SHOCK: Le fasi

SHOCK COMPENSATO

SHOCK PROGRESSIVO

SHOCK IRREVERSIBILE

Lo *shock* è una patologia progressiva, che, se non trattata, porta a morte

Se il danno non è rapidamente letale (per esempio emorragia massiva da rottura di un aneurisma aortico), lo *shock* evolve attraverso tre stadi

- 1. **stadio iniziale** non progressivo durante il quale sono attivati meccanismi compensatori riflessi e la perfusione degli organi vitali è mantenuta
- 2. **stadio progressivo** caratterizzato da ipo-perfusione tissutale e peggioramento delle alterazioni circolatorie e metaboliche, tra cui l'acidosi
- 3. **stadio irreversibile** che si verifica dopo che nell'organismo si è avuto un danno cellulare e tessutale così severo che anche se le alterazioni emodinamiche vengono corrette, la sopravvivenza non è più possibile

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COMPENSATORY STAGE

•**Pathophysiology**: Early response to volume loss as the body attempts to maintain cardiac output and tissue perfusion.

•**Mechanisms**:
- **Sympathetic activation**: Release of catecholamines (epinephrine, norepinephrine) increases heart rate and contractility.
- **Vasoconstriction**: Systemic vascular resistance increases to maintain blood pressure.
- **Blood redistribution**: Blood is shunted away from less vital organs (e.g., skin, GI tract) to protect critical organs (heart, brain).

•**Clinical Signs**:
- Tachycardia
- Slightly decreased blood pressure or near-normal
- Cool, clammy skin
- Oliguria (reduced urine output)
- Restlessness or anxiety (due to hypoxia)

•**Outcome**: Reversible if treated early with fluid resuscitation.

2

PROGRESSIVE STAGE

•**Pathophysiology**: Compensatory mechanisms begin to fail, leading to more severe hypoperfusion and metabolic imbalances.

•**Mechanisms**:
- **Prolonged hypoperfusion**: Results in tissue hypoxia, causing a switch to anaerobic metabolism.
- **Lactic acidosis**: Accumulation of lactate leads to worsening acidosis, impairing cellular function.
- **Capillary leakage**: Endothelial damage increases capillary permeability, exacerbating fluid loss into tissues.

•**Clinical Signs**:
- Hypotension (marked decrease in blood pressure)
- Tachycardia remains or worsens
- Cold extremities, cyanosis (bluish discoloration)
- Altered mental status (confusion, lethargy)
- Oliguria or anuria (little to no urine output)

•**Outcome**: May progress to irreversible damage without timely and aggressive intervention.

3

IRREVERSIBLE (REFRACTORY) STAGE

•**Pathophysiology**: Critical organ damage due to prolonged hypoxia and acidosis, leading to irreversible cellular death.

•**Mechanisms**:
- **Severe metabolic acidosis**: Widespread cellular dysfunction leads to failure of organs, including the heart and kidneys.
- **Multiorgan dysfunction syndrome (MODS)**: Loss of function in multiple vital organs.
- **Cardiovascular collapse**: Inability to maintain blood pressure and circulation.

•**Clinical Signs**:
- Profound hypotension (unresponsive to fluid resuscitation or vasopressors)
- Bradycardia or arrhythmias
- Coma or loss of consciousness
- Anuria (complete cessation of urine output)
- Cold, mottled skin

•**Outcome**: Irreversible; death is imminent without intervention, and recovery is rare even with aggressive therapy.

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SHOCK ipovolemico
Fase di compenso

Perdita volume < RV e < precarico < perfusione polmonare e < GC

Risposta neuroendocrina ed emodinamica
> att. Simpatico (recettori alfa1)
> catecolamine
ADH, RAS

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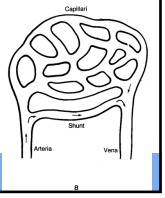
SHOCK ipovolemico
Fase di compenso

- aumento ritorno venoso
- tachicardia (scarica adrenergica) > GC
- iperventilazione (in risposta ipoperfusione) ed aumento profondità respiro
> scambi gassosi
- riduzione perfusione renale e del filtrato glomerulare
RAS – ADH > Na+
oliguria ed aumento volemia



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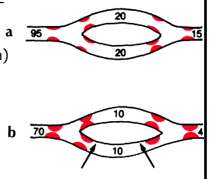
SHOCK ipovolemico
Fase di compenso

Riassorbimento di liquidi dall'interstizio
vasocostrizione periferica riduzione P nel letto capillare
passaggio di acqua ed elettroliti dall'interstizio allo spazio intravascolare (70-150 ml/h)
(refilling* transcapillare con emodiluizione compensatoria)

* riempimento compartimento vascolare a spese di quello interstiziale



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SHOCK ipovolemico
Fase di compenso

- pochi minuti
- mantenimento pressione arteriosa normale
- organi nobili —>buona perfusione cerebrale e miocardica (! PA 70-80 mmHg)
- ! danno circolo periferico : fase dell'**anossia ischemica** (80% micro

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SHOCK IPOVOLEMICO
FASE DI SCOMPENSO

Insufficienza dei meccanismi di compenso metabolico-circolatorio
- mantenuto funzionamento attività metaboliche epatiche
- riduzione resistenze vascolari periferiche
- acidosi metabolica da glicolisi anaerobia e iperproduzione acido lattico
- alterazione membrane cellulari

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SHOCK ipovolemico
Fase di scompenso
Danno circolo periferico

- anossia ischemica (ipoperfusione tissutale) accumulo di metaboliti acidi
- **azione vasodilatatrice sugli sfinteri precapillari ma non sulle venule (> resist. Ph basso)—> STASI EMATICA**
  - riduzione volemia
  - aumento pressione nel sistema capillare
  - passaggio liquido nell' interstizio —> refilling inverso

> IPOVOLEMIA
ANOSSIA STAGNANTE

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SHOCK IPOVOLEMICO
FASE IRREVERSIBILE

- ipossia tissutale ed acidosi metabolica (aumento dei lattati)
- danno endoteliale ed aumento permeabilità
- trasudazione liquidi (plasma, proteine) e danno cellulare con mancata risposta allo stimolo vasocostrittore

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SHOCK
DANNO FUNZIONALE ORGANICO

Alterazione cellulare (da difetto di trasporto O_2 o da suo difetto di utilizzazione) organ dysfunction (MOD)/ organ failure (MOF)

Tempi e modi dipendono da:
- Causa e tipo shock
- Durata e gravita'
- Resistenza a ipossia e ipoperfusione
- Condizioni cliniche base e la "riserva funzionale"

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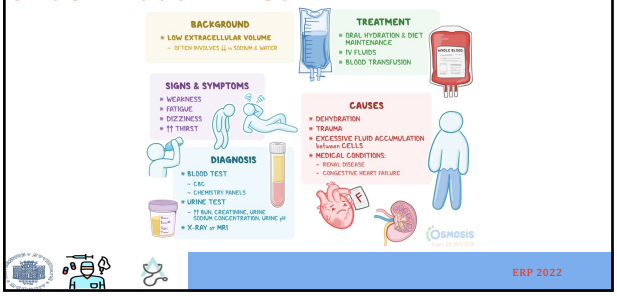
SHOCK IPOVOLEMICO



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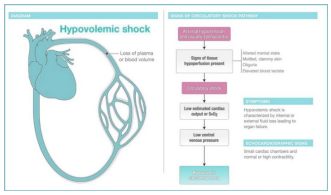
MECHANISMS OF ORIGIN

- **External fluid loss**: Hemorrhage (trauma, surgery), dehydration (vomiting, diarrhea, burns).
- **Internal fluid shift**: Third-spacing (e.g., pancreatitis, bowel obstruction).
- **Pathophysiology**: Reduced intravascular volume leads to decreased preload, reduced stroke volume, and decreased cardiac output, compromising tissue perfusion.



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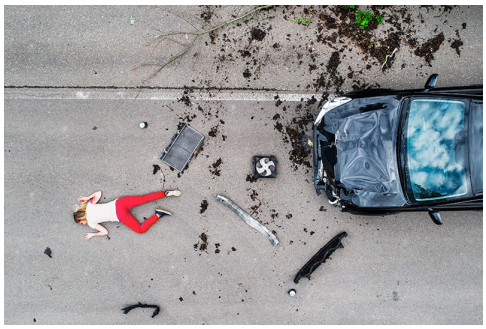

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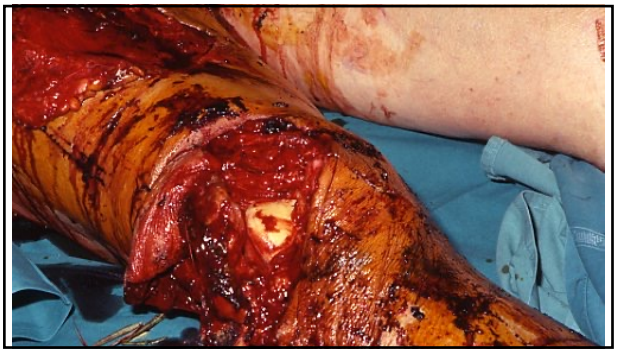

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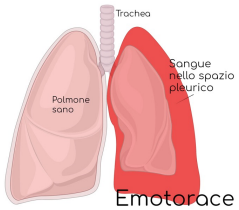
SHOCK IPOVOLEMICO

da perdita interna

- di sangue in toto: emotorace, emoperitoneo, ematoma retroperitoneale, ematomi delle parti molli
- della sola componente liquida: ascite, occlusione intestinale



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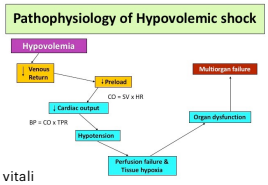
SHOCK IPOVOLEMICO

Fisiopatologia: risposte

- Neuroendocrina
- Immunologica
- Emodinamica (cardiovascolare)
- Metabolica



a. mantenere adeguata perfusione organi vitali
b. ripristinare volemia adeguata
c. facilitare mobilizzazione e utilizzazione substrati energetici

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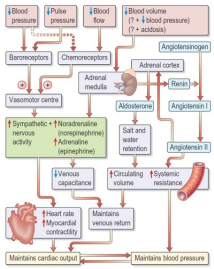
RISPOSTA NEUROENDOCRINA

Attivazione recettori in risposta a sollecitazioni
(ipovolemia, dolore, ipossiemia, ipertermia)

Barocettori (seno carotideo, arco aortico): PA
volocettori (atriali): V riempimento atriale
chemocettori (glomo): pO2, CO2
Osmocettori (ipotalamo): osmolarita' plasmatica
Nocicettori (cutanei, viscerali, scheletrici)



The sympatho-adrenal response to shock showing the effect of increased catecholamines on the left of the diagram and the release of angiotensin and aldosterone on the right. Both mechanisms result in maintaining the cardiac output in shock.

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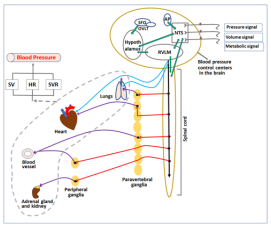
NEUROENDOCRINE RESPONSE

- **Sympathetic nervous system activation:**
  - Release of catecholamines (e.g., epinephrine, norepinephrine) to increase heart rate and contractility.
- **Renin-angiotensin-aldosterone system (RAAS) activation:**
  - **Renin release** → Angiotensin II causes vasoconstriction, while aldosterone promotes sodium and water retention.
- **Antidiuretic hormone (ADH)** secretion:
  - Increased water reabsorption in kidneys to expand intravascular volume.



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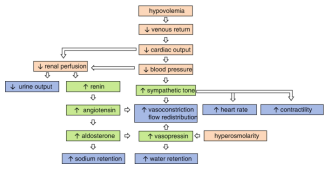
HAEMODYNAMIC RESPONSE

- **Reduced preload**: Decreased venous return to the heart.
- **Compensatory tachycardia**: Increased heart rate to maintain cardiac output.
- **Peripheral vasoconstriction**: Increases systemic vascular resistance to maintain blood pressure.
- **Decreased stroke volume**: Low cardiac output leads to tissue hypoperfusion.



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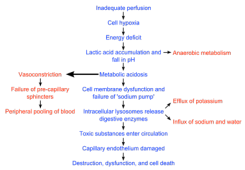
METABOLIC RESPONSE

•**Anaerobic metabolism**: Oxygen deprivation forces tissues to switch to anaerobic metabolism.

•**Lactic acidosis**: Accumulation of lactate due to anaerobic metabolism leads to metabolic acidosis.

•**Energy depletion**: Cellular ATP production is compromised, impairing organ function.



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PROGRESSION OF HYPOVOLEMIC SHOCK

Initial Stage:
•Fluid loss results in reduced intravascular volume, causing decreased preload and cardiac output.
•Sympathetic activation tries to compensate with tachycardia and vasoconstriction.

Compensatory Stage:
•Neuroendocrine response kicks in to maintain perfusion to vital organs (heart, brain).
•Peripheral organs experience ischemia as blood is shunted to vital organs.

Decompensatory Stage:
•Prolonged hypoperfusion leads to lactic acidosis, organ dysfunction, and multiorgan failure.
•If untreated, progression to irreversible shock and death.

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CLINICAL MANIFESTATIONS OF HYPOVOLEMIC SHOCK

Cardiovascular:

Hypotension: Low blood pressure due to reduced intravascular volume.
Tachycardia: Compensatory mechanism to maintain cardiac output.
Weak, thready pulse: Due to reduced stroke volume.

Skin:

Pale, cool, clammy skin: Result of peripheral vasoconstriction and poor perfusion.

Renal:

Oliguria: Low urine output due to reduced renal perfusion.

Neurological:

Altered mental status: Confusion or lethargy due to cerebral hypoperfusion.

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TREATMENT OF HYPOVOLEMIC SHOCK

Fluid Replacement:
Crystalloids (e.g., normal saline, lactated Ringer's) as first-line therapy.
Blood transfusion for hemorrhagic shock.
Rapid volume expansion to restore tissue perfusion and oxygen delivery.

2. Hemodynamic Support:

Vasopressors (e.g., norepinephrine) may be needed if hypotension persists after fluid resuscitation, but only after adequate volume replacement.

3. Monitoring:

Invasive hemodynamic monitoring (central venous pressure, arterial pressure) to guide fluid therapy.
Frequent assessments: Mental status, urine output, lactate levels, and vital signs.

4. Address Underlying Cause:

Control bleeding (e.g., surgical intervention for trauma).
Treat dehydration or underlying disease (e.g., infections).

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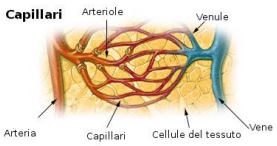
Perfusione tissutale e pressione arteriosa sistemica
Fattori determinanti

PA = GC X R

- Gittata cardiaca (GC) : GS X FC
- Preload (riempimento V)
- Contrattilita'

- Resistenze periferiche (RP)
- Tono, lunghezze e diametro vasi
- Viscosita'sangue



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