Der Pädiatrische Schock

Erkennen und Management

Dr. Anna Aichinger
Pädiatrische Intensivstation (PICU)
MedUni Wien

Fallpräsentation

- 10 Jahre, 50 kg, männlich
- keine Vorerkrankungen

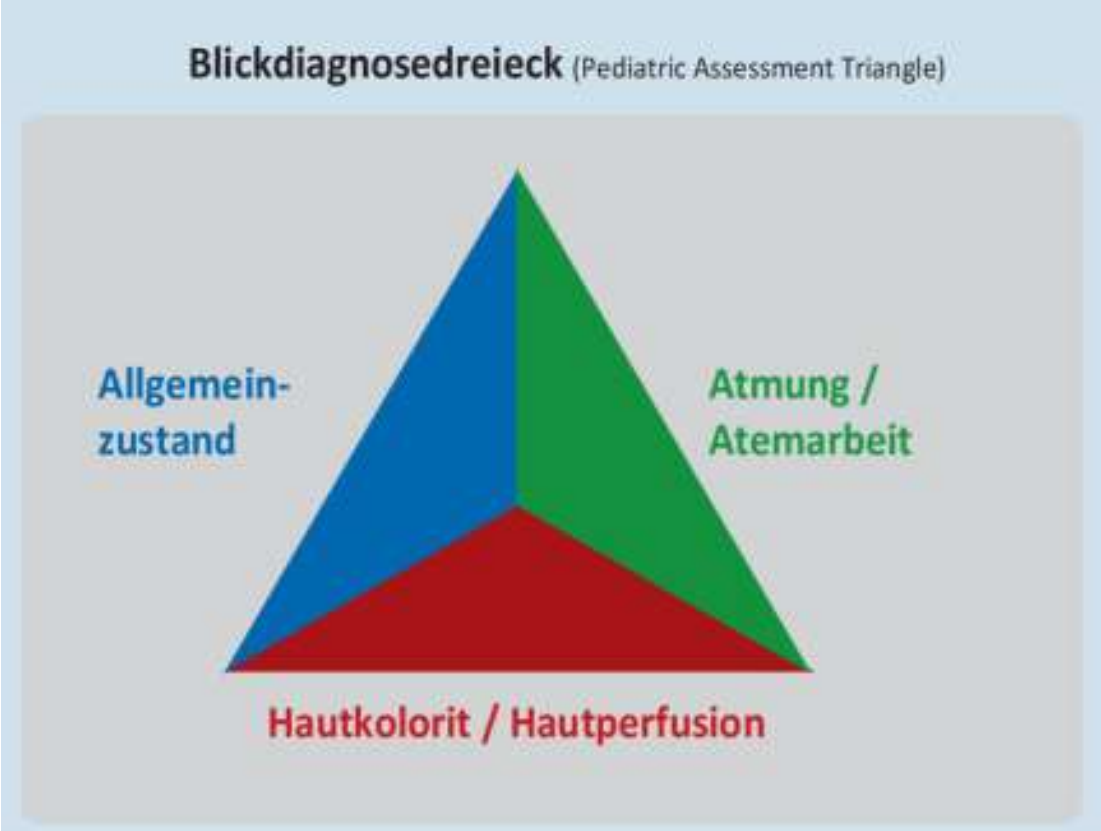
- Kinderambulanz Wien
  - Fieber seit 3 Tagen
  - guter Allgemeinzustand, Rachen etwas gerötet
  - viraler Infekt, Konjunktivitis links
  - Empfehlung: symptomatische Therapie

- darauffolgender Tag abends Ambulanz

Ersteinschätzung

Vigilanz vermindert

nicht ansprechbar



spO2 78%

Lippenzyanose

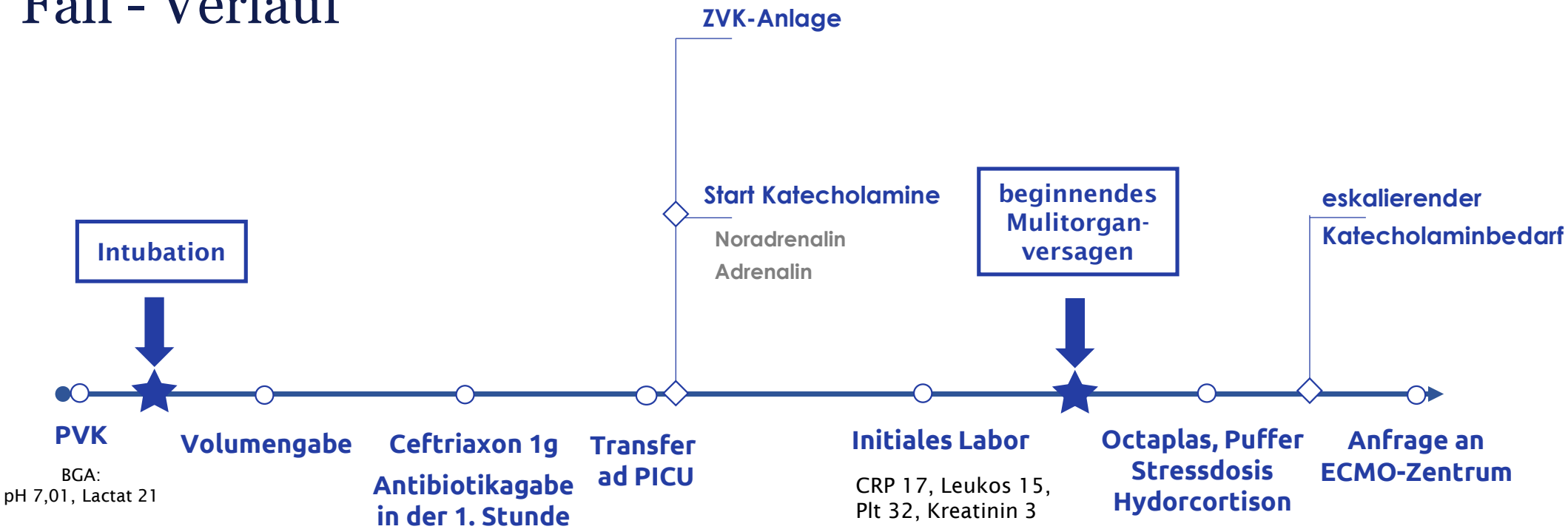
blass, zentralisiert

Blutdruck nicht messbar

→ kritisch krankes Kind


Septic Shock

Fall - Verlauf



Verlauf – PICU AKH


Septic Shock

Sepsis

Sepsis is a major cause of morbidity and mortality in the paediatric population and can be very challenging to diagnose and manage

Practice Guideline > Pediatr Crit Care Med. 2020 Feb;21(2):e52-e106.

doi: 10.1097/PCC.0000000000002198.

Surviving Sepsis Campaign International Guidelines for the Management of Septic Shock and Sepsis-Associated Organ Dysfunction in Children

SEPTISCHER SCHOCK

ist ein **KLINISCHER NOTFALL**

!

For every hour a child remains in septic shock the mortality risk doubles. Care delivered in the first hour after presentation or sepsis identification is crucial in ensuring the optimum outcome for the patient.

Welt-Sepsis-Tag

ROBERT KOCH INSTITUT

rki.de/sepsis

Paediatric Sepsis

Recognition: A child with suspected or proven infection AND at least 2 of the following:

- Core temperature < 36°C or > 38°C (observed or reported in previous 4 hours)
- Inappropriate tachycardia (Refer to National PEWS)
- Altered mental state (including: sleepiness / irritability / lethargy / floppiness)
- Reduced peripheral perfusion / prolonged capillary refill / cool or mottled peripheries

Alert

Hypotension is a late, and often terminal, sign in paediatric septic shock.

Erhöhtes Sepsis-Risiko

Reduce Threshold:

Some children are at higher risk of sepsis. You may co
above. These include, but are not restricted to;

- Infants < 3/12
- Immunosuppressed / Immunocompromised / ch
- Recent surgery
- Indwelling devices / lines
- Complex neurodisability / Long term conditions (may not present with high PEWS but observations may vary from their baseline)
- High index of clinical suspicion (tachypnoea, rash, leg pain, biphasic illness, poor feeding)
- Significant parental concern


A
Airway
B
Breathing
C
Circulation
D
Disability
E
Exposure

Think is this SEPSIS? If yes

Management algorithm

DO

0 min

15 min
ideal

60 min
acceptable

Think is this SEPSIS? If yes

DO

0 min

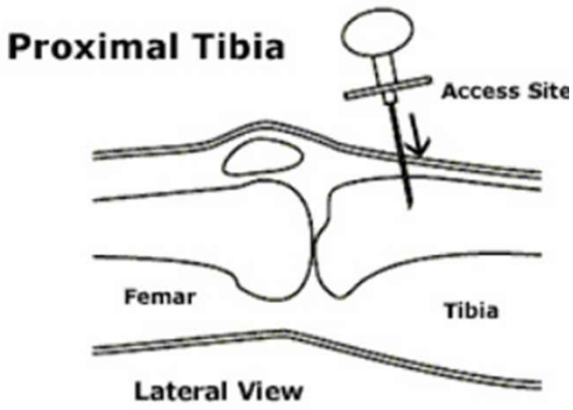
15 min
ideal

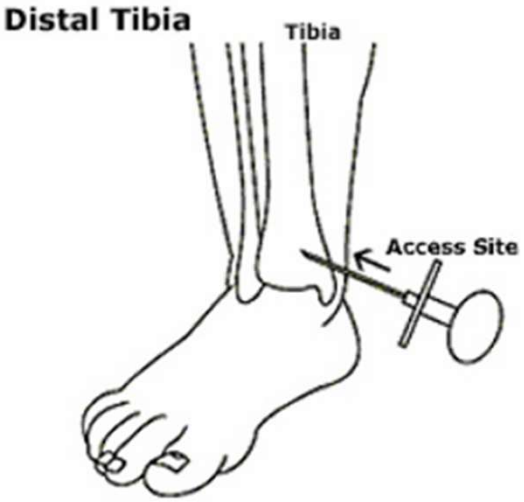
60 min
acceptable

Respond with Paediatric Sepsis 6 within 1 hour:

1. **Give high flow oxygen**
2. **Obtain intravenous or intraosseous access and take blood tests:**
   - Blood cultures
   - Blood glucose - treat low blood glucose
   - Blood lactate (or gas)


Right leg


Proximal Tibia
Access Site
Femar
Tibia
Lateral View


Distal Tibia
Tibia
Access Site

Think is this SEPSIS? If yes

DO

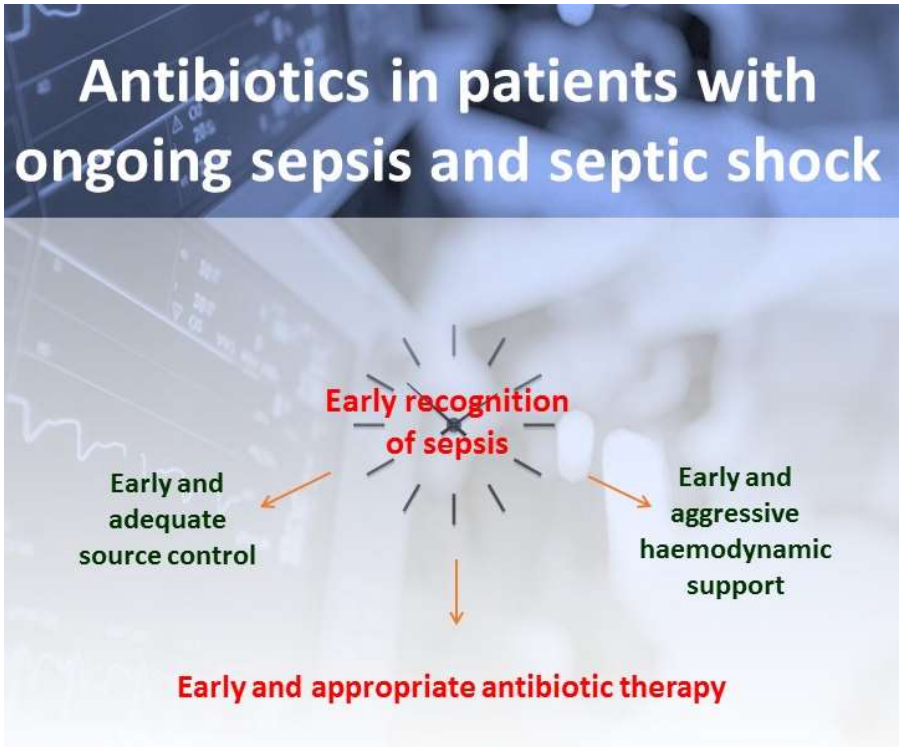
0 min

15 min
ideal

60 min
acceptable

Respond with Paediatric Sepsis 6 within 1 hour:

1. **Give high flow oxygen**
2. **Obtain intravenous or intraosseous access and take blood tests:**
   - Blood cultures
   - Blood glucose - treat low blood glucose
   - Blood lactate (or gas)
3. **Give IV or IO antibiotics:** Broad spectrum as per local policy



Alert

In septic shock, do not delay antibiotic administration for specimen collection or testing beyond ONE hour.

Think is this SEPSIS? If yes

DO

0 min

15 min
ideal

60 min
acceptable

Respond with Paediatric Sepsis 6 within 1 hour:

1. **Give high flow oxygen**
2. **Obtain intravenous or intraosseous access and take blood tests:**
   - Blood cultures
   - Blood glucose - treat low blood glucose
   - Blood lactate (or gas)
3. **Give IV or IO antibiotics:** Broad spectrum as per local policy

If shocked:

4. **Consider fluid resuscitation**:
   - Titrate 20 ml/kg isotonic fluid over 5 - 10 min and repeat if neces
   - Aim to reverse shock – trend to normal heart rate, BP and periph
   - assess for fluid overload after ≥ 40 ml/kg fluids.
   - If no signs of fluid overload and remains shocked titrate further 20mls/kg fluid
5. **Consider inotropic support early:**
   - Adrenaline (reconstitute whilst administering 3rd fluid bolus. 0.3mg/kg in 50mls 5% dextrose. Commence 1ml/hr = 0.1mic/kg/min).
   - Can be given via peripheral IV or IO access
6. **Involve senior clinicians / specialists early**
   - Discuss with PICU if inotropes commenced

Prepare for **intubation/ventilation**

- May be required at any step

Alert

⚠ Child may arrest from cardiovascular collapse on RSI /intubation.

Consider hydrocortisone for fluid refractory, catecholamine resistant shock and suspected absolute adrenal insufficiency.

Rapid estimates: IM/IV

0 – 6 months	12.5 mg	6 months – 5 years	25 mg
5 – 10 years	50 mg	> 10 years	100 mg

OR 75-100 mg/mg/m2 IV

PICU

Poor response to therapy

-Check fluid responsiveness with colloids: can persist beyond the initial period
-Consider transfusion if haemoglobin < 10g/dl in children & < 12 g/dl in neonates
-Correct coagulopathy with FFP/Cryoprecipitate
-Repeat USCOM to optimise inotropes
-Double check other co-morbidities
    Unidentified source with poor source control: necrotic tissue
    Ammonia and Amylase/Lipase
    Consider central line removal if suspected a source at initial presentation
    Draining Pleural effusions if suspected to be infective
-Echocardiogram for peri-cardial effusions/ systolic or diastolic function and end-diastolic volume
-Pneumothorax
-Optimising iCa particularly in neonates
-TFT particularly in neonates and children with T21
-Measure IAP (Increase MAP/diuresis if > 12 mmHg or consider peritoneal centesis/ laparotomy if > 20 mmHg
-CRRT particularly with purpura and fluid over-load
-Consider white cell transfusion in severe neutropenia
-Consider plasma exchange in multi-organ failure with severe coagulopathy
-Consider ECMO (see ECMO guideline)

Persistent Catecholamine-resistant shock?

Evaluate Pericardial Effusion or Pneumothorax,
Maintain IAP < 12mmHg

Refractory Shock?

ECMO

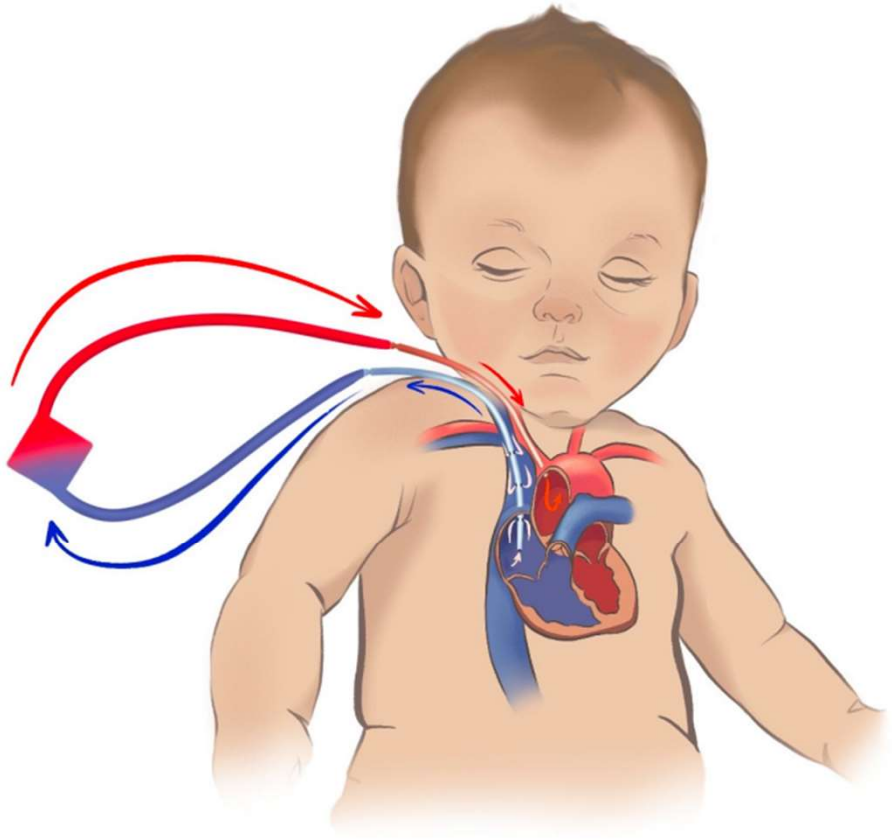
Journal of
Clinical Medicine

MDPI

Review

Extracorporeal Membrane Oxygenation for Septic Shock in Adults and Children: A Narrative Review

Lars Mikael Broman [1,2,*], Olga Dubrovskaja [3] and Martin Balik [4]



Key principles in managing severe sepsis or septic shock

1. **Early recognition**
2. **Rapid vascular access:** Intravenous (IV) or intraosseus (IO) access within 5 minutes
3. **Empiric antibiotic therapy:** as soon as possible after access obtained
4. **Rapid, judicious, fluid resuscitation:** 20ml/kg fluid boluses of isotonic crystalloids (e.g. Normal saline or Plasma-Lyte 148) or albumin 4%
5. **Early initiation of inotropes** via peripheral access if shocked and not fluid responsive. Transfer to PICU as soon as possible
6. **Source control (if possible)**: For example, to operating theatre as soon as stabilised if suspected abdominal source

SCHOCK ist ein **KLINISCHER NOTFALL**

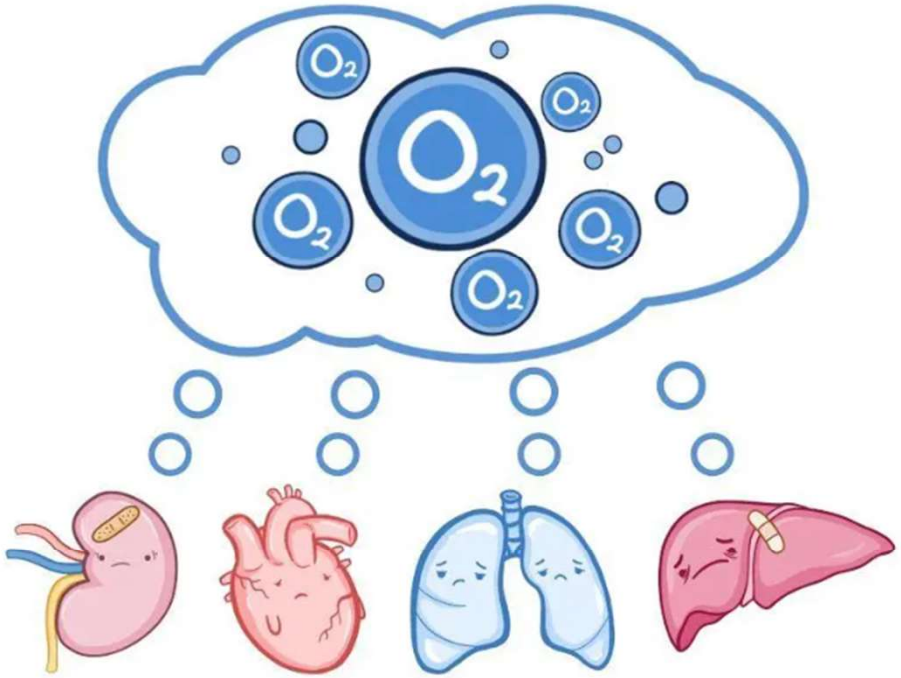
frühzeitiges Erkennen und **frühzeitiges Management**

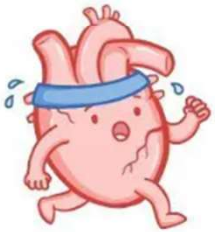
verbessern das Outcome

SHOCK
LIFE-THREATENING CONDITION

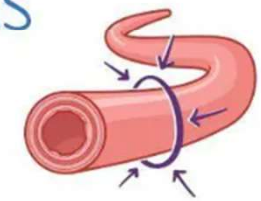
SHOCK

* CIRCULATORY FAILURE that IMPAIRS DELIVERY of OXYGEN & NUTRIENTS to PERIPHERAL TISSUES

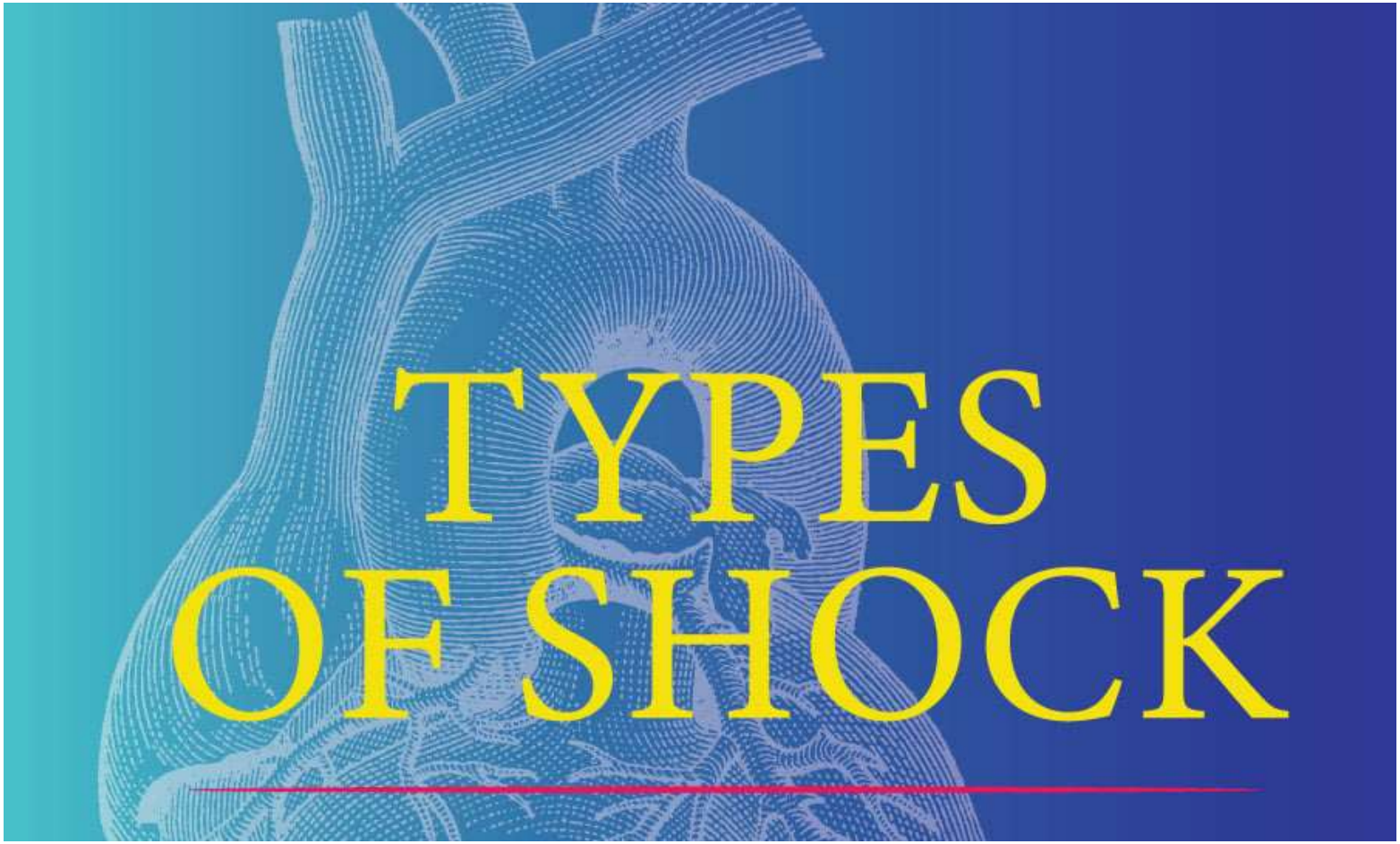




* EARLY STAGES
  ↳ COMPENSATORY MECHANISMS
  |
  can fail
  ↓



* ORGAN FAILURE & DEATH


TYPES
OF SHOCK

Frage ans Auditorium

Wie viele Schockformen gibt es?

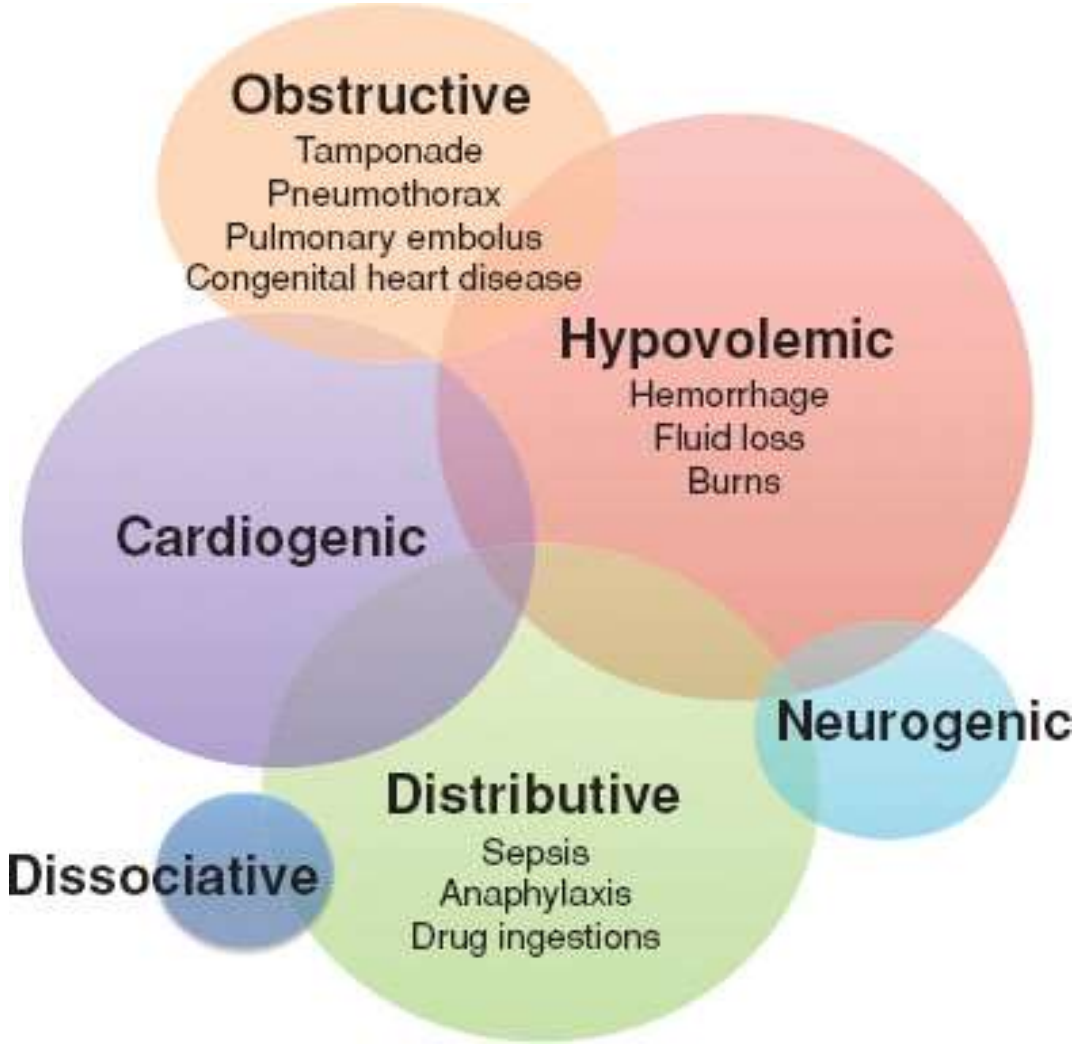
A) vier

B) fünf

C) sechs

D) sieben

E) acht

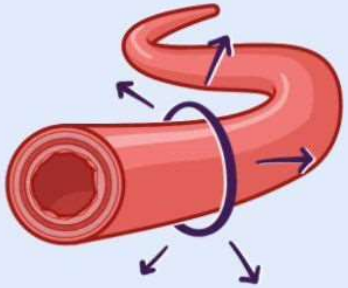

Obstructive
Tamponade
Pneumothorax
Pulmonary embolus
Congenital heart disease
Hypovolemic
Hemorrhage
Fluid loss
Burns
Cardiogenic
Neurogenic
Distributive
Sepsis
Anaphylaxis
Drug ingestions
Dissociative

SHOCK

LIFE-THREATENING CONDITION

DISTRIBUTIVE

EXCESSIVE VASODILATION



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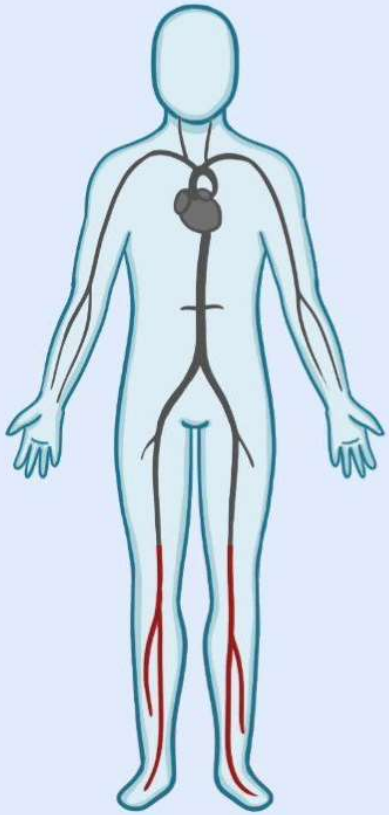
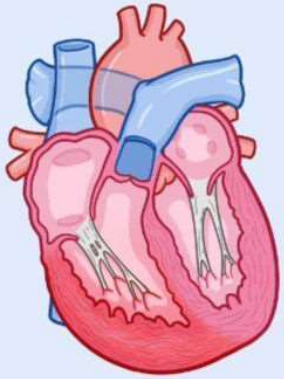
IMPAIRED BLOOD FLOW DISTRIBUTION

HYPOVOLEMIC



CARDIOGENIC

COMPROMISE of MYOCARDIAL PERFORMANCE

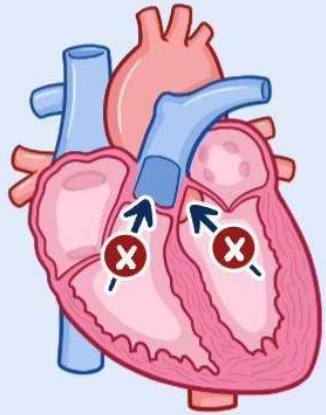


↓

↓↓ OUTPUT

OBSTRUCTIVE

OBSTRUCTION
- FILLING HEART
- EJECTING into GREAT VESSELS



↓

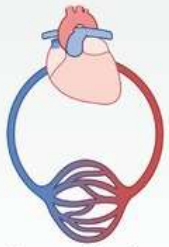
↓↓ OUTPUT

Classification of Shock

Volume

Shift — Distributive shock

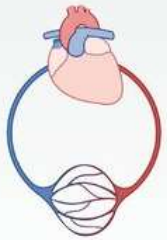
Septic	Capillary leakage
Anaphylactic Anaphylactoid Neurogenic	Vascular tone dysregulation



Vasodilation

Loss — Hypovolemic shock

Hemorrhagic (traumatic or nontraumatic)	Blood (whole)
Nonhemorrhagic (nontraumatic)	Body fluids (e.g., GI loss)
Nonhemorrhagic (traumatic)	Plasma (e.g., from burns)

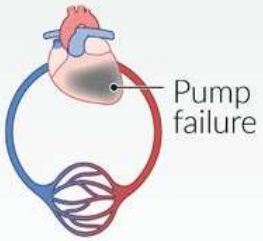


Hypovolemia

Output

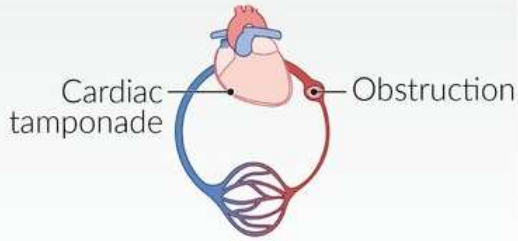
Cardiac — Cardiogenic shock

Myocardial causes	Myocardium
Arrhythmias	Conduction system
Valvular heart disease	



Extracardiac — Obstructive shock

Impaired diastolic filling	E.g., cardiac tamponade
↑ Ventricular afterload	E.g., massive PE
Obstruction of venous return	E.g., tension pneumothorax



SCHOCK ist ein **KLINISCHER NOTFALL**

frühzeitiges Erkennen und **frühzeitige Therapie**

verbessern das Outcome

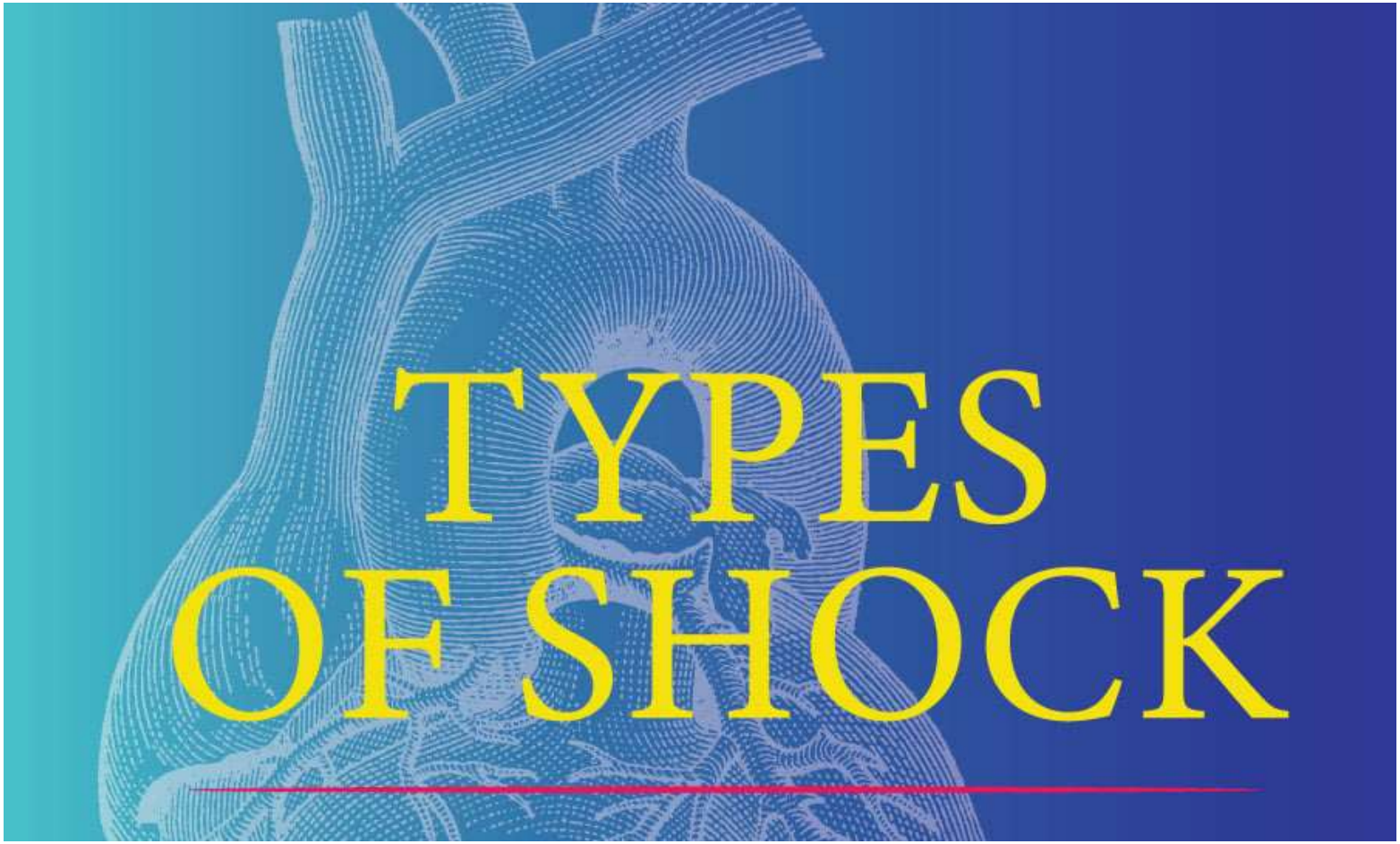
Frage ans Auditorium

Was sind frühe Zeichen von Schock?

Zeichen von Schock

- **Frühe Zeichen**
  - krank wirkend
  - Tachykardie > 98 P. (außer bei Hypothermie)
  - Tachyponoe
  - RKZ > 2 sec oder flash capillary refill
  - abnormaler Pulsdruck
    - schmaler Pulsdruck – Herzversagen
    - weiter Pulsdruck – septischer oder vasodilatatorischer Schock

- **Späte Zeichen**
  - arterielle Hypotension
  - Anionenlücke bei metabolischer Azidose > 16 mEq/L
  - erhöhtes Laktat > 2 mmol/L


TYPES
OF SHOCK

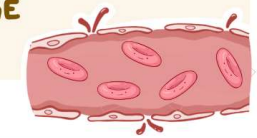
DISTRIBUTIVE

Vasodilatory-↓↓ SVR

-septic shock/SIRS/TSS
- Anaphylaxis
- neurogenic shock
- Drug/toxin
- Addisonian crisis

BACKGROUND

* SYSTEMIC VASODILATION & ↓↓ BLOOD FLOW to VITAL ORGANS
* LOSS of BLOOD VOLUME through **CAPILLARY LEAKAGE**



CAUSES

* SEPTIC SHOCK

- ~ BACTERIA ⟶ **STAPHYLOCOCCUS AUREUS** & GROUP A STREPTOCOCCI
- ~ FUNGI
- ~ VIRUSES

* ANAPHYLACTIC SHOCK

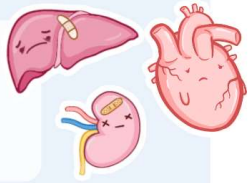
- ~ CHILDREN ⟶ FOOD PRODUCTS
- ~ ADULTS ⟶ INSECT VENOM
- ~ **MEDICATIONS**

* NEUROGENIC SHOCK

- ~ TRAUMA to SPINAL CORD
- ~ **CEREBRAL ISCHEMIA**
- ~ SUBARACHNOID HEMORRHAGE
- ~ MENINGITIS

SUBCATEGORIES

* SEPTIC SHOCK

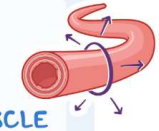


- ~ BP DANGEROUSLY ↓↓
- ~ END ORGAN DAMAGE
  ↳ CRYPTIC SHOCK

* ANAPHYLACTIC SHOCK

- ~ ACUTE SYSTEMIC REACTION to ENVIRONMENTAL TRIGGERS

* NEUROGENIC SHOCK



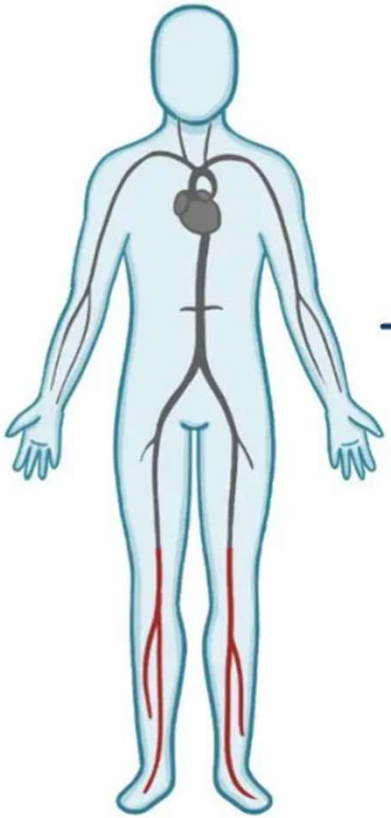
- ~ IMBALANCE between SYMPATHETIC & PARASYMPATHETIC REGULATION of HEART & VASCULAR SMOOTH MUSCLE

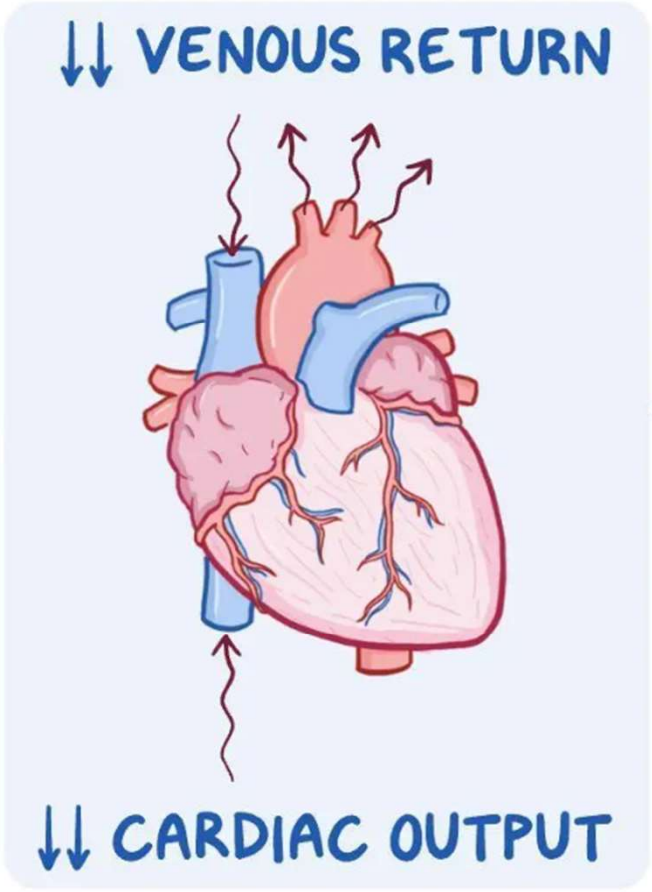
SIGNS & SYMPTOMS

* TACHYPNEA
* TACHYCARDIA
* HYPOTENSION
* ALTERED MENTAL STATUS
* SHORTNESS of BREATH
* COUGH
* FEVER

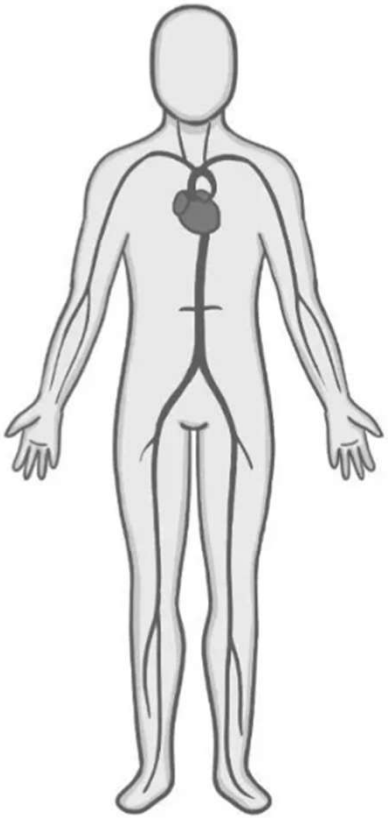
HYPOVOLEMIC SHOCK
LIFE-THREATENING CONDITION

↓↓ INTRAVASCULAR VOLUME





SHOCK



BACKGROUND

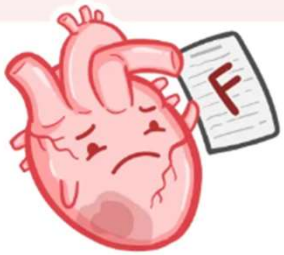
* LOW EXTRACELLULAR VOLUME
  ~ OFTEN INVOLVES ↓↓ in SODIUM & WATER

TREATMENT

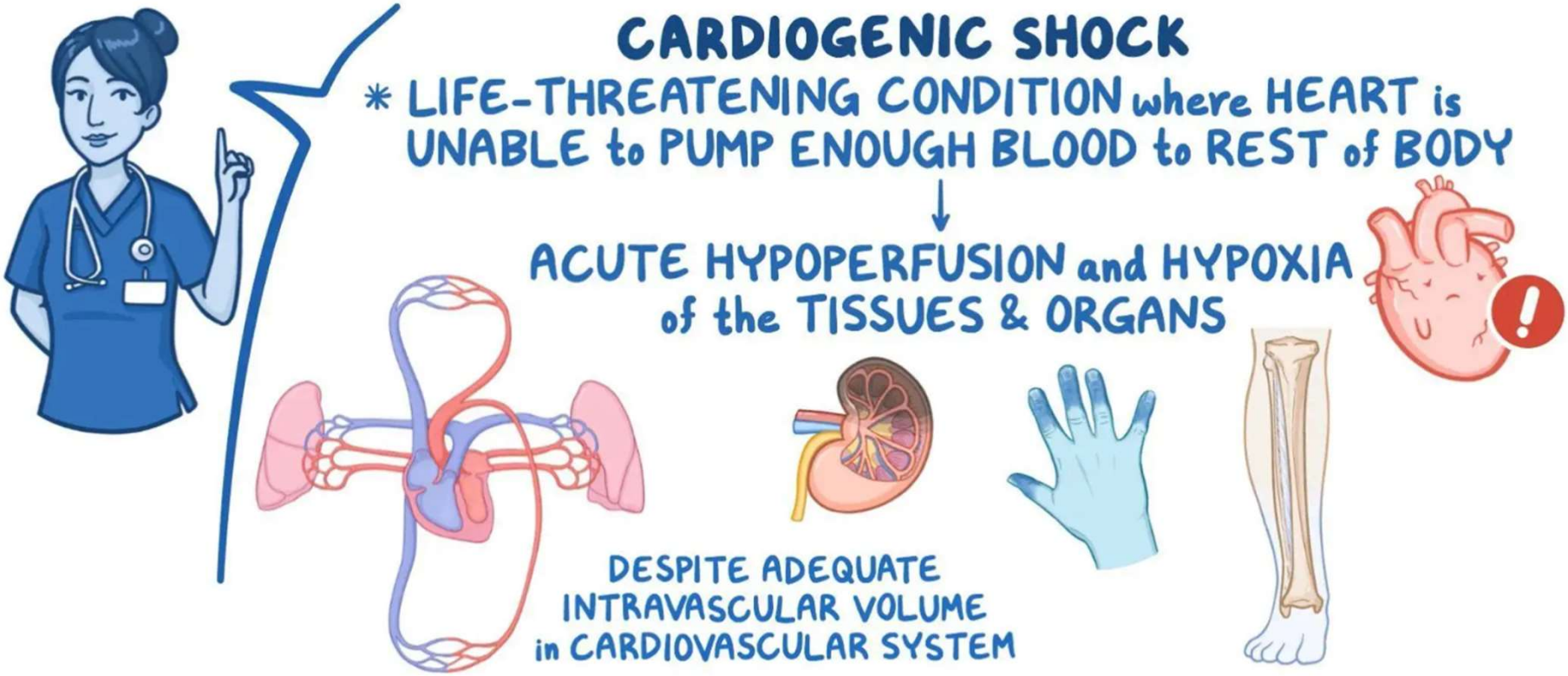
* ORAL HYDRATION & DIET MAINTENANCE
* IV FLUIDS
* BLOOD TRANSFUSION

CAUSES

* DEHYDRATION
* TRAUMA
* EXCESSIVE FLUID ACCUMULATION between CELLS
* MEDICAL CONDITIONS:
  ~ RENAL DISEASE
  ~ CONGESTIVE HEART FAILURE



Cardiogenic Shock



Erkennen:

- RKZ verlängert, erhöhtes Laktat

Management

- Katecholamine, Ursache beheben

Identify cause of CS

Acute myocardial infarction

Cardiac arrest

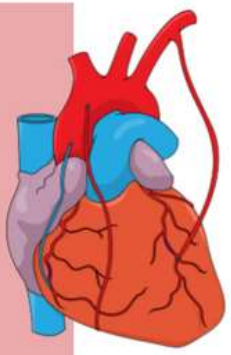
Ventricular / Supraventricular arythmia

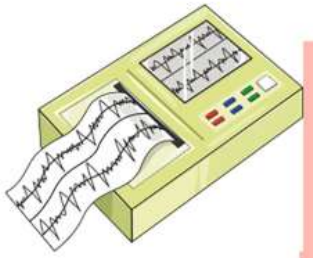
Conduction disorders

Infectious disease

Iatrogenesis

Non-compliance





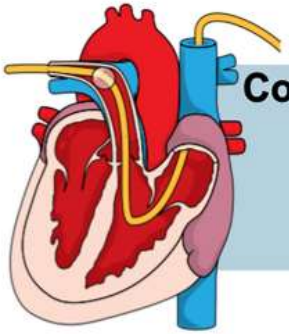
Key investigations for etiological assessment

12-lead ECG, X-Ray, Laboratory tests

Transthoracic +/- transoesophageal echocardiography

Lung and pleural ultrasound

Tomography scan for Aortic syndrome or Pulmonary embolism



Considering invasive hemodynamic monitoring

Arterial catheter

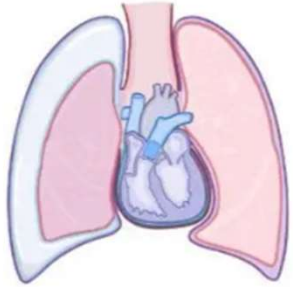
Transpulmonary thermodilution

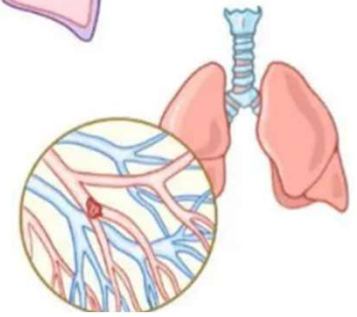
Discussed pulmonary artery catheterization

Obstructive shock

OBSTRUCTIVE SHOCK

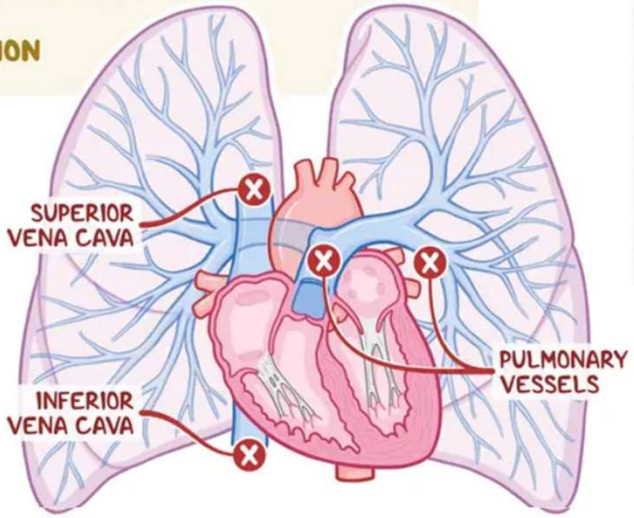
* MECHANICAL OBSTRUCTION PREVENTS HEART from FILLING PROPERLY or PUMPING enough BLOOD through CARDIOVASCULAR SYSTEM
  - ↓ AMOUNT of OXYGENATED BLOOD that REACHES TISSUES
    - → ACUTE HYPOPERFUSION
    - → TISSUE HYPOXIA





BACKGROUND

* ANATOMICAL OBSTRUCTION of the GREAT VESSELS of the HEART
  ↳ LEADS to ↓↓ VENOUS RETURN, ↑↑ AFTERLOAD, & ↓↓ CARDIAC OUTPUT
* LIFE-THREATENING CONDITION



SIGNS & SYMPTOMS

* RESPIRATORY DISTRESS
* TACHYCARDIA
* HYPOTENSION
* TACHYPNEA
* AIR HUNGER
* CHEST PAIN
* DILATED & ENGORGED NECK VEINS

CAUSES

* BLOCKAGE of the PULMONARY VASCULAR SYSTEM → ALTERED BLOOD FLOW from RIGHT to LEFT
  ~ SIGNIFICANT PULMONARY EMBOLISM
  ~ SEVERE PULMONARY HYPERTENSION
* EXTRINSIC MECHANICAL COMPRESSION of the GREAT VESSELS → ALTERED CARDIAC OUTPUT
  ~ TENSION PNEUMOTHORAX
  ~ PERICARDIAL TAMPONADE
  ~ RESTRICTIVE CARDIOMYOPATHY
  ~ CONSTRICTIVE PERICARDITIS

General Treatment Principles

	IV Fluids (↑ CVP)	Vasopressors (↑ SVR)	Inotropes (↑ Contractility)
Hypovolemic	+	Temporary use only	-
Distributive	+	+	+/-
Cardiogenic	-	-	+
Obstructive	+/-	+/-	+/-

Ziele der Schocktherapie

- **Normalisierung** des klinischen, hämodynamischen und biochemischen Derangements

- **Klinisch**
  - Nicht-toxisches Aussehen, adäquater neurologischer Zustand
  - Atemfrequenz, Temperatur, RKZ < 2 sec
  - Harnrate > 1ml/kg/h

- **Hämodynamik und Sauerstoffverbrauch**
  - Herzfrequenz, Perfusionsdruck/Blutdruck
  - SvO2 Sättigung >70%
  - Verbesserter Herzindex (Cardiac index CI)

- **Biochemisch**
  - Anionenlücke < 16 mEq/L
  - Laktat < 2 mmol/L
  - Normoglykämie

Team Sport

SCHOCK ist ein **KLINISCHER NOTFALL**

frühzeitiges Erkennen und **frühzeitige Therapie**

verbessern das Outcome

SCHOCK - Take home message

- Rasches Erkennen
  - Klinische Zeichen
- Rasche Therapieeinleitung
  - → ZEIT!!!

- Schockformen
- Therapieziel - Normalisierung

Vielen Dank für Ihre Aufmerksamkeit

Dr. Anna Aichinger

Universitätsklinik für Kinder- und Jugendheilkunde, Pädiatrische Intensivstation (PICU)

anna.a.aichinger@meduniwien.ac.at