DEGLI STUDI DI TRIESTE

ANESTESIOLOGIA

Valutazione del paziente critico

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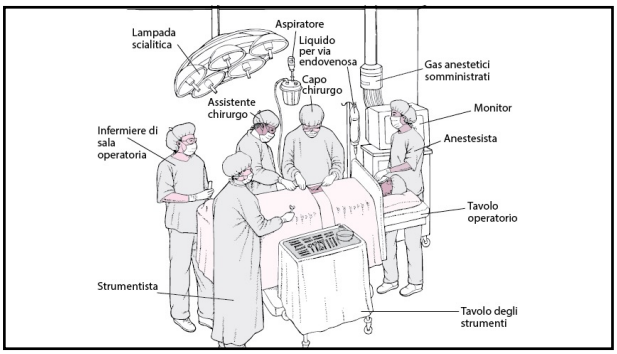

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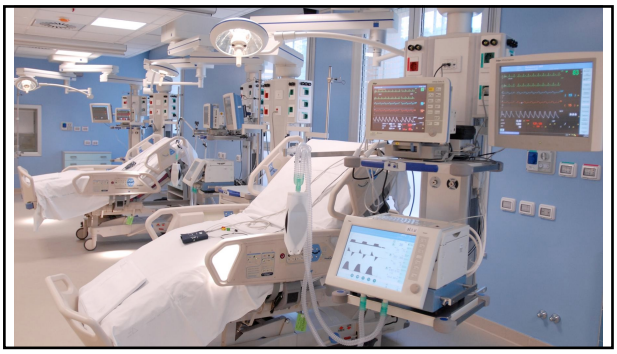

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CHI E' IL PAZIENTE CRITICO?

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

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

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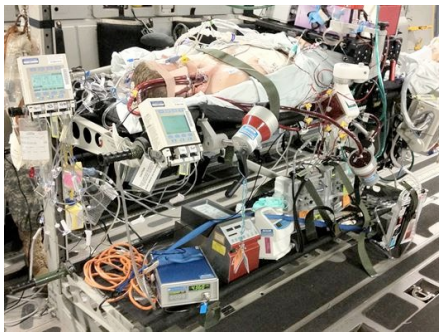

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CRITERI DI GRAVITA'

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

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SHOCK

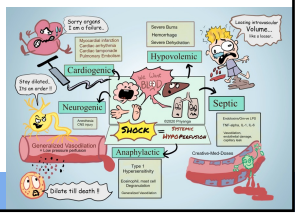
Riduzione della PA associata a sintomi e segni di ipoperfusione tissutale secondaria a:

IPOTENSIONE
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 O_2 e metaboliti
Alterazioni metaboliche cellulari e nel tempo anche d'organo



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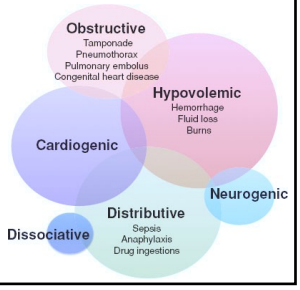
SHOCK

Definizione:

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



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Types of Shock

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

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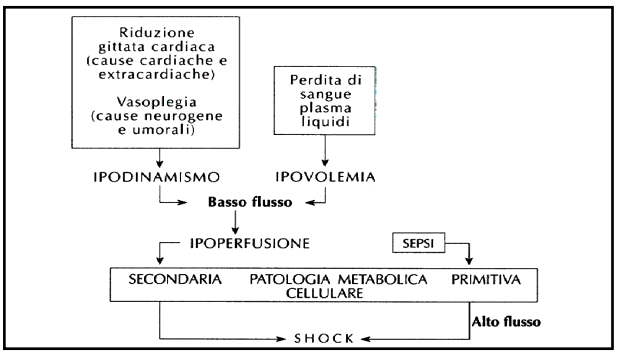

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Tipi di SHOCK

- Puri
- Misti

1. persistere evento patogenetico
2. validita' risposta sistemica
3. 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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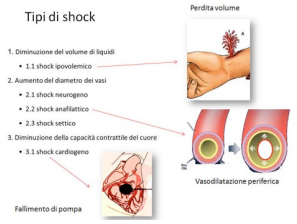
Tipi di SHOCK

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



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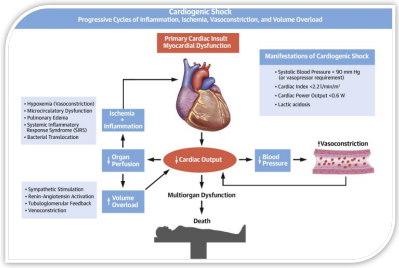
SHOCK CARDIOGENO



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Cardiogenic Shock Overview

Mechanisms of Origin

•Cause: Primary pump failure of the heart due to:
- Acute myocardial infarction (most common)
- Severe heart failure
- Cardiomyopathy, myocarditis
- Valve dysfunction (e.g., mitral regurgitation, aortic stenosis)

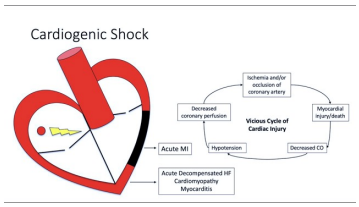
•Pathophysiology:
- Decreased stroke volume → low cardiac output
- Inadequate perfusion of vital organs → tissue hypoxia

Progression

•Initial Stage: Ischemia reduces cardiac contractility.

•Compensatory Stage: Sympathetic activation increases heart rate and systemic vascular resistance.

•Decompensation: Failure of compensatory mechanisms leads to:
- Severe tissue hypoxia
- Metabolic acidosis
- Multiorgan dysfunction syndrome (MODS)



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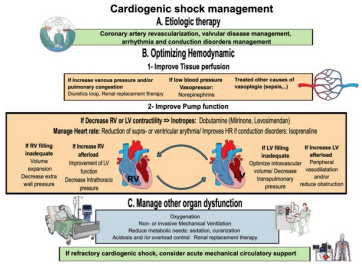
Cardiogenic Shock Manifestations & Treatment

Clinical Manifestations

- **Cardiovascular:** Hypotension, tachycardia, elevated JVP, cold extremities
- **Pulmonary:** Pulmonary edema, dyspnea, hypoxemia
- **Renal:** Oliguria
- **Neurological:** Altered mental status (confusion, lethargy)

Treatment Strategies

- **Initial Stabilization:**
  - **Oxygenation:** Supplemental oxygen or mechanical ventilation.
  - **Hemodynamic Support:**
    - **Inotropes (e.g., dobutamine) for contractility.**
    - **Vasopressors (e.g., norepinephrine) to maintain BP.**
- **Revascularization:**
  - **PCI or CABG if myocardial infarction.**
- **Mechanical Support:**
  - **IABP, VADs, or ECMO in refractory cases.**
- **Treat Underlying Causes:** Valve repair/ replacement, arrhythmia treatment.



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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.

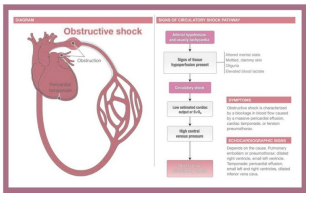
Mechanisms of Origin

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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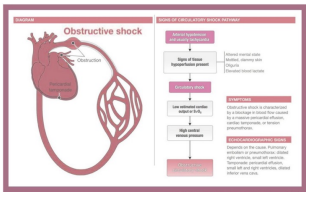
Obstructive Shock Progression, Manifestations

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



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Obstructive Shock Treatment

Treatment Strategies

Pulmonary Embolism:

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

Cardiac Tamponade:

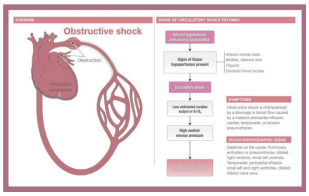
Immediate pericardiocentesis to remove excess fluid.

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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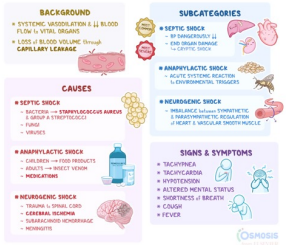
SHOCK DISTRIBUTIVO

Vasodilatazione periferica (mediatori umorali)

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



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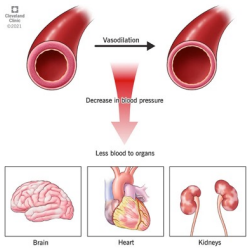
SHOCK DISTRIBUTIVO

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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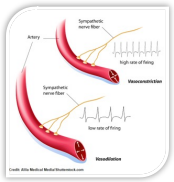

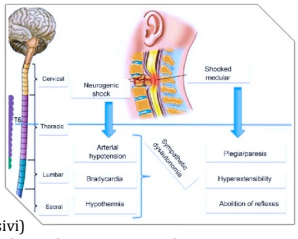

Neurogeno
- Traumi midollari, (farmaci antipertensivi)
- Danno SN autonomo: < resistenze vascolari e alterato tono vagale

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