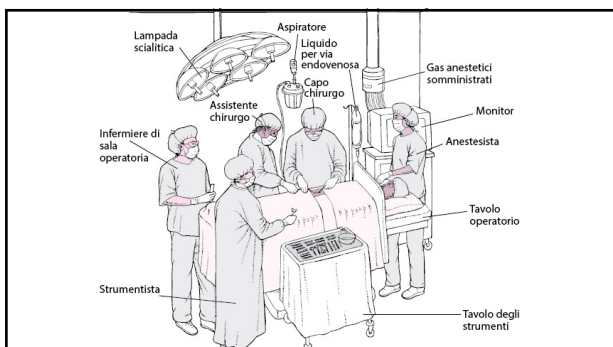
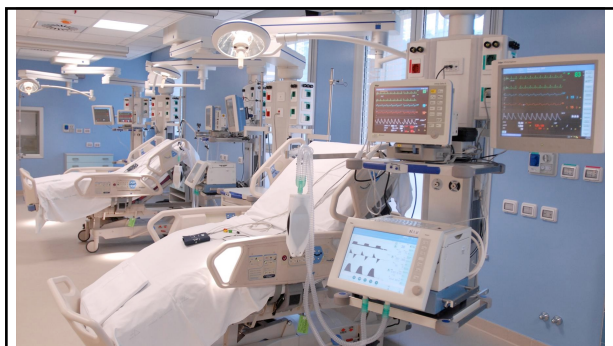




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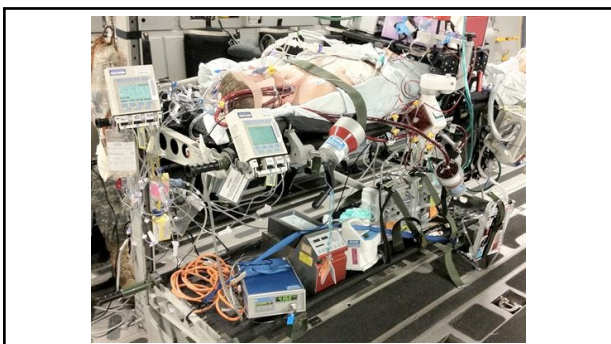
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CHI E' IL PAZIENTE CRITICO?

Patologie che necessitano di un trattamento immediato con percorsi diagnostico terapeutici preospedalieri e intraospedalieri adeguati

- Shock
- Insufficienza respiratoria acuta
- Stroke - ESA - ICH - SAH
- Sindrome coronarica acuta (NSTEMI - STEMI)
- Arresto cardiaco
- Trauma
- Patologie vascolari (shock emorragico)
- Patologie renali gravi

5




6

CRITERI DI GRAVITA'

Frequenza cardiaca < 40 bpm o >140 bpm
 Pressione sistolica <90 mmHg
 Saturazione arteriosa < 90%
 Riempimento capillare >3 s
 Decadimento neurologico - GCS

Instabilità emodinamica

Segni clinici:
 Astenia
 Dispnea (con o senza cianosi)
 Dolore toraco addominale
 Pallore cutaneo, cute fredda, sudorazione profusa e algida
 Tachicardia o bradicardia (aritmie)
 Stato di agitazione, alterazioni dello stato di coscienza (confusione, sopore)
 Turgore delle giugulari, stasi epatica, edemi arti inferiori
 Contrazione della diuresi



7

SHOCK

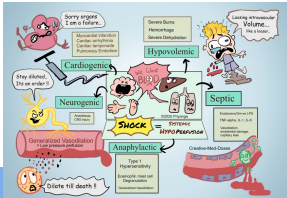
Riduzione della PA associata a sintomi e segni di ipoperfusione tissutale secondaria a:

IPTENSIONE
IPOPERFUSIONE
ALTERAZIONE CELLULARE

(< flusso sanguigno): Inadeguata perfusione tissutale
 Inadeguato trasporto/disponibilità di O₂

(sepsi): Alterazione utilizzo cellulare di O₂
 Insoddisfatte esigenze metaboliche cellulari

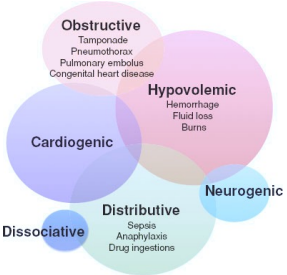
Ipotensione riduce la perfusione e di conseguenza apporto di O₂ e metaboliti
 Alterazioni metaboliche cellulari e nel tempo anche d'organo



8

SHOCK

Definizione:
 Insufficienza acuta del circolo periferico con tendenza all' irreversibilità, ad eziologia molteplice che determina ipoperfusione tissutale ed anossia cellulare.

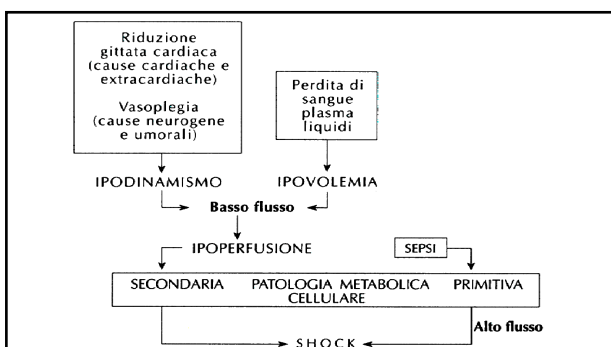


9

Types of Shock

- Hypovolemic Shock**
 - Cause: Severe blood or fluid loss (e.g., hemorrhage, dehydration).
 - Pathophysiology: Reduced circulating volume leads to inadequate tissue perfusion.
 - Clinical Presentation: Tachycardia, hypotension, cold extremities, and oliguria.
- Cardiogenic Shock**
 - Cause: Heart pump failure (e.g., myocardial infarction, heart failure).
 - Pathophysiology: Impaired cardiac output despite adequate intravascular volume.
 - Clinical Presentation: Hypotension, distended jugular veins, pulmonary edema.
- Distributive Shock**
 - Cause: Abnormal blood distribution (e.g., septic shock, anaphylactic shock, neurogenic shock).
 - Pathophysiology: Vasodilation causes a relative hypovolemia despite normal or increased cardiac output.
 - Clinical Presentation: Warm extremities (septic), hypotension, altered mental status.
- Obstructive Shock**
 - Cause: Mechanical obstruction of the heart or great vessels (e.g., pulmonary embolism, cardiac tamponade).
 - Pathophysiology: Impaired ventricular filling or emptying, leading to decreased cardiac output.
 - Clinical Presentation: Hypotension, jugular vein distention, pulsus paradoxus.

10



11

Tipi di SHOCK

- Puri
- Misti

- persistere evento patogenetico
- validita' risposta sistemica
- tempestivita' ed adeguatezza intervento terapeutico

Caratteristiche

ipovolemia assoluta (emorragia)
 vasoplegia (trauma midollo spinale)
 alterazione cardiaca (trauma toracico)
 iperdinamismo circolatorio (sepsi post-traumatica)

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12

Tipi di SHOCK

Settico (62%) ... ICU
 Cardiogeno (16%)
 Ipovolemico (16%)
 Emorragico: Aree urbane, trauma center
 Distributivo (4%)
 Ostruttivo (2%)

Tipi di shock

1. Diminuzione del volume di liquidi
 - 1.1 shock ipovolemico
2. Aumento del diametro dei vasi
 - 2.1 shock neurogeno
 - 2.2 shock anafilattico
 - 2.3 shock settico
3. Diminuzione della capacità contrattile del cuore
 - 3.1 shock cardiogeno

Perdita volume
 Vasodilatazione periferica
 Fallimento di pompa

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13

SHOCK CARDIOGENO

Cardiogenic Shock
 Progressive Cycles of Inflammation, Ischemia, Vasoconstriction, and Volume Overload

Primary Cardiac Insult Myocardial Dysfunction

Manifestations of Cardiogenic Shock

- Systolic Blood Pressure < 90 mm Hg (or reduction > 40 mmHg)
- Cardiac Index < 2.2 L/min/m²
- Cardiac Power Output < 0.6 W
- Lactic acidosis

Causes of Primary Cardiac Insult:

- Myocardial Infarction
- Myocardial Dysfunction
- Myocarditis
- Valvular Disease
- Systemic Hypertension
- Coronary Artery Disease
- Myocardial Bridging
- Myocardial Bridge
- Myocardial Bridge
- Myocardial Bridge

Pathophysiology:

- Sympathetic Stimulation
- Renin-Angiotensin Activation
- Adrenergic Feedback
- Neurohormonal

Progression:

- Organ Perfusion
- Blood Pressure
- Vasoconstriction
- Mitogen Dysfunction
- Death

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14

Cardiogenic Shock Overview

Mechanisms of Origin

Cause: Primary pump failure of the heart due to:

- Acute coronary infarction (most common)
- Severe heart failure
- Cardiac tamponade
- Valvular dysfunction (e.g., mitral regurgitation, aortic stenosis)

Pathophysiology:

- Increased stroke volume → low cardiac output
- Inadequate perfusion of vital organs → tissue hypoxia

Progression:

- Initial Stage: Ischemia reduces cardiac contractility.
- Compensatory Stage: Sympathetic activation increases heart rate and systemic vascular resistance.
- Decompensatory Failure of compensatory mechanisms leads to:
 - Severe tissue hypoxia
 - Metabolic acidosis
 - Multiorgan dysfunction syndrome (MODS)

Cardiogenic Shock

Acute MI → Decreased coronary perfusion → Ischemia → Myocardial injury/death

Acute Decompensated HF / Cardiomyopathy / Myocarditis → Decreased CO → Hypotension → Vicious Cycle of Cardiac Injury → Hypotension and/or occlusion of coronary artery → Myocardial injury/death

15

Cardiogenic Shock Manifestations & Treatment

Clinical Manifestations

- Cardiogenic: Hypotension, tachycardia, elevated JVP, cold extremities
- Pulmonary: Pulmonary edema, dyspnea, hypoxemia
- Renal/Liver: Elevated mental status (confusion, lethargy)

Treatment Strategies

Initial Stabilization

- Oxygenation: Supplemental oxygen or mechanical ventilation
- Fluids: Fluids (cautious)
- Inotropes: Inotropes (e.g., Dobutamine) for contractility, Loop diuretics (e.g., furosemide) to maintain JVP

Revascularization

- PCI or CABG of myocardial infarction

Mechanical Support

- LVAD, VAD, or ECMO in refractory cases

Treat Underlying Cause (e.g., valve repair/replacement, arrhythmia treatment)

Cardiogenic shock management

A. Etiologic therapy
Coronary artery revascularization, ventricular disease management, arrhythmic and conduction disorders management

B. Optimizing Hemodynamic

1- Improve Tissue Perfusion

- If increase venous pressure and/or pulmonary congestion: Diuretics (e.g., furosemide) therapy
- If low blood pressure: Vasopressors (e.g., norepinephrine)
- Treated other causes of vasoplegic shock(s)

2- Improve Pump Function

- If Decrease RV or LV contractility to inotropes: Dobutamine (Dobutrex, Landiolol), Levosimendan

Manage Heart rate: Reduction of tachy- or bradycardia improves RV/ LV conduction disorder, improving

- If RV filling inadequate: Volume, Inotropes, Decrease afterload pressure
- If LV filling inadequate: Volume, Decrease afterload pressure, Reduce obstruction

C. Manage other organ dysfunction

- Oxygenation: Non- or limited Mechanical Ventilation. Reduce metabolic needs: sedation, paralytics. Acidosis and/or metabolic control: Renal replacement therapy

If refractory cardiogenic shock, consider acute mechanical circulatory support

16

Obstructive Shock Overview

Types of Obstructive Shock

- Pulmonary Embolism (PE):** Large clots obstruct pulmonary circulation.
- Cardiac Tamponade:** Fluid accumulates in the pericardium, compressing the heart.
- Tension Pneumothorax:** Air trapped in the pleural space increases intrathoracic pressure, compressing the heart and lungs.
- Aortic Dissection:** A tear in the aortic wall disrupts blood flow.

Mechanisms of Origin

- Pulmonary Embolism:** Obstruction of the pulmonary arteries increases right ventricular afterload, leading to reduced cardiac output.
- Cardiac Tamponade:** Excess fluid restricts heart expansion, reducing ventricular filling and output.
- Tension Pneumothorax:** Increased intrapleural pressure compresses the heart, reducing venous return and cardiac output.
- Aortic Dissection:** Disruption of blood flow from the aorta leads to impaired perfusion of vital organs.

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17

Obstructive Shock Progression, Manifestations

Progression

- Initial obstruction (e.g., embolism, tamponade) leads to increased pressure and decreased venous return.
- Compensatory Stage:** sympathetic activation to maintain cardiac output.
- Decompensatory Stage:** Failure to maintain adequate perfusion leads to tissue hypoxia and organ dysfunction.

Clinical Manifestations

- Pulmonary Embolism:**
 - Sudden dyspnea, pleuritic chest pain, tachypnea, hyperinflation
- Cardiac Tamponade:**
 - Beck's triad: Hypotension, muffled heart sounds, distended jugular veins.
- Tension Pneumothorax:**
 - Traichal deviation, hypotension, decreased breath sounds on the affected side.
- Aortic Dissection:**
 - Severe tearing chest pain, unequal blood pressures between arms.

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18

Obstructive Shock Treatment

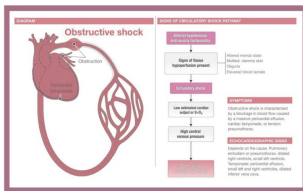
Treatment Strategies

Pulmonary Embolism:
Anticoagulation (e.g., heparin) or thrombolysis for severe cases.
Surgical/Endovascular embolectomy if unstable.

Cardiac Tamponade:
Immediate pericardiocentesis to remove excess fluid.

Tension Pneumothorax:
Needle decompression followed by chest tube insertion.

Aortic Dissection:
Blood pressure control (e.g., beta-blockers) and surgical repair.



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19

SHOCK DISTRIBUTIVO

Vasodilatazione periferica (mediatori umorali)

Sepsi:
Batteriche (gram+, gram-),
Virali (cytomegalovirus, varicella,
ebola)
Fungine (candida, aspergillo),
Mycobatteri (tbc)



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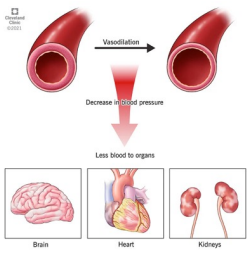
20

SHOCK DISTRIBUTIVO

Vasodilatazione periferica e discrepanza tra letto circolatorio e volume circolante

Anafilassi (reazione IgE mediata)
Punture insetti, alimenti e farmaci

- Triade: IL2, IL6, TNF alpha
- Mast cell (masticati) rilascia: istamina, prostaglandine e trombossani: vasodilatazione



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21

SHOCK DISTRIBUTIVO

Neurogeno
Traumi midollari, (farmaci antipertensivi)
Danno SN autonomo: < resistenze vascolari e alterato tono vagale

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