cystic fibrosis, lung cancer, or pneumonia are also more susceptible. Small air sacs in the lung, known as blebs, can rupture and release air into the pleural space. The use of positive pressure ventilation can disrupt the pressure balance in the lungs, making them more prone to collapse. Furthermore, both genetics and smoking, even in the absence of emphysema, increase the risk. There are even familial forms of pneumothorax that are passed down through families. Tension pneumothorax tends to be more common in men, especially those between 20 and 40 years old who are tall and underweight. Having a previous episode of pneumothorax also increases the likelihood of recurrence.

Tension pneumothorax is a severe medical condition where air accumulates in the pleural space, the area surrounding the lungs.



Tension pneumothorax is a reversible cause of cardiac arrest, often grouped with other critical conditions like tamponade, thrombosis, and toxins, and it demands immediate treatment to prevent circulatory collapse.

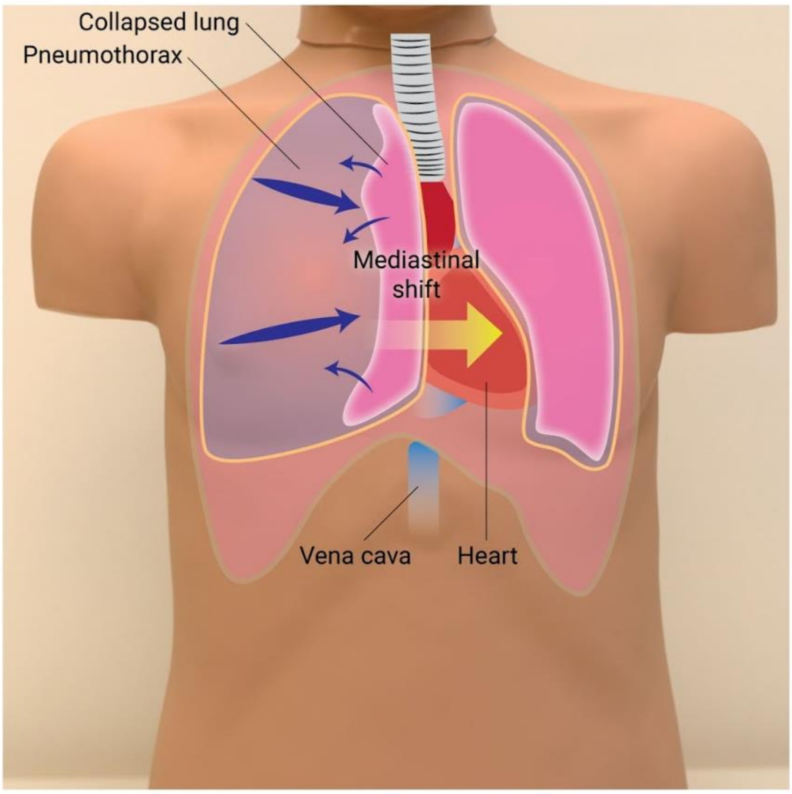


Figure. Tension Pneumothorax

Recognizing the Signs

The symptoms of tension pneumothorax can vary. Patients breathing on their own often experience rapid breathing and a feeling of air hunger, while those on mechanical ventilation may exhibit signs of cardiovascular collapse. Common signs and symptoms include:

- Sudden chest pain
- Severe shortness of breath
- Low blood pressure
- Rapid heart rate
- Tracheal deviation (the trachea shifts away from the affected side of the chest)
- Absent breath sounds on the affected side
- An elevated and immobile side of the chest
- Distended neck veins
- Cyanosis (a bluish discoloration of the skin) - a late sign of severe oxygen deprivation

Pathophysiology

The process leading to tension pneumothorax begins with air becoming trapped in the pleural space, causing the lung to collapse. As the mediastinum shifts and gas exchange is compromised, the heart and lungs struggle to function effectively, potentially leading to shock and cardiac arrest.

Diagnosis

In many cases, treatment starts immediately based on a strong clinical suspicion even before imaging results are available. Critically ill patients require a rapid assessment using the ABCDE approach. This involves:

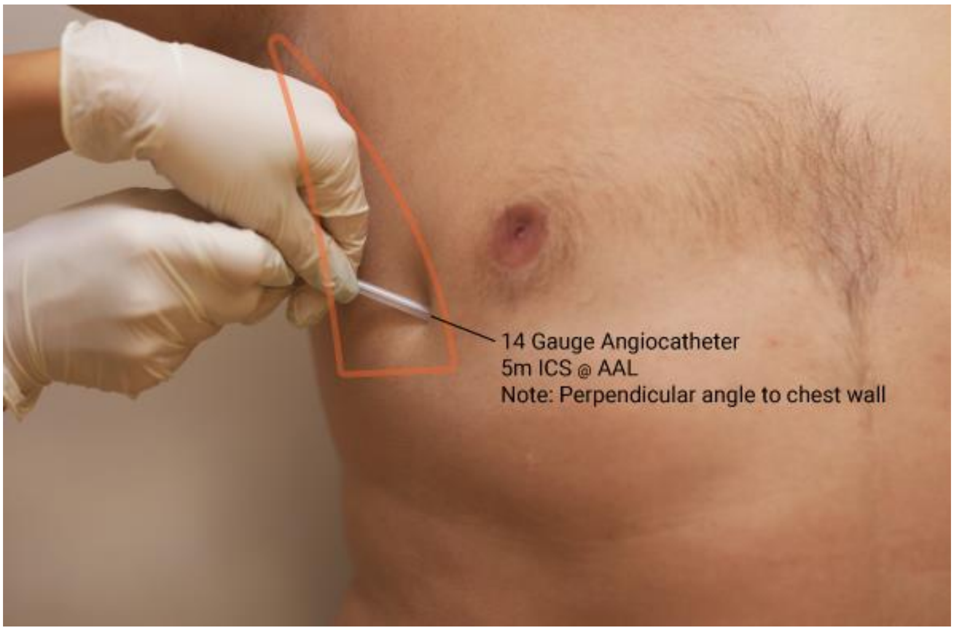
- Ensuring the airway is open
- Assessing breathing for signs of distress
- Evaluating circulation for signs of shock
- Checking for neurological impairment
- Examining the chest for injuries

Chest X-rays are essential for confirming the diagnosis. They may reveal:

- A flattened diaphragm
- A darker appearance on the affected side
- A collapsed lung
- Shifting of the mediastinum
- Spread-out ribs

Point-of-care ultrasound can also be used for quick bedside diagnosis.

Figure. Needle decompression in tension pneumothorax



Emergency Management

Immediate decompression of the chest is crucial to alleviating pressure within the pleural cavity. This is often done through:

- **Needle decompression**: A large-bore needle is inserted into the pleural space to release the trapped air.
- **Tube thoracostomy**: A chest tube is inserted to allow the lung to re-expand and prevent further air accumulation. This procedure demands careful technique and close monitoring. After the chest tube is placed, an X-ray is taken to ensure it's in the correct position.

Following decompression, the patient needs ongoing monitoring and further stabilization. In some cases, consultation with a thoracic surgeon is necessary.

Tension pneumothorax is a severe medical condition where air accumulates in the pleural space, the area surrounding the lungs.

Chapter 7.
Management of Cardiac Tamponade Due to Trauma

Iryna Kozlovska, Ruslan Knut

Chapter 7. Management of Cardiac Tamponade Due to Trauma

Cardiac tamponade is a serious medical emergency where fluid, usually blood, builds up in the pericardial sac, the space surrounding the heart. This fluid accumulation increases pressure on the heart, preventing its chambers from filling properly and drastically reducing cardiac output. If left untreated, cardiac tamponade quickly leads to cardiovascular collapse and cardiac arrest. It's crucial to recognize and treat this condition promptly, as it's a reversible cause of cardiac arrest.

Causes and Risk Factors

Cardiac tamponade can result from various factors, with injuries being a common cause. These injuries can include blunt trauma (like falls or blows to the chest), penetrating trauma (such as stab wounds or gunshot wounds), aortic dissection, rupture of the heart wall (often after a heart attack), and complications from invasive procedures or cardiac surgeries.

Blunt trauma can cause blood to accumulate rapidly in the pericardial space (hemopericardium). Penetrating injuries that puncture the pericardium or nearby blood vessels can also lead to rapid tamponade. Medical procedures, such as inserting central venous catheters or pacemakers, or even chest compressions during CPR, can inadvertently damage the pericardium and cause bleeding.

Non-traumatic causes of cardiac tamponade include autoimmune diseases, radiation damage, chronic kidney failure (uremic pericarditis), idiopathic pericarditis, cancer, and other chronic pericardial diseases. These conditions often lead to a gradual accumulation of fluid, which can also result in tamponade, although it typically develops more slowly.

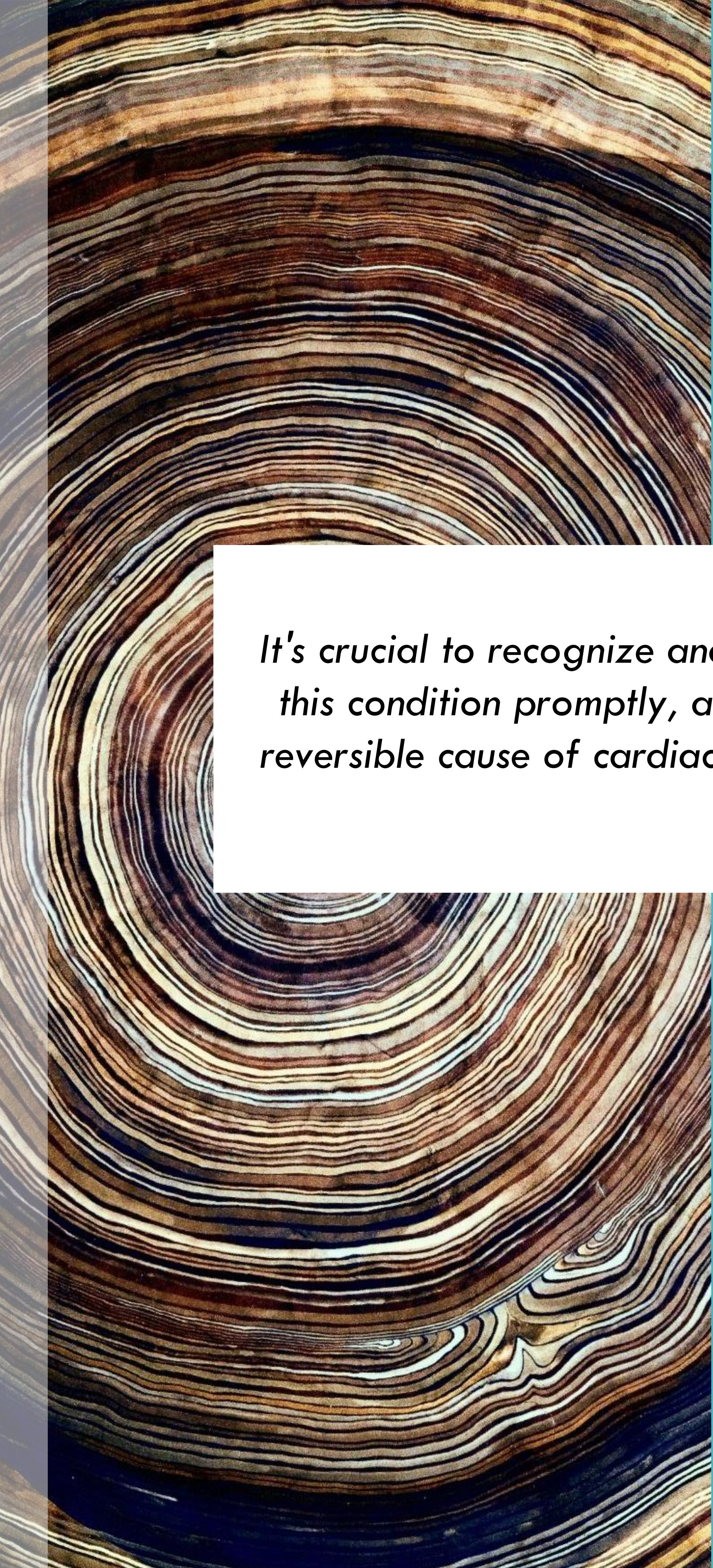
Recognizing the Signs

Clinical diagnosis of cardiac tamponade is essential. The speed of fluid accumulation and the underlying cause influence the presenting symptoms. Acute tamponade, especially after trauma, often causes more obvious symptoms, while chronic tamponade may develop more subtly. The hallmark signs of tamponade result from impaired venous return and reduced cardiac output.

Beck's Triad is a classic presentation of cardiac tamponade and includes:

- Hypotension (low blood pressure) due to decreased cardiac output
- Jugular venous distension (bulging neck veins) reflecting increased venous pressure
- Muffled heart sounds due to the fluid surrounding the heart

Cardiac tamponade is a serious medical emergency where fluid, usually blood, builds up in the pericardial sac, the space surrounding the heart.



It's crucial to recognize and treat this condition promptly, as it's a reversible cause of cardiac arrest.

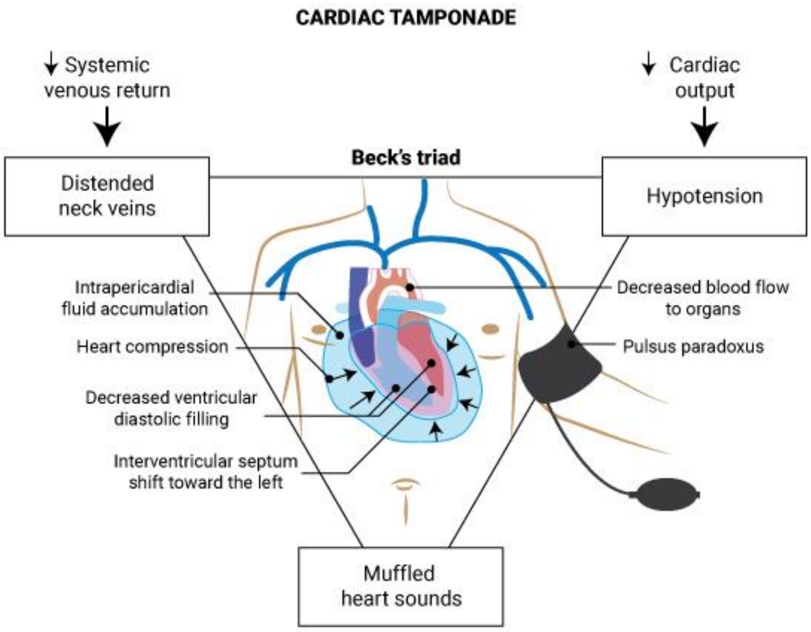


Figure. Beck's triad

Other signs and symptoms may include:

- Tachycardia (rapid heart rate) as a compensatory mechanism to maintain cardiac output
- Pulsus paradoxus, a significant drop in systolic blood pressure during inhalation
- Signs of poor perfusion, such as anxiety, agitation, weak or absent peripheral pulses, dizziness, shortness of breath, and chest pain that worsens with breathing or coughing
- Kussmaul's sign, a paradoxical rise in jugular venous pressure during inspiration
- Cyanosis (bluish discoloration of the skin), cold and clammy skin, and signs of shock in severe cases

Several diagnostic tools can help confirm the clinical suspicion of cardiac tamponade.

Diagnostic Tools

Several diagnostic tools can help confirm the clinical suspicion of cardiac tamponade:

- Echocardiography is a highly reliable method for visualizing the fluid accumulation, chamber collapse, and changes in ventricular filling during breathing. The collapse of the right ventricle and atrium during diastole is a strong indicator of tamponade.
- Chest X-ray may show an enlarged heart silhouette with clear lung fields in cases of significant fluid buildup, sometimes referred to as the "water bottle" sign.
- Electrocardiogram (ECG) may reveal tachycardia, electrical alternans (a beat-to-beat variation in the QRS complex), or low voltage QRS complexes.
- Computed tomography (CT) scans or cardiac magnetic resonance imaging (CMR) can provide more detailed information in non-emergency situations but are usually not necessary for initial diagnosis.

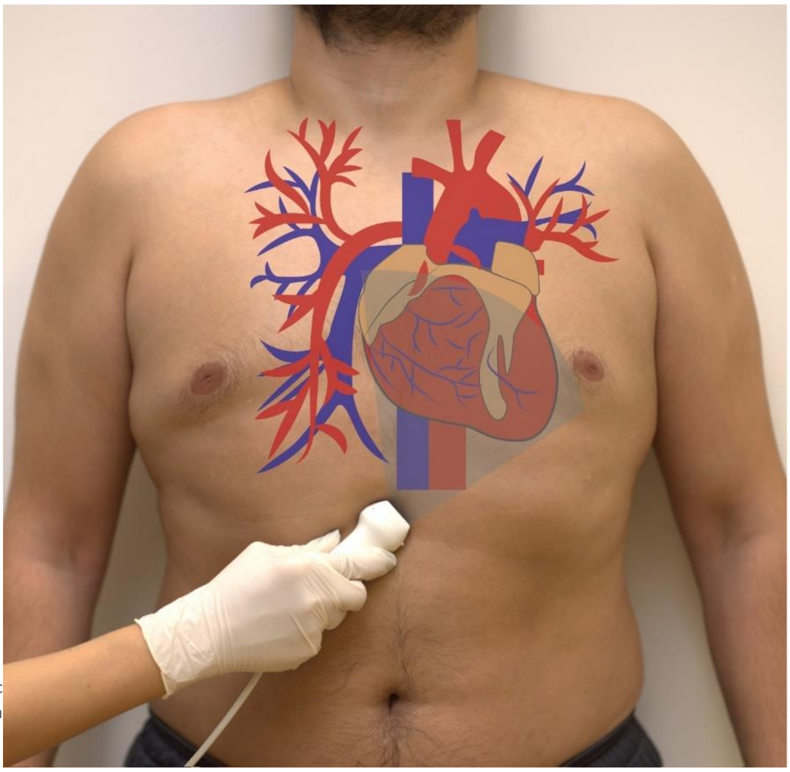


Figure. Two-dimensional echocardiogram illustrating cardiac tamponade with right atrium collapse or indentation

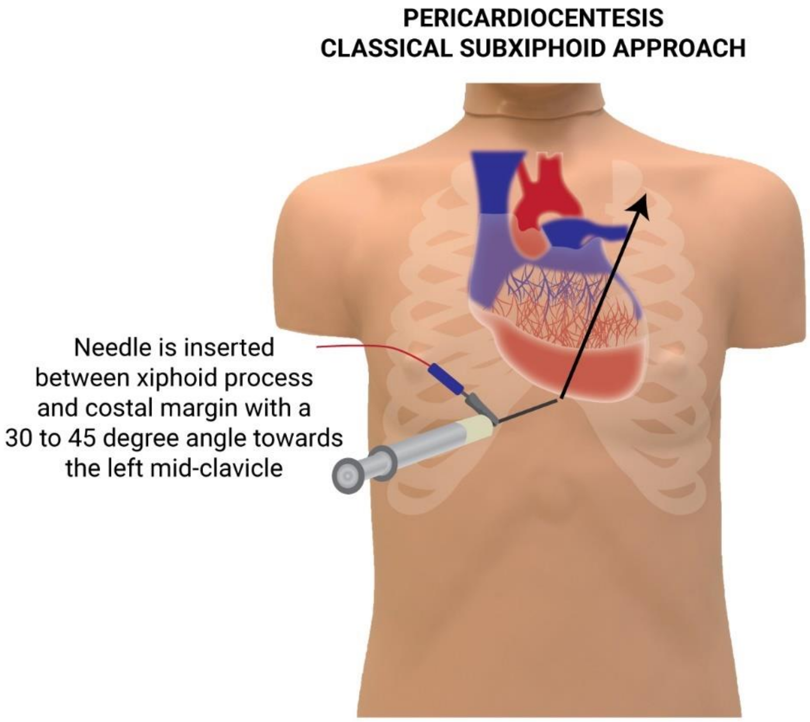
Emergency Treatment

The primary goal of emergency treatment is to relieve pressure on the heart and restore cardiac function. Two main interventions are commonly used:

- Pericardiocentesis involves inserting a needle into the pericardial sac to drain the fluid, often guided by ultrasound. A catheter is usually left in place to ensure continuous drainage.
- In cases of recurrent fluid buildup, loculated effusions (fluid trapped in pockets), or ongoing bleeding, a pericardial window may be necessary. This surgical procedure creates an opening in the pericardium to prevent fluid from re-accumulating and allow for continuous drainage.

In critical situations where cardiac arrest is imminent, resuscitative measures are performed concurrently with efforts to drain the fluid. Supportive care includes oxygen administration, intravenous fluids or blood products, and inotropic medications (like dobutamine) to help the heart pump effectively until definitive treatment can be provided.

Figure. A procedure that uses a needle to remove fluid from the tissue that surrounds the heart will be done



Emergency Care and Monitoring

The ABCDE approach (airway, breathing, circulation, disability, and exposure) guides the management of patients with suspected cardiac tamponade. Monitoring is crucial and includes:

- Close observation of hemodynamic parameters, such as heart rate, blood pressure, and central venous pressure
- Pulse oximetry to ensure adequate blood oxygen levels
- Continuous cardiac monitoring to detect arrhythmias and monitor heart rate and rhythm
- Avoiding interventions that decrease venous return, such as positive pressure mechanical ventilation, which can worsen cardiac filling

Cardiac tamponade is a serious medical emergency where fluid, usually blood, builds up in the pericardial sac, the space surrounding the heart.

Chapter 8. Pulseless Electrical Activity (PEA)

Serhii Malaiko, Oleksii Godovanets

Chapter 8. Pulseless Electrical Activity (PEA)

Pulseless electrical activity (PEA) is a critical form of cardiac arrest where organized electrical activity is present on an electrocardiogram (ECG), but there is no palpable pulse. This occurs when the heart's electrical system appears to be functioning, but the heart muscle fails to contract effectively, resulting in inadequate blood circulation. PEA accounts for a significant proportion of cardiac arrests, both in and out of the hospital setting. Rapid recognition and treatment are essential to address reversible causes and restore blood flow.

Understanding PEA and its Causes

PEA can be presented in two main forms: narrow-complex (where the QRS complex on the ECG is narrow) and wide-complex (where the QRS complex is wide). Narrow-complex PEA often results from mechanical obstruction of blood flow into or out of the right ventricle, while wide-complex PEA is commonly associated with left ventricle failure due to ischemia (lack of blood flow) or metabolic disturbances.

It's important to distinguish between:

- **Pseudo-PEA:** where there is some electrical activity and weak contractions, but not enough to generate a palpable pulse
- **True PEA:** where electrical activity is present, but the heart muscle is completely unresponsive, leading to no blood circulation

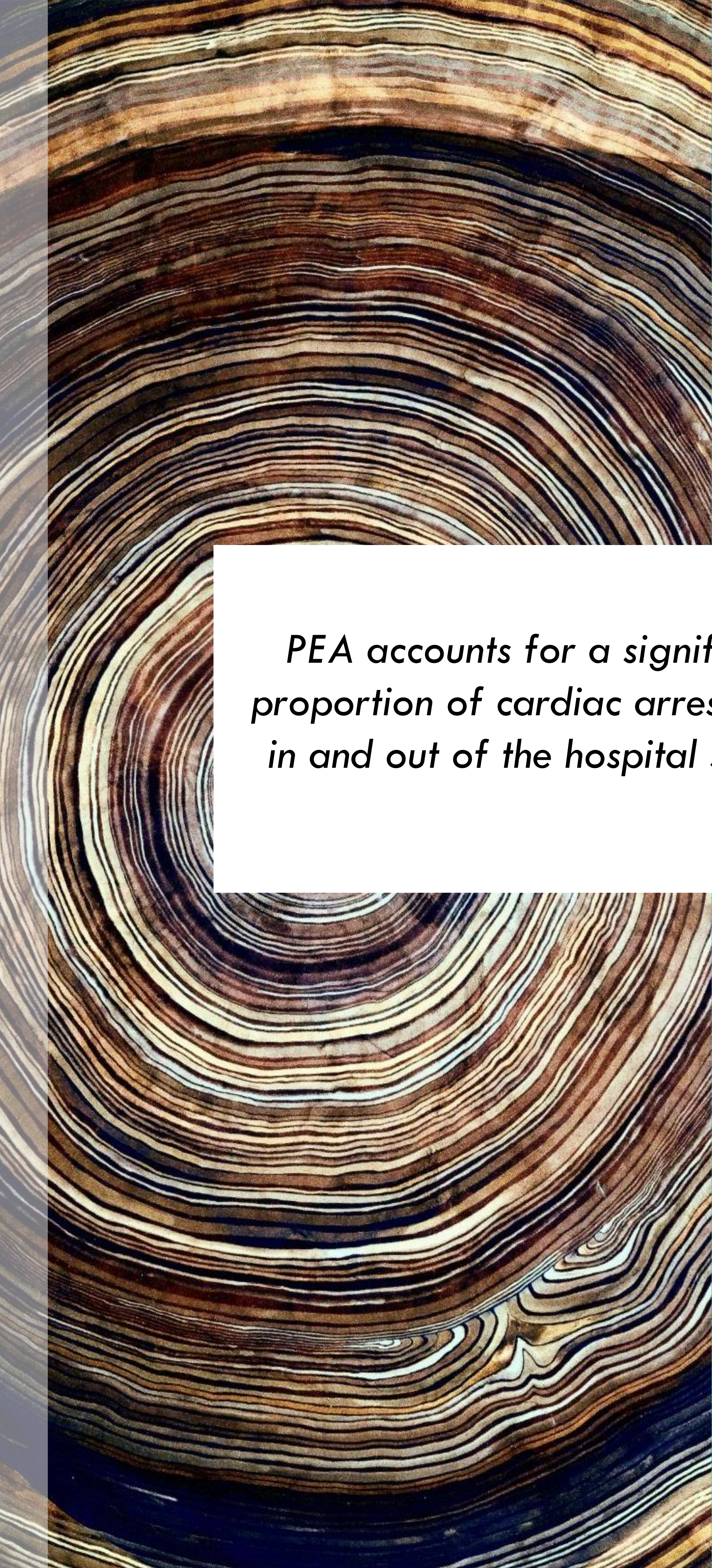
The "4 Hs and 4 Ts" mnemonic helps remember the most common reversible causes of PEA:

- **4 Hs:** Hypovolemia, Hypoxia, Hypokalemia/Hyperkalemia, Hypothermia/Hyperthermia
- **4 Ts:** Toxins, Tamponade (cardiac), Tension pneumothorax, Thrombosis (coronary or pulmonary)

Recognition and Diagnosis

PEA typically presents with loss of consciousness and absence of breathing. To confirm the diagnosis, quickly assess the airway for obstruction, observe for breathing effort, and check for a carotid pulse (for no more than 10 seconds). An ECG should be obtained immediately to determine the type of PEA (narrow or wide complex) and guide the identification of potential causes.

Pulseless electrical activity (PEA) is a critical form of cardiac arrest where organized electrical activity is present on an electrocardiogram (ECG), but there is no palpable pulse.



PEA accounts for a significant proportion of cardiac arrests, both in and out of the hospital setting.

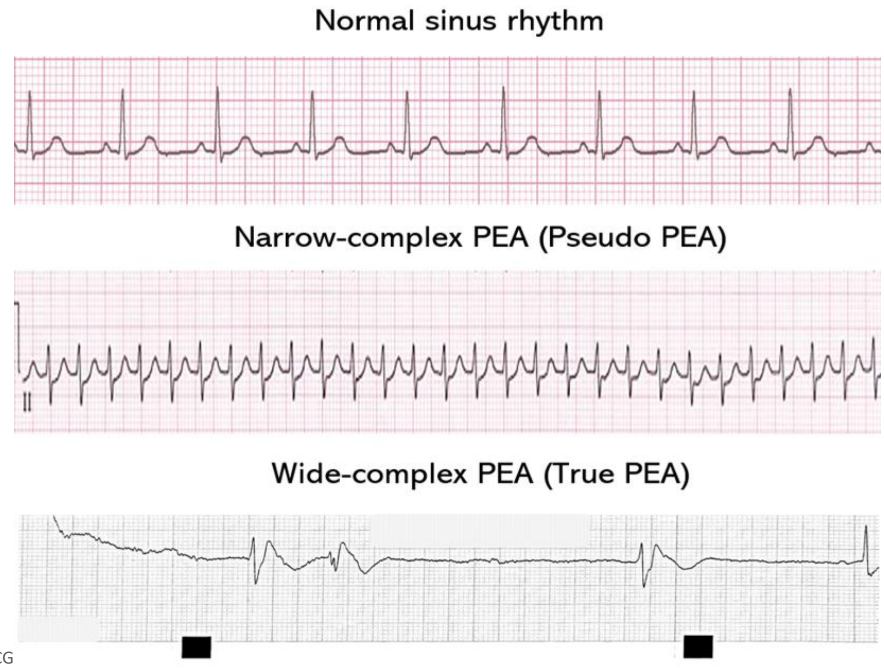


Figure. ECG

Treatment Protocol

The Advanced Cardiac Life Support (ACLS) protocol emphasizes the ABCDE approach for managing PEA:

- **A (Airway):** Ensure a patent airway
- **B (Breathing):** Provide ventilation with Ambu bag 500 ml per every breath and 100% oxygen support until oxygen saturation is above 94%.
- **C (Circulation):** Provide high-quality chest compressions at a rate of 100-120 compressions per minute with minimal interruptions. Establish cardiac monitoring to assess heart rhythm and blood pressure. Administer epinephrine 1 mg intravenously or intraosseously every 3-5 minutes to improve blood flow to the heart and brain.
- **D (Disability):** Assess neurological status.
- **E (Exposure):** Examine for underlying causes and address reversible factors. This may involve fluid administration for hypovolemia, ventilation for hypoxia, needle decompression for tension pneumothorax, and so on.

If the rhythm changes to ventricular tachycardia (VT) or ventricular fibrillation (VF), defibrillation should be performed according to the VF/VT algorithm.

Addressing Reversible Causes

Each reversible cause of PEA requires specific treatment:

- **Hypovolemia:** Control bleeding and administer fluids or blood products.
- **Hypoxia:** Provide ventilation if needed and administer high-flow oxygen.
- **Hyperkalemia/Hypokalemia:** Manage potassium levels with medications like calcium gluconate, insulin-dextrose, or potassium replacement.
- **Hyperthermia/hypothermia:** Use infusion therapy with warmed/cooled solutions, and thermal blankets.
- **Toxins:** Follow specific guidelines for antidote administration, activated charcoal, or enhanced elimination based on the substance involved.
- **Cardiac Tamponade:** Perform pericardiocentesis to relieve pressure.
- **Tension Pneumothorax:** Perform needle thoracostomy for decompression.
- **Thrombosis:** Consider thrombolysis or surgical intervention depending on the location and type of clot.

Medications and Interventions

- Epinephrine is a key medication in PEA to increase coronary and cerebral perfusion pressure.
- Atropine can be considered for bradycardia (slow heart rate) with hypotension, but its use is limited to a maximum of three doses.
- Sodium bicarbonate is generally reserved for specific situations like hyperkalemia, severe metabolic acidosis, or tricyclic antidepressant overdose.
- Surgical interventions, such as pericardiocentesis or thoracotomy, may be necessary in certain cases, like cardiac tamponade or trauma-induced PEA. Prompt consultation with cardiothoracic surgery or relevant specialists is crucial for definitive management.

Table. The 4 Hs and the 4 Ts

4 Hs	4 Ts
• Hypoxia • Hypokalemia/Hyperkalemia • Hypothermia/Hyperthermia • Hypovolemia	• Tension pneumothorax • Tamponade • Thrombosis (pulmonary/coronary) • Toxins

Prognosis and Survival

The prognosis for PEA is generally poor unless a rapidly reversible cause is identified and treated promptly. Survival depends on the underlying cause and the duration of resuscitation efforts. Patients with PEA from out-of-hospital cardiac arrest tend to have better outcomes than those with in-hospital cardiac arrest, likely due to a higher prevalence of reversible causes like hypothermia or electrolyte imbalances.

Despite advances in treatment, the survival rate for PEA remains low, highlighting the importance of effective and rapid resuscitation and treatment of reversible causes.

Pulseless electrical activity (PEA) is a critical form of cardiac arrest where organized electrical activity is present on an electrocardiogram (ECG), but there is no palpable pulse.

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