Shock,MOF,SIRS,sepsis, compartment syndrome

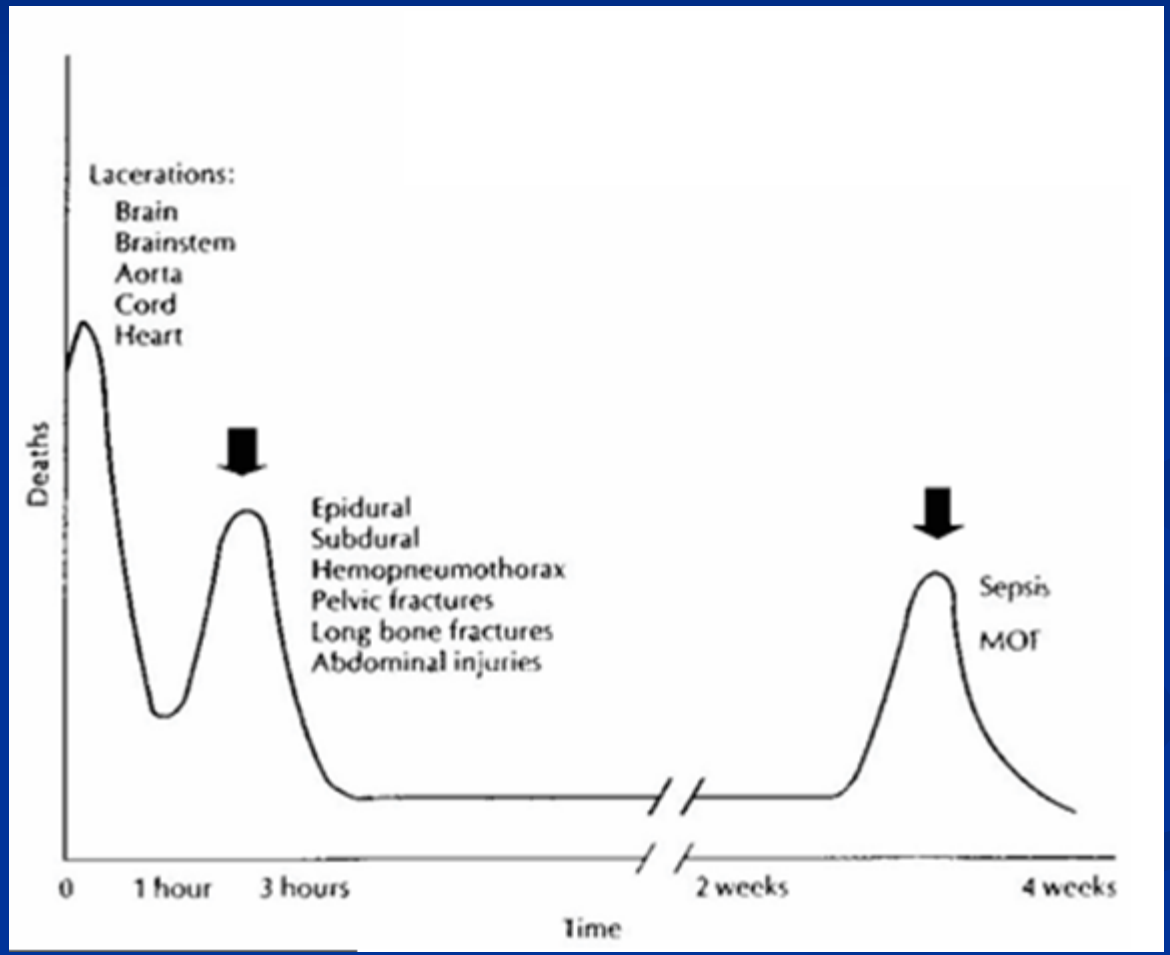
Klinika úrazové chirurgie
FN Brno-Bohunice

Shock

- Major cause of death of critically ill patients
- Condition in which the cardiovascular system fails to perfuse tissues adequately
- Life threatening decrease of blood perfusion in internal organs, systemic state of hypoperfusion
- Imbalance between demand and supply of oxygen for organ tissues
- Centralisation of volume circulation - brain, heart, lungs

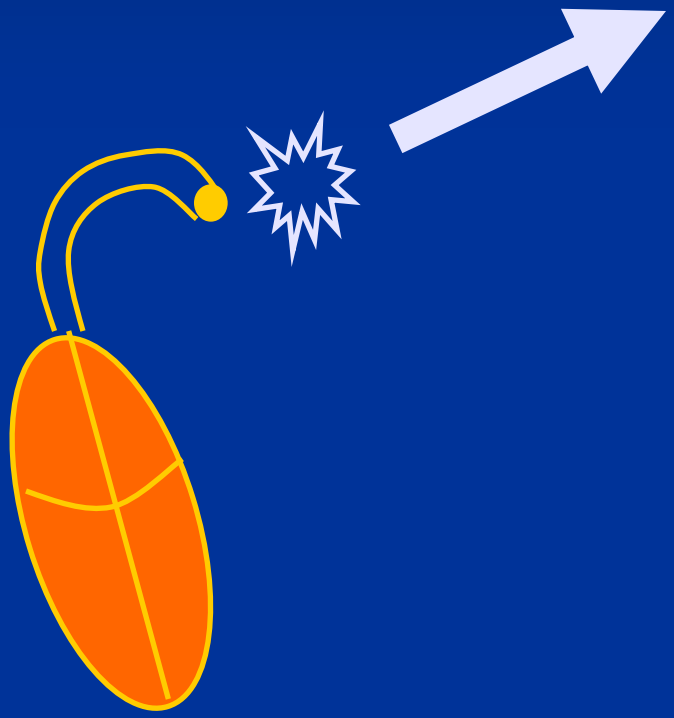
Shock pathofysiology

- cellular hypoxemia – energy deficit, > aerobic metabolism switch to anaerobic, > lactic acid accumulation – metabolic acidosis > cellular function failure, Na/K pump impaired > mitochondria damage, cell death


Lacerations:
Brain
Brainstem
Aorta
Cord
Heart
Epidural
Subdural
Hemopneumothorax
Pelvic fractures
Long bone fractures
Abdominal injuries
Sepsis
MOF
Deaths
0
1 hour
3 hours
2 weeks
4 weeks
Time

Compensatopry mechanisms

- Baroreceptors , volumoreceptors i aorta – afferental signaling to CNS – sympathetic stimulation, Adrenaline – vasoconstriction / defensive responce/

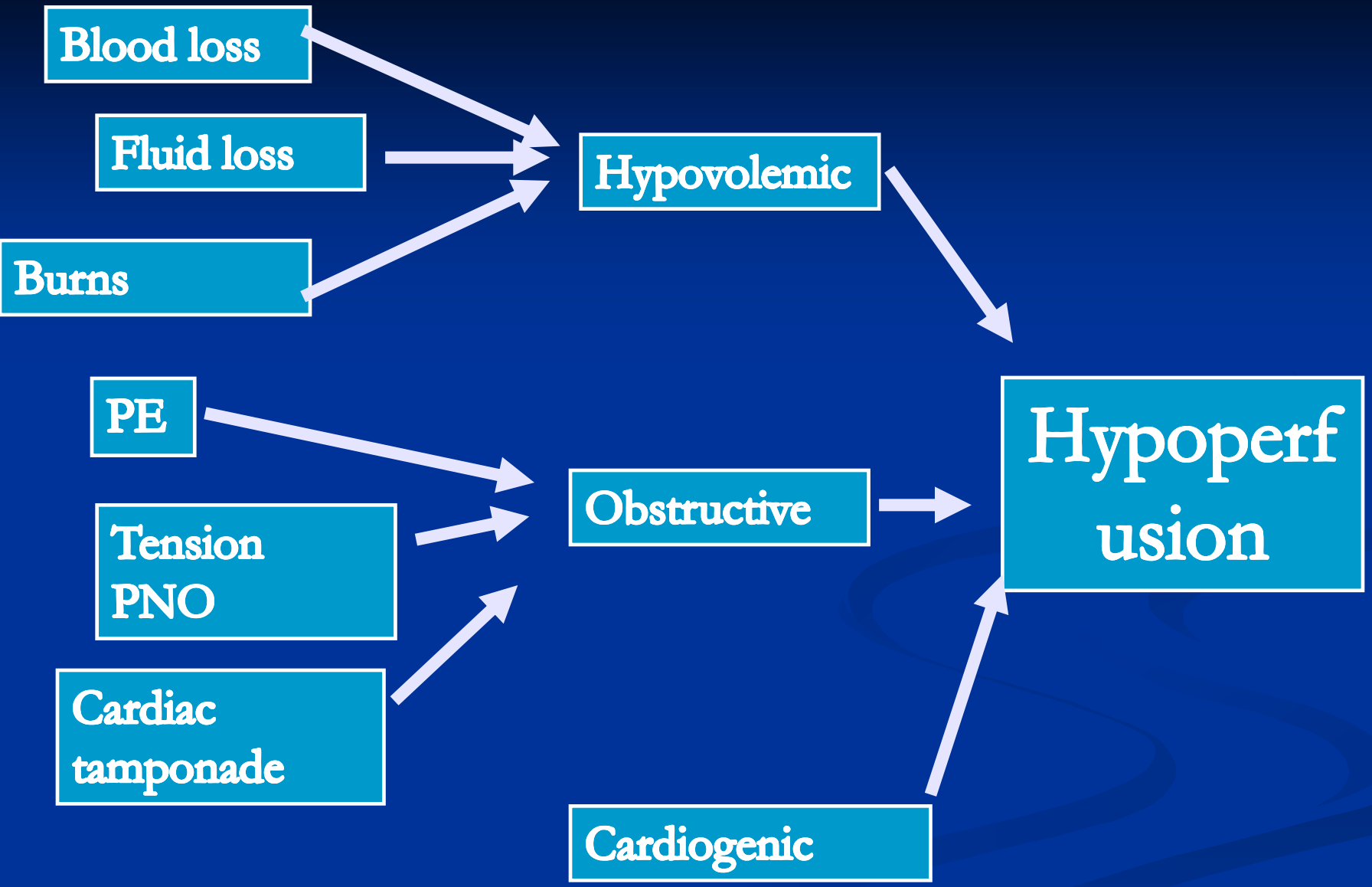


ympathetic stimulation

Classification of shock

Based on initiating mechanism

- Hypovolemic
- Cardiogenic
- Obstrctive
- Distributive
- Septic
- Anaphylaxis
- Neurogenic


Blood loss
Fluid loss
Burns
Hypovolemic
PE
Tension PNO
Cardiac tamponade
Obstructive
Cardiogenic
Hypoperf usion

Hypovolemic shock

- reduced circulating volume (Empty tank) due to haemorrhagic/ non haemorrhagic causes (dehydration)
- -1. blood loss – trauma
- - 2. fluid loss – diarrhea, nausea and vomiting

Severity estimation

- Allgower shock index (PR/SBP)

60/120 – 0,5 normal condition

100/100 – 1 – impending – loss of 30% of volime

120/80 – 1,5 – moderate/severe shock – life threatening

- Estimation of blood loss

humerus 200-1000 ml forearm 400 ml shin 500-1000 ml

pelvis 2000-3000 ml / more femur 1500-2000 ml

	I	II	III	IV
Blood loss ml	<750	750-1000	1500-2000	>2000
Blood loss %	<15	15-30	30-40	>40
HR	<100	>100	>120	>140
BP	normal	orthostatic	hypotension	Severe hypotension
Mental status	normal	anxious	confused	coma
Urine output	normal	normal	reduced	anuric
Lactic acidosis	+	++	++	+++

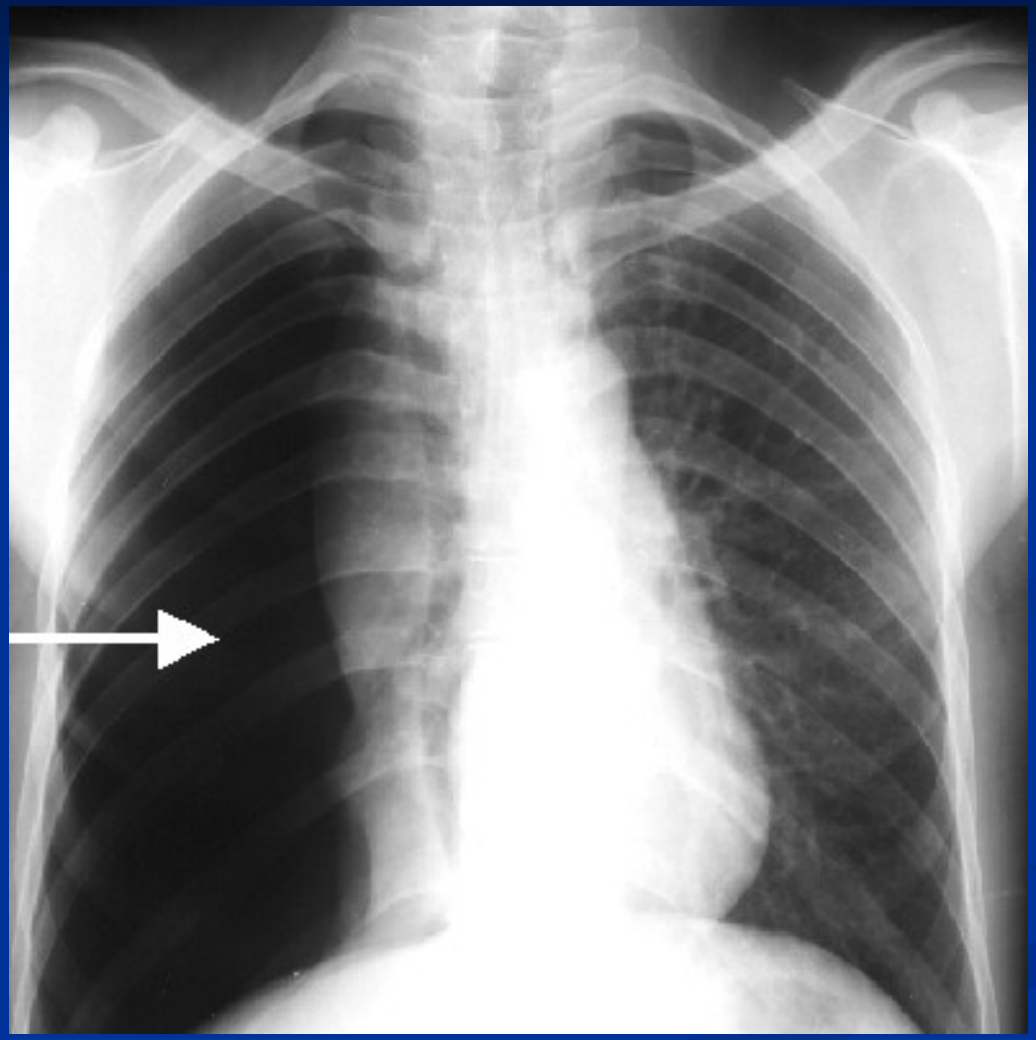
- **Obstructive shock** - mechanical obstruction
- PE
- Tension PNO
- Cardiac tamponade

Tension pneumothorax

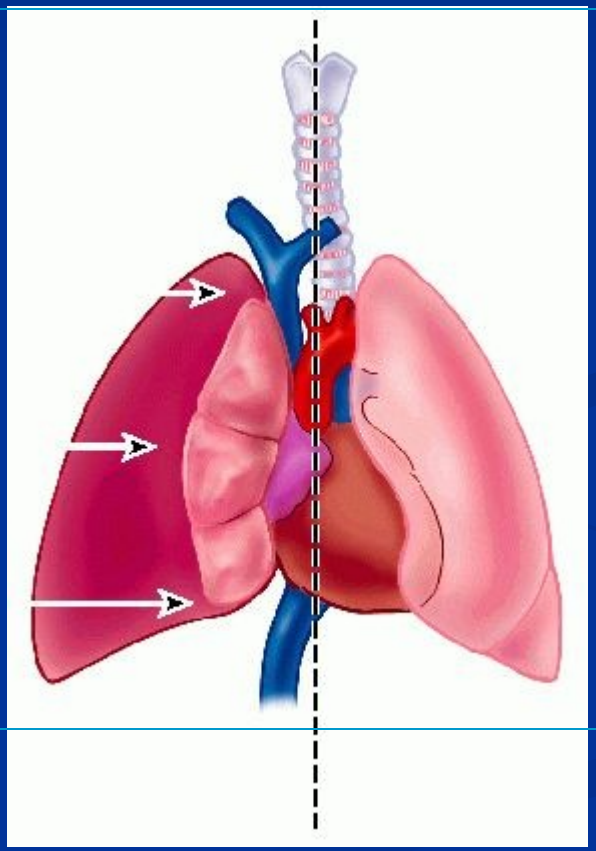
- occurs when air enters the pleural space from a lung injury or through the chest wall without a means of exit
- results in death if it is not immediately recognized and treated
- when air is allowed to leak into the pleural space during inspiration and becomes trapped during exhalation, an increase in the pleural pressure results

Tension pneumothorax

- increased pleural pressure produces mediastinal shift
- mediastinal shift results in:
  - compression of the uninjured lung
  - kinking of the superior and inferior vena cava, decreasing venous return to the heart
- progression of simple or open pneumothorax



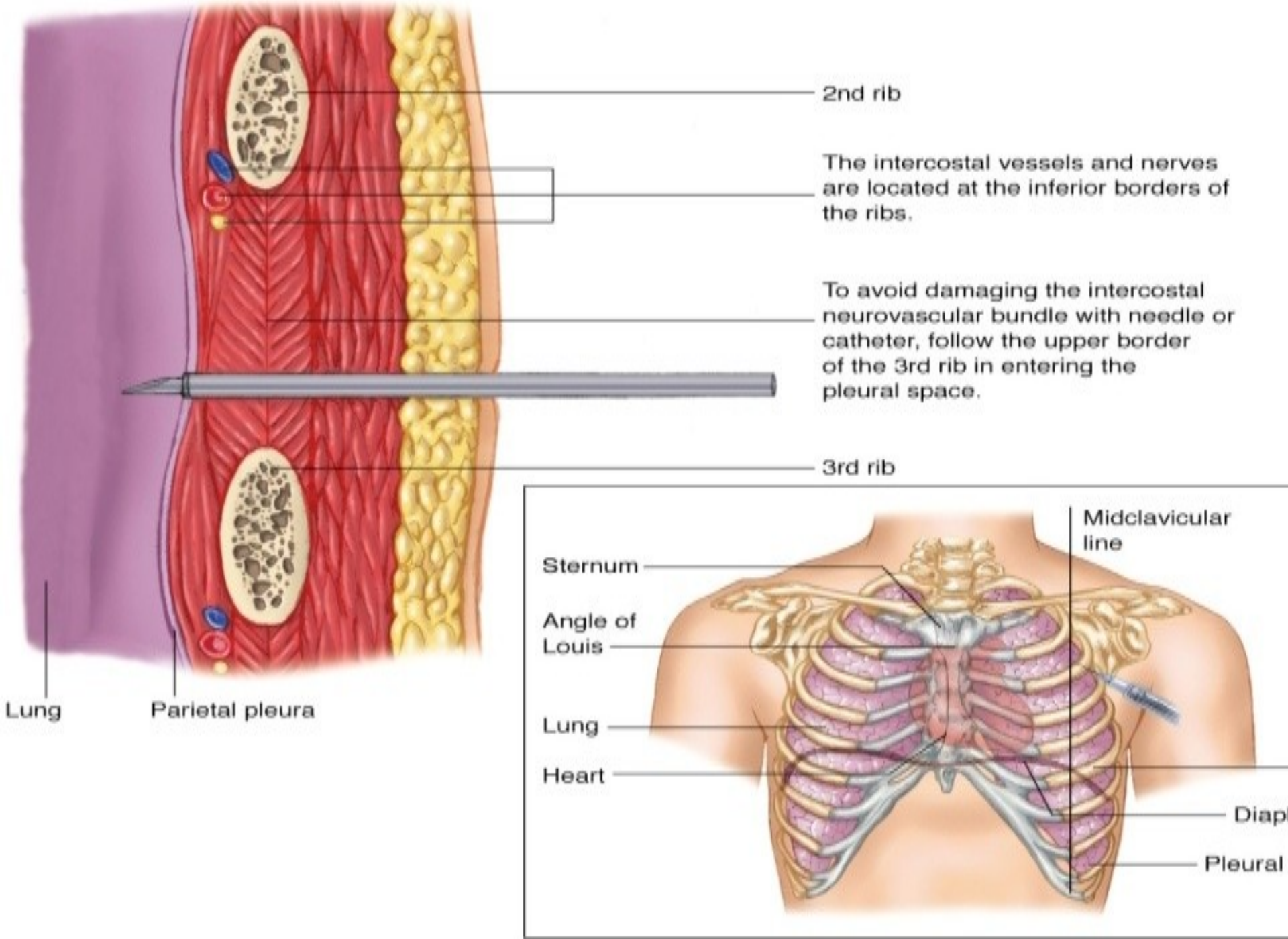
Tension pneumothorax



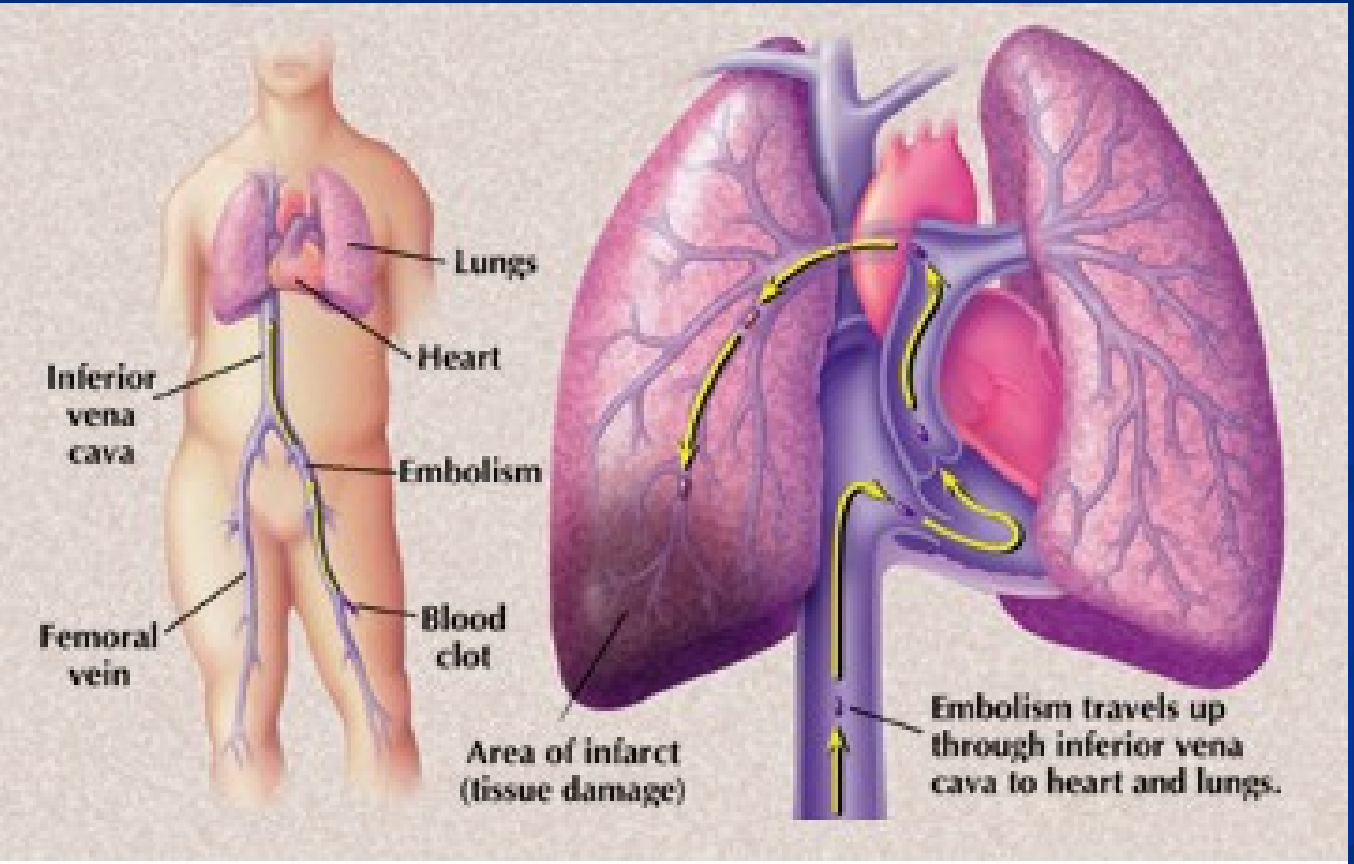
Tension pneumothorax

- management
- emergency care is directed at reducing the pressure in the pleural space

- occlude open wound
- needle thoracostomy
- tube thoracostomy – in-hospital management
  - 4nd intercostal space in mid-axillary line
    - TOP OF RIB


2nd rib
The intercostal vessels and nerves are located at the inferior borders of the ribs.
To avoid damaging the intercostal neurovascular bundle with needle or catheter, follow the upper border of the 3rd rib in entering the pleural space.
3rd rib
Lung
Parietal pleura
Midclavicular line
Sternum
Angle of Louis
Lung
Heart
Diap
Pleural

Pulmonary embolism



Pulmonary embolism

- ECG
- X-ray
- D-Dimery
- **CT pulmonary angiogram (CTPA)**
- ECHO

Pericardial tamponade

- restriction to cardiac filling caused by blood or other fluid within the pericardium
- occurs in <2% of all serious chest trauma
  - however, very high mortality
- results from tear in the coronary artery or penetration of myocardium
  - blood seeps into pericardium and is unable to escape
  - 200-300 ml of blood can restrict effectiveness of cardiac contractions

Pericardial tamponade

- signs and symptoms
  - tachycardia
  - respiratory distress
  - Becks triad - narrowing pulse pressure
    - neck vein distention
    - muffled heart sounds
  - ECG changes

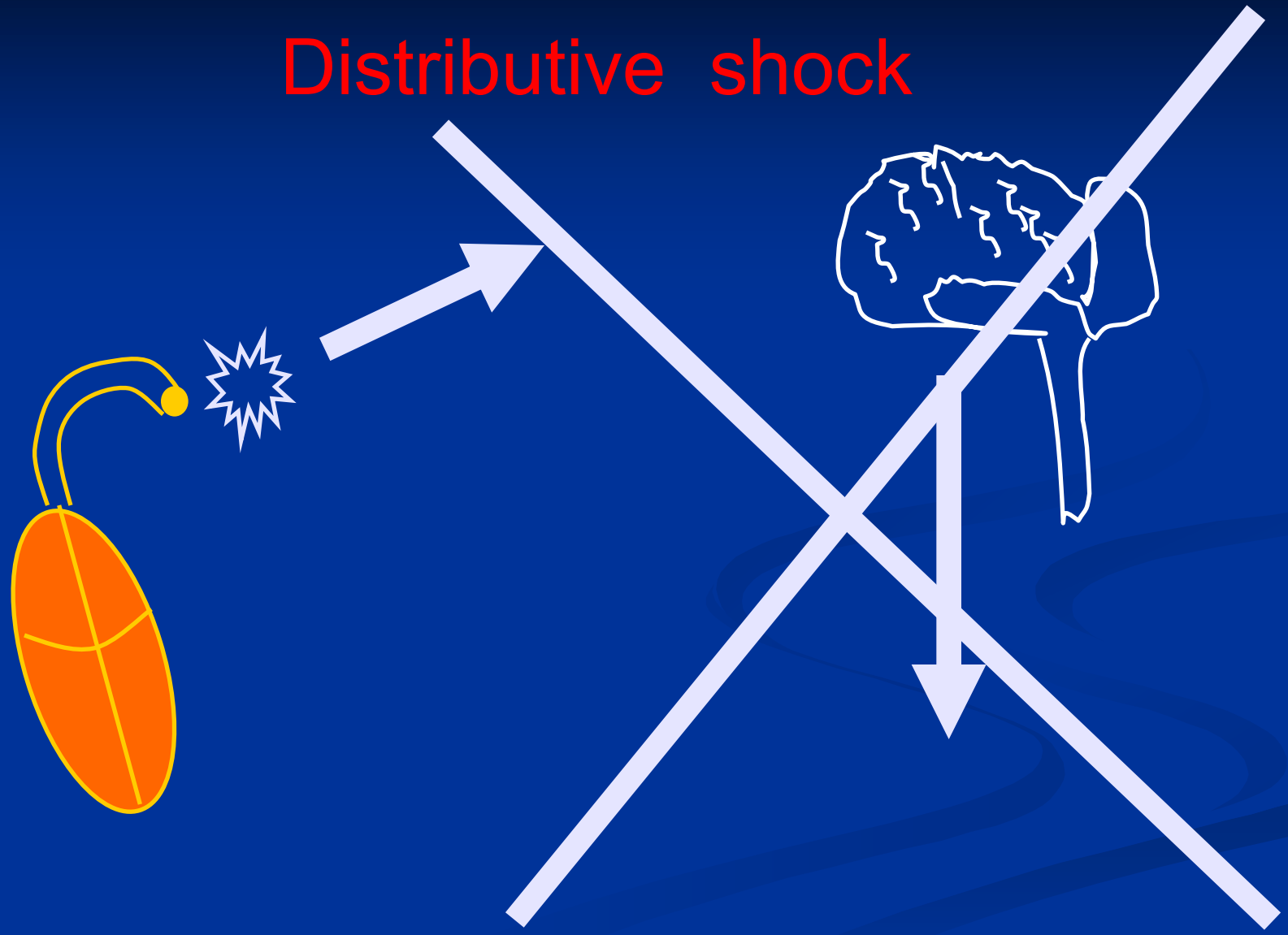
Pericardial tamponade

- management
  - high flow O2
  - IV therapy
  - pericardiocentesis – needle insertion through the skin incision directed toward the left shoulder at a 45 degree angle to the abdominal wall.

Cardiogenic shock

- primary failure of heart pump ,(IM, valvular disease, malignant arrhytmia)

Distributive shock



Distributive shock

- Blood vessel problem - hypoperfusion due to periferal vasodilation with hypotension
- Cardiac pump and blood volume are normal but blood is not reaching the tissues

Distributive shock

- **Septic shock** – infectious agent (endotoxines, SIRS) provoked periferal vasodilation, sepsis + acute circulatory failure with hypotension
- **Anaphylactic shock** – a type of shock that results from widespread systemic allergic reaction to an antigen
- **Neurogenic shock** - a type of shock that results from the loss or suppression of sympathetic tone

Septic shock – risk factors

- Age
- Malnutrition
- Use of invasive catheters
- Traumatic wounds
- Drug therapy

Septic shock – risk factors

- Initiated by G neg. or G posit. Bacteria, fungi or viruses
- Cell walls of organism contain endotoxins – endotoxins release inflammatory mediators – vasodilatation and increase capillary permeability leads to shoc due to alteration in peripherial circulation and massive dilation

- **Inflammation** – complex defensive reactio of vascularized tissue to harm
- **Infection** – inflammation with identified microorganism in host tissue
- **Bacteremia** – presence of bacterias in bloodtream
- **SIRS** – systematic inflammatory responce syndrom
- **Sepsis**

Inflammation

- Rapid, complex biological response of body tissue to harmful stimulus

- elimination of harm causes, clear out damaged tissues and cells, prepare site for reparation of tissue defects,

- innate defensive responce and reparative process, complex of immune cells and blood vessels and molecular mediators

Inflammatory reaction

- Injured tissue cells release chemokines, histamin, etc.,

- **Vascular phase** – activation of endothelial cells – adjacent capillaries dilate, increase blood flow, endothelial cells contract, increase intercellular gaps, increase permeability, exudation of fluids, solubile proteins to soft tidssue

- activation of endothelial surface

SIRS
(Systemic Inflammatory Response Syndrome)

Massive tissue inflammation, long lasting – overproduction of proinflammatory cytokines – all tissues affected.

SIRS

- SIRS to infection manifested by two or more of following -
- BT > 38 / < 36 dg.
- HR > 90/min
- Tachypnea > 20/min
- Leukocytosis > 12 0000 or leukopenia < 4 000

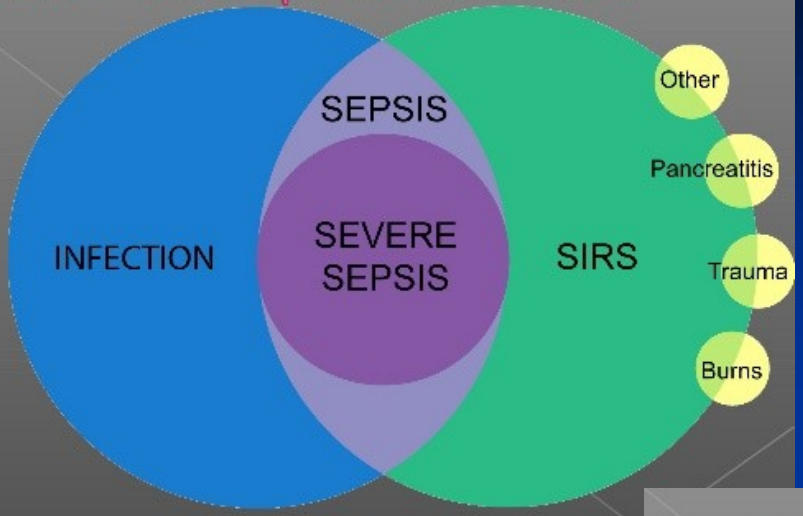
Sepsis

- Life threatening clinical syndrome
- Specific type of SIRS condition + confirmed infection

- Clinical signs
- Lab / CRP, Procalcitonin /
  Imaginig / X-rax, CT scan /
- prove of infection

- Sepsis
- Severe sepsis – sepsis + new-onset organ dysfunction
- Septic shock – severe sepsis + acute circulatory failure with hypotension
- MODS

Sepsis: A Complex Disease



Adapted from: Bone RC et al. *Chest* 1992;101:1644-55.
Opal SM et al. *Crit Care Med.* 2000;28:S81-2.

Sepsis: Defining a Disease Continuum

Infection/ Trauma → *SIRS* → *Sepsis* → **Severe Sepsis**

A clinical response arising from a nonspecific insult, including ≥2 of the following:

- *Temperature ≥38°C or ≤36°C*
- *HR ≥90 beats/min*
- *Respirations ≥20/min*
- *WBC count ≥12,000/mm³ or ≤4,000/mm³ or >10% immature neutrophils*

SIRS with a presumed or confirmed infectious process

SIRS = systemic inflammatory response syndrome.

Bone et al. *Chest* 1992;101:1644.

Typical Features of Shock States

	Heart rate	Blood pressure	CVP	CO	SVR	Lactate
Hypovolaemic shock	↑	↓	↓	↓	↑	↑
Cardiogenic shock	↑	↓	↑	↓	↑	↑
Distributive shock	↑	↓	↓	↑	↓	↑
Obstructive shock	↑	↓	↑	↓	↑	↑

Table 1 The relationship between the four types of shock and associated clinical measurements

Clinical signs

- Tachycardia – BP >100/min
- Hypotension – SBP < 90 torr
- Altered mental status
- Pallor, cold sweat, capillary refill
- Tachypnea, hyperventilation > 20/min

- Oliguria(<500ml/d or 30ml/h)
- Anuria (<100ml/d or 0,5ml/h/kg)
- Decrease CVP <2cm H2O, /5-7/
- Lactate > 2mmol/l hypoxia, > 4 mmol/l

- Monitoring

- ECG,
- cont. saturation,
- hourly urine output

- Invasive monitoring

- CVP
- invasive arterial BP
- Cardiac output
- Lactate, Base deficit,

therapy

- Signs of shock - begin therapeutic management + complete dif. dg. of shock cause

- Fluid resuscitation - refill volume
- Dynamic fluid responce – 500ml /20min, maesure response
- Adequate oxygenation
- Surgical management
- Pharmacological therapy – inadequate effect of volume refill

- vascular resistance – Noradrenaline / Norepinephrine sepsis first choise
- Inotropic suppurt – low CO - Dobutamine

Septic shock management

- Signs of shock
- vital signs + lactate level
- Microbiological exam samples
- Empiric ATB
- Fluid resuscitation 20ml/kg
- CVP 8-10

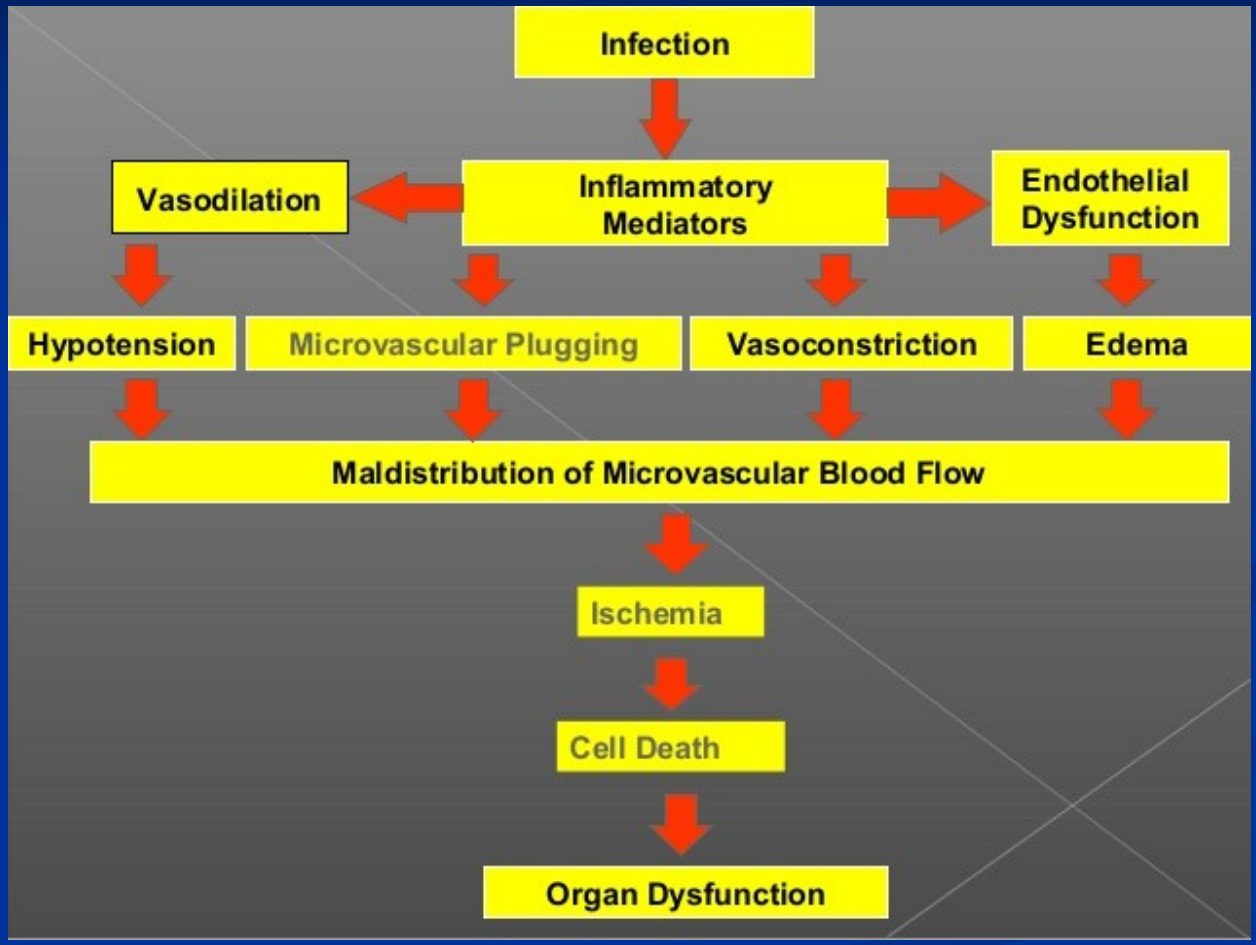
MODS
(Multipl Organ Dysfunction Syndrome)/ MOF (multiple organ failure)

- most severe condition , high mortality rate > 60%
- 2 or more organ systems dysfunction in acutely ill patients such that homeostasis cannot be maintained without intervention
- Complication of prolonged shock

MODS
(Multipl Organ Dysfunction Syndrome)

- Primary MODS – direct insult/ injury to organ causing failure of function / aspiration…/

- Secondary MODS – complication, distant organ affected due to profound SIRS/sepsis


Infection
Vasodilation
Inflammatory Mediators
Endothelial Dysfunction
Hypotension
Microvascular Plugging
Vasoconstriction
Edema
Maldistribution of Microvascular Blood Flow
Ischemia
Cell Death
Organ Dysfunction

Organ dysfunction

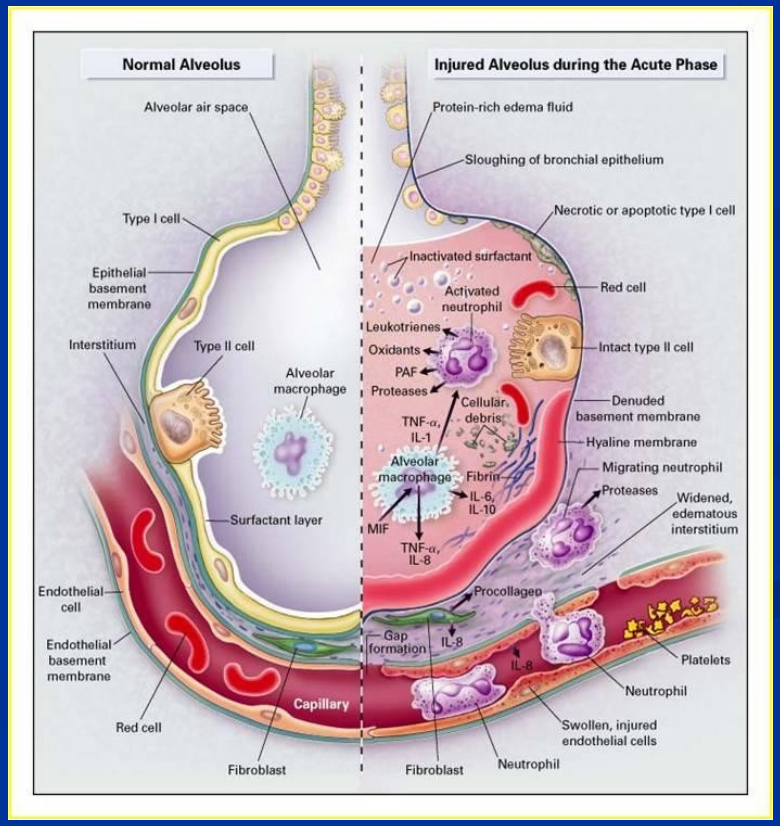
- Lungs – ARDS
- Kidneys – AKI – acute tubular necrosis
- CvS – shock
- CNS – metabolic encephalopathy
- Coagulation – DIC
- Endocrine – adrenal insufficiency
- Liver - cholestasis
- Skeletal muscle – rhabdomyolysis
- GIT – gastroparesis, ileus, ulcer

ARDS/ ALI
(Adult Respiratory Distress Syndrom/ Acute lung injury)

- Chest trauma, aspiration, sepsis
- Pulmonary endothelial cell reactio to inflammatory mediators, hypoxia, acidosis
- Most common, lung first filter

- Dynamic acute formation in 12-48 h.
- X-ray/ CT – bilateral flaky/patchy infiltrates

- Create higher permeability of pulmonary vessels – exudate, progresion of gas exchange imparement, interstitial pulmonary oedema, loss of pneumocytes > lack of suffactant > alveolar pulmonary oedema, or athelectasis



- Stage I - latent – dyspnoe, mild hypoxemia, hyperventilation, X-ray – flaky opacities aroun hilus
- Stage II – ortopnoe, moderate hypoxemia, cyanosys, tachacardia, tachypnoe, X-ray – pulmonary oedema
- Stage III – terminal – sebere hypoxia, shock, AV , X-ray diffuse infiltrates

End-stage – global respiratory insufficiency – reductio of alveolar surface, fibrosis

- Th
- Oxygen support
- artefitial lung ventilation+ PEEP
- Glucocorticoids
- prone position

DIC
(Disseminated Intravascular Coagulation)

- Acquired coagulopathy – multiple systemic formation of blood clots – trombus- in microvascular circulation – consumption of coagulation factors – diffuse tissue/parenchamal bleeding

  periferal tissue ischemia
- Hematologic examination (fibrinogen, DD, FDP, aPTT, INR, AT) InR >1,5, aPTT > 60sec, PLT < 80 000
- LMWH
- Supplementation of AT III, FFP, EBR, fibrinogen, platelets
- Therapy of primar cause

DIC

Source: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ: *Fitzpatrick's Dermatology in General Medicine*, 7th Edition: http://www.accessmed

AKI / Acute kodnes injury

- Kidney vessels – autoregulation - perfusion maintain in BP 50-180mmHg

- Prerenal failure - low volume
- Intrincis cause – acute tubular necrosis – ischemic/ toxic – muddy brown cast during urinanalysis

- Oligoanuria, /<500/50ml/, fluid balance, uremia, creatinin > 177

- RRT - iRRT/ CVVH

Liver failure

- Due to hepatocyte necrosis – impairement of excretion function – icterus due to hyperbilirubinemia, bilirubin > 35mmol/l

- Imparement of synthetic and metabolic function

- hypoceagulation, haemorrhage, hypoglycemia

Liver ecephalopaty - due to hyperamonemia

Symptomatic therapy, supplemtally therapy

Metabolic complications

- Hyperglycemia
- Insulinoresistence
- Catabolic reactions - rhabdomyolysis

GiT

- Stress gastric ulcer – upper GiT haemorrhage

- Gut ischemis - loss of barrier finction against intestinal bacterias and toxins > translocation, inflmmatory response, souce of bacteremia and sepsis of critically ill patients

- PPI
- early enteral nutrition / glutamin/

Local/ infectious complication

- I.v. ATB therapy , high dose, sensitivity
- Surgical debridement of necrotic tissue/ drainage of abscess
- Surgical management of initial harmful insult / surgical bleeding, PNO, pleural effusion /

Compartment syndrome

- A condition in which increased compartment pressure within a confined space, compromises the circulation and viability of the tissues within that space

Compartment syndrome

- Increase in volume of contents

  and/or

- Reduction in size of compartment

  ↓

- increased pressure within the compartment

  ↓

- compression of muscles, nerves & vessels

  ↓

- impaired blood flow

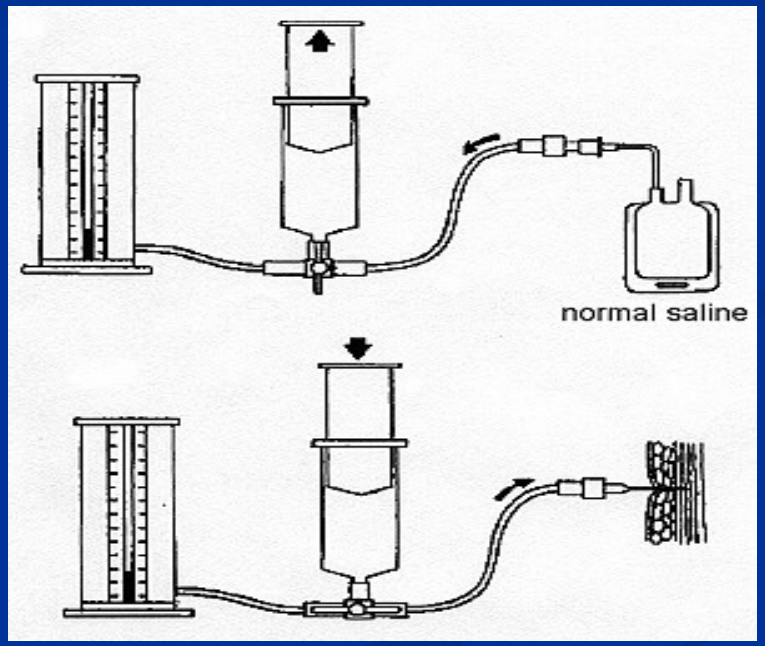
  ↓

- ischemia & necrosis

Compartment syndrome Symptoms

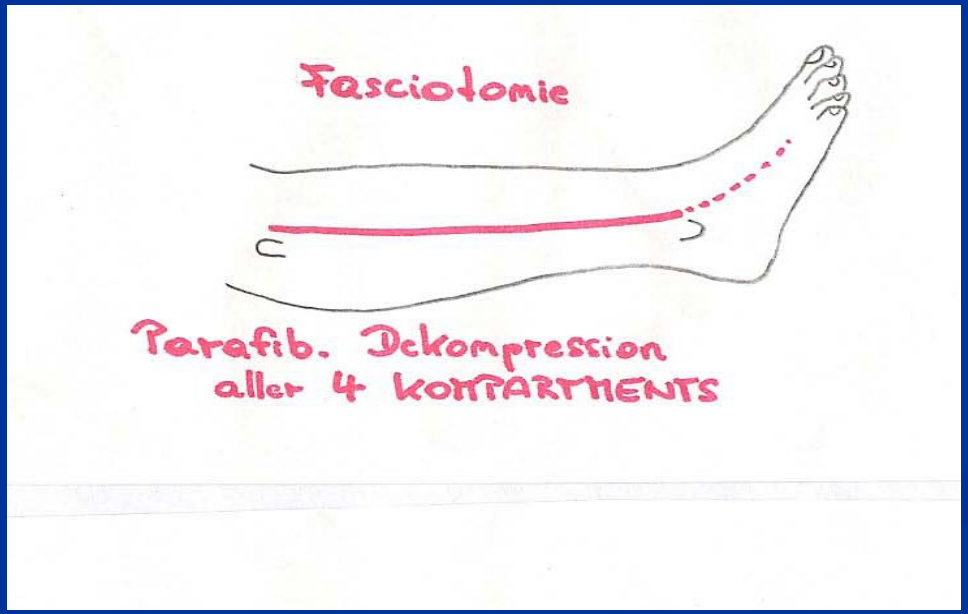
- Pain
- Paraesthesia
- Paresis
- Pulses present
- Functional failure

Compartment syndrome-pressure monitoring

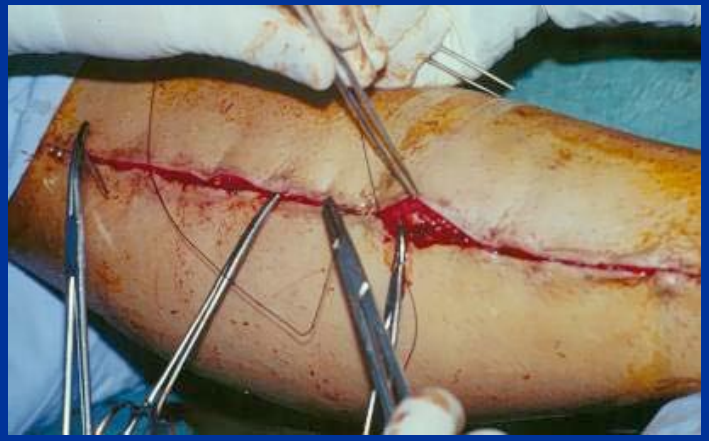


Compartment syndrome- Therapy

- Surgical decompression with a **fasciotomy** is the **definitive treatment**



Compartment syndrome- Therapy



Compartment syndrome-abdominal