RESUSCITATION, TRAUMA, AND CRISIS MANAGEMENT

PHYSIOLOGY

Shock

Describe the systemic inflammatory response and its physiological effects

SIRS = systemic response characterised by >2 of the following:
- HR >90
- RR>20
- Temp <36 or >38
- WCC <4x10⁹/L or >12x10⁹/L

Sepsis = SIRS 2o confirmed or suspected infection
Severe sepsis = involves organ dusfunction or hypoperfusion
Septic shock = sepsis with circulatory failure (SBP<90mmHg)

Define shock

Shock = clinical condition of inadequate tissue perfusion relative to demand → insufficient metabolic substated (1o O2) provided to sustain cellular homeostasis
Classification of shock
- Hypovolaemic: 2o depletion of intravascular vol
  - Excessive losses e.g. blood loss, pre-op dehydration, recent haemodialysis, GI losses
  - Inadequate intake/ replacement
  - Expansion of vascular tree g during re-warming
  - Fluid shifts into 3rd space compartments
- Obstructive: 2o impairment of blood flow to or from the heart
  - Tension pneumothorax
  - Embolism: thrombus, gas, fat, air, amniotic fluid
  - Abdominal compartment syndrome → IVC compression
  - Pericardial tamponade
  - Valvular pathology e.g. AS, MR
  - Aortic dissection
- Cardiogenic: 2o impaired pump function → ↓CO state
  - Primary contractility failure: MI, LVF, CMP
  - ↑myocardial work: pain, fever
  - myocardial depression: anaesthetic agents, acodisos, BB
  - dysrhythmias
  - cardiac contusion
- distributive: 2o loss of SVR
  - inflammatory/ distributive: sepsis, anaphylaxis
  - loss of SY tone: high spinal, spinal cord injury
  - adrenal insufficiency: addisonian crisis

Integrate knowledge of factors determining cardiac output to classify causes of shock

- Shock = mismatch of supply and demand
- MAP = CO x TPR
- CO = SV x HR where SV is dependent on preload, afterload (or wall tension), and contractility
- Therefore MAP = SV x HR x TPR
- Causes can be classified according to these determinants of CO
  - SV
    - ↓Preload
      - Hypovolaemia: haemorrhage, addisonian crisis, dehydration
      - Distributive: sepsis, anaphylaxis → ↓SVR
    - ↑Afterload
      - Critical aortic stenosis
      - PE: ↑RV afterload
    - ↓Contractility
      - Cardiogenic: 2o myocardial sischaemia, myocarditis
      - Cardiac depression from acidosis
  - HR: Cardiogenic 2o dysrhythmia e.g. profound brady/ tachyarrhythmia
  - TPR: Distributive

Describe the physiological consequences of shock

- conversion to anaerobic metabolism → lactic acidosis
- anaerobic metabolism
  - inefficient process that results in formation of large amounts of lactic acid
  - glucose + 2NAD+ + 2ATP → 2pyruvate + 2NADH + 4ATP
  - pyruvate + NADH → lactate + NAD+
  - lactate can be used as energy substrate by heart or reconverted to glucose via Cori cycle in liver
- hypoperfusion measns that lactate may not be cleared efficiently by liver
- ↑lactic acid → ↑H ions → ↓pH → metabolic acidosis
- compensated by several mechanisms
  - chemical buffers
  - resp compensation: hours to days
  - renal correction: days to weeks: HCO3 reabsorption; H secretion; formation of new HCO3; buffering to PO4
  - if compensation inadequate → intracellular acidosis + acidaemia may impair enzyme processes inc essential metabolic processes e.g. glycolysis which can lead to cell damage/ death

Describe oxygen delivery and outline the use of indicators of tissue oxygenation (base deficit, lactate, mixed venous oxygen saturation) in resuscitation

O2 carriage

O2 is carried either bound to Hb or dissolved in solution

- Normal arterial blood has a PO2 of 100mmHg and is 97.5% saturated
- Venous blood has a PO2 of 40mmHg and is 75% saturated

O2 content

- Vol of O2 carried in each 100ml blood
- **CaO2 = (Hb x sats x 1.34) + (PaO2 x 0.003)**
  - 1.34 = Hufners constant at 37degrees
  - 0.003 = ml of O2 dissolved per 100ml plasma per mmHg
  - O2 content per 100mL arterial blood = 20ml; venous blood = 15ml

O2 delivery (DO2)

- Amount of O2 delivered to the peripheral tissue
- O2 delivery = CaO2 x CO

O2 uptake (VO2)

- VO2 = DO2 – O2 return

Extraction ratio

- Extraction ratio = VO2:DO2
- Usually 25%

Base deficit

- Arterial base deficit = amount of additional base that must be added to ↑IL of whole blood to predicted pH based PaCO2
- Calculated vale based on PaCO2, pH, HCO3
- ↑ in base deficit are classified as mild (2-5mmol/L), moderate (6-14mmol/L) or severe (>15mmol/L) and are used as surrogate marker of metabolic acidosis
- often used as simple indicator of O2 delivery
- shown to predict hospital mortality, length of stay, severity of sepsis and organ failure
- drawbacks:
  - requires arterial puncture or IAL
  - less accurate in acute EtOH intoxication, renal dysfunction, large vol crystalloid resus and hypoalbuminaemia
  - weak or poor correlation with serum lactate

Lactate

- Lactic acidosis = serum lactate >5mmol/L + arterial pH <7.35
- Bi product of anaerobic metabolism (thus suggestive of tissue hypoxia)
- ↑in all forms of shock + degree of ↑ corresponds to severity of shock
- often used to guide effectiveness of therapies/ interventions and as a predictor of outcomes
- ½ life 20mins
- drawbacks
  - ↑levels may not always be 2o anaerobic metabolism
  - ↑may be 2o ↓hepatic clearance, trauma, profound dehydration

Mixed venous O2 saturation

- via aspiration of blood from distal port of unwedged PA catheter → ensures complete admixture of blood from SVC + IVC + coronary sinus
- At a PvO2 of 26 mmHg → average intracellular PO2 ↓from 11 to 0.8 mmHg
  - SvO2 = 0.5 → theoretical critical PvO2 of 26 mmHg; lactic acidosis appearing between 0.3 and 0.5.
  - SvO2 > 0.8 → high-flow states e.g. sepsis, hyperthyroidism, severe liver disease.
- Drawbacks:
  - Requires PA catheter
  - SvO2 as a therapeutic target (> 0.7) failed to improve survival in a multicentre trial
  - SvO2 is insensitive to cytopathic hypoxia and tissue shunting

Outline how the signs of shock can be altered by age

- Lack of physiological reserve in extremes of age – much less able to compensate and deteriorate more rapidly than adults
- Neonates:
  - CNS signs are less obvious
  - Look for lethargy, not crying or weak cry
  - Not feeding, dec no wet nappies, skin turgidity
  - MAP is more HR dependent and less contractility-dependent
- Adults:
  - Compensation can be so effective that underlying shock state may only manifest as tachycardia and more fully revealed by a postural BP drop (greater than 20mmHg)
  - Are able to tolerate shock for longer but when compensation is overwhelmed, they may suddenly deteriorate
- Elderly:
  - Early signs - confusion, ↓ urine output, cool peripheries are often overlooked as being 'normal' in elderly people
  - Incapable of mounting a tachycardic response due to medications (beta-blockers) or underlying cardiac disease

Other questions covered elsewhere in syllabus

Describe the physiological basis of anaphylactic and anaphylactoid reactions

See immunology

Describe blood groups and the physiological basis of transfusion reactions

See haematology section

Outline the changes that occur in stored blood

See haematology section

Describe physiological consequences of massive transfusion

See haematology section

Outline the factors determining intracranial pressure and discuss its regulation

See Neurophysiology section

Describe the cerebral circulation, the regulation of cerebral blood flow and factors leading to the loss of autoregulation

See Neurophysiology section

Discuss cerebral perfusion pressure

See Neurophysiology section

Describe the blood supply to the spinal cord and the regulation of spinal cord blood flow

See Neurophysiology section

Discuss spinal cord perfusion pressure

See Neurophysiology section

PHARMACOLOGY

With reference to the management of shock, describe the pharmacology of vasopressors and inotropes, including: adrenaline, noradrenaline, phenylephrine, metaraminol, dopamine, dobutamine, phosphodiesterase inhibitors, vasopressin

See CVS/ autonomic pharmacology

With reference to cardiopulmonary resuscitation, describe the pharmacology of adrenaline, vasopressin, amiodarone and lignocaine

See CVS/ autonomic pharmacology

Outline the pharmacological managementof ventricular fibrillation in an adult

General;

- VF = life threatening arrhythmia
- Rapid, irregular ventricular activation

Aim = cease irregular rhythm + maintain perfusion

Electrical defibrillation

- Only effective treatment
- Biphasic 200K; monophasic 360J

Drug therapy

- Adrenaline: 1st line drug
  - 1mg repeated every 3 mins
  - MOA: α/ β agonist → 1o action in arrest; α1 GPCR → ↑phospholipase C → ↑DAG, IP3, Ca2+ → ↑SVR 2o vasoconstriction, ↑CBF/ ↑corBF
- Antiarrhythmics: 2nd line drug
  - Amiodarone
    - 300mg
    - MOA: partrial antagonist α/ β receptors: ↑cardiac AP 2o ↑K channel opening + class 1 properties → ↓Na+ opening + class 4 properties → ↓opening Ca2+ channels (↓plateau)
    - Adverse effects: AV node block → complete hear tblock; if hypokalaemia → ↑risk arrhythmias
  - Lignocaine
    - Class Ib antiarrhythmic
    - 1.5mg/kg
    - MOA: block fast Na channels → ↓rate of depolarisation, ↓peak + membrane stabiliser
    - less effective than amiodarone
  - NaHCO3
    - Reverse acidosis + correct ↓K+

With reference to the treatment of malignant hyperthermia, describe the pharmacology of dantrolene

For pharmacology of dantrolene – see General Anaesthesia section

Malignant hyperthermia

- Pharmacogenetic disease of skeletal muscle
  - Induced by exposure to : volatile agents; depolarising muscle relaxants i.e. sux
  - Inherited autosomal dominant condition
  - Caused by loss of normal Ca homeostasis within excitation-contraction coupling process on exposure to trigger
- Most likely site:
  - Junction between T tubules
  - Voltage sensor of dihydropyridine receptor (DHPR) and ryanodine receptor (RYR) → efflux Ca2+ channel in sarcoplasmic reticulum
- Epidemiology
  - Rare; 1:10 000
  - Mortality ↓due to awareness + dantrolene
  - Young adults; males > females
  - Previous uneventful anaesthetic does not prevent occurrence
- Clinical features
  - Varied presentation: florid + life threatening vs. insidious; acutely vs. 2-3 d postop with massive myoglobinuria + rhabdo
  - ↑metabolism: tachy/ arrhythmia; ↑CO2 production; metabolic acidosis; fever (late) with ↑2°/hour; DIC
  - muscle signs: masseter muscle spasm post sux
  - generalised rigidity
  - ↑K
  - ↑CK
  - myoglobinuria → renal failure
- DDx: rebreathing, sepsis, awareness, neuroleptic malignant syndrome, ectasy, thyroid storm
- Treatment
  - ABC
  - Stop volatiles
  - Hyperventilate: 100% O2
  - Declare problem to team and get help
  - Dantrolene 2-3mg/kg IV (20mg ampoules)
  - Stop surgery or use TIVA
  - ↓core temp: ice, cold fluid
- Peri MH treatment
  - Clotting screen + CK
  - Urine samples
  - Monitor renal function
  - Invasive monitoring
- Post episode care

- Refer to MH investigation unit for mm biopsy and testing
- Warn patient and family
- Offer screening

- Anaesthesia for known MH
  - TIVA with no sux
  - All Las are safe
  - Dantrolene should NOT be given prophylactically
  - Standard monitoring
  - Baseline temp 2hr preop + temp monitored for 4hrs post op
  - Use vapour free maching

ANATOMY

Anatomy relevant to vascular access in resuscitation: specifically for safe cannulation of antecubital, saphenous, jugular and subclavian veins and placement of intraosseous infusion devices

Antecubital, jugular, subclavian – *see General Anaesthesia: Vascular Access*

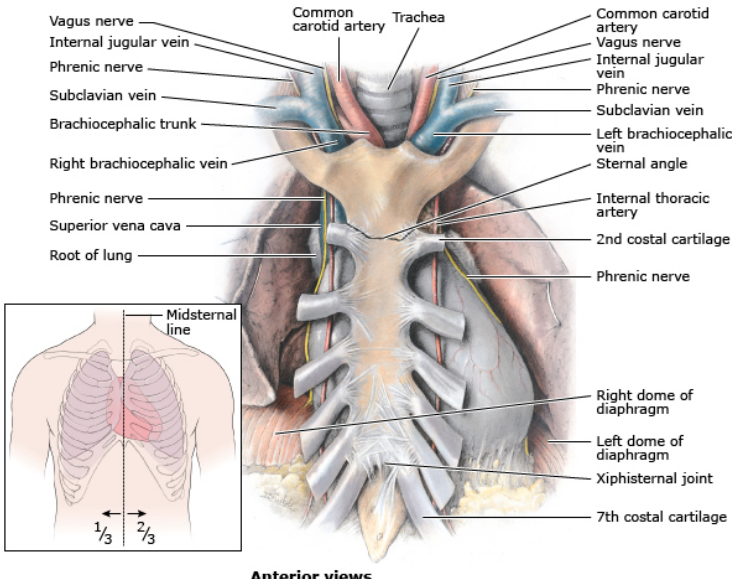
Intraosseous

Anatomy relevant to the drainage of pericardial fluid

Anatomy

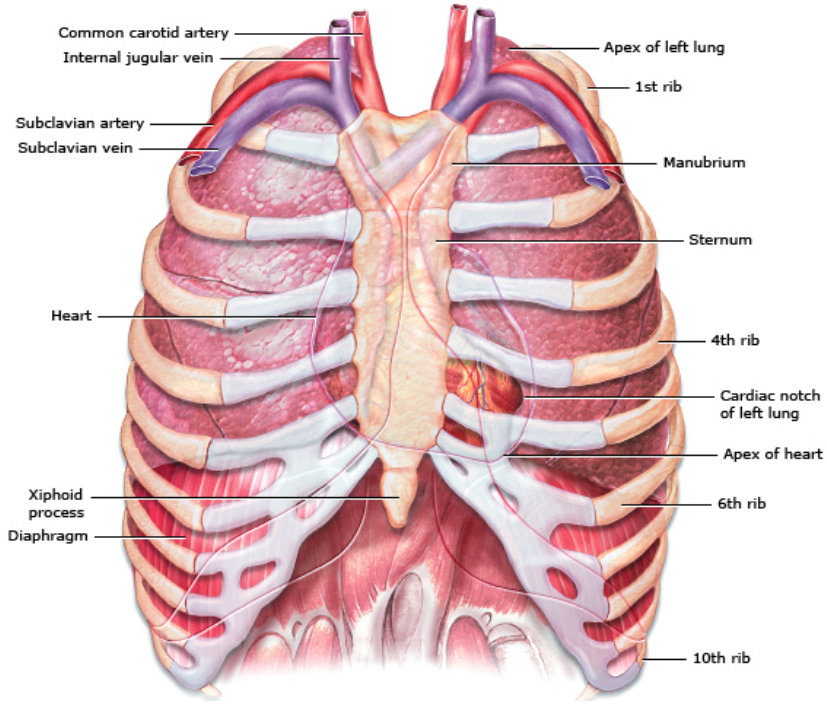
- pericardial sac:
  - 2 layers of tough, fibrous tissues surrounding heart
  - inner viscerla layer separated from parietal pericardium by lubricating pericardial fluid
  - pericardial fluid: ~15-50ml; produced by visceral mesothelium cells; drained by lymphatic system into mediastinum and R heart
  - pericardial viscera envelops heart + fuses at point where great vessels exit into mediastinum
  - visceral pericardium is continuous on inner side with epicardium
  - function: buffer heart from external impact, ↓reisstance during motion of heart; barrier against infection
-
- pericardial sac: lies ~1/3 to the right of the midsternal line + 2/3 to the L
-

Sternum, pericardium, and related anatomy



Anterior views

Rib and chest anatomy



Blind approaches
- subcostal (subxiphoid)
  - needle introduced substernally 1cm inferior to L xiphocostal anle → once beneath the cartilage, lower needle to 15-30o angle with abdo wall
  - aim needle toward L shoulder + advance slowly while aspirating. In most cases 7-9cm needle is adequate
- Parasternal
  - L sternal border = landmark
  - Insert needle perpendicular to skin over border of 5th/ 6th rib adjacent to sternal margin (cardiac notch of L lung exposes pericardium at this site)
  - Avoid puncturing more laterally to prevent injury to internal thoracic vessels
- apical
  - 5cm lateral to parasternal approach within 5-7 intercostal space
  - advance needle over top of rib and towards R shoulder

USS guided
- USS via subcostal, parasternal, apical
- select target flid lacer (distance from pericardium to epicardium) of at least 1cm to avoid cardiac puncture

NB loculation occurs in up to 1/3 of nontraumatic effusions

Other

Definitions
- cardiac tamponade = an accumulation of fluid in the pericardial sac → ↑pressure within pericardial space that impairs the ability of the heart to fill and pump
- ↓pump → ↓CO + ↓systemic perfusion → organ dysfnction
- NB slowly accumulating fluid allows physiological compensatory mechanisms to limit the ↑in pericardial pressure
- Tamponade vs. effusion:
  - Pericardial effusion = anatomical diagnosis: presence of abnormal fluid within the pericardial sac with no haemodynamic compromise
  - Cardiac tamponade = physiological diagnosis; pericardial effusion + evidence of obstructive shock

Indications
- symptomatic pericardial effusion (tamponade)

Complications
- Cardiac tamponade
- Arrhythmias
- Myocardial laceration
- Pneumothorax, pneumopericardium
- Liver laceration

Anatomy relevant to drainage of the pleural space

Needle thoracostomy (tension)
- 14G / 16G cannula
- midclavicular line, 2nd intercostal space
- always place ICC post

Pleurocentesis (pleural effusion)
- USS chest to confirm presence+ identify/mark site
-

Intercostal catheter
- placed in the safe triangle
  - anterior: pec major
  - post: Latissimus dorsi (NB too post will injure long thoracic nerve)
  - sup: base of axilla
  - inf: 5th intercostal space (NB too inf risks placement in liver / spleen)
- Site
  - mid-axillary line, 3-5th intercostal space
- Layers of dissection
  - Skin
  - Subcut tissue
  - External intercostal
  - Internal + innermost intercostal muscles: NB neurovascular bundle sits on inferior aspect of the ribs → therefore aim to place ICCon top of the bottom rib
  - Parietal pleura

Other:
- indications
  - pneumothorax
  - tension pneumo post needle thoracostomy
  - haemothorax
  - pleural effusion
- Procedure
  - USS site
  - LA; sterile technique
  - Arm above head
  - Blunt dissection: 2-3cm skin incision parallel to ribs; blung dissect; soft tube in
  - Seldinger
- Complications
  - Incorrect placement
  - Pulmonary laceration
  - Pneumothorax
  - Bleeding: incision site, intercostal vessels, lung, IMA
  - Infection
  - Mechanical: kinking, luminal obstruction

Anatomy of the cerebral and spinal cord circulation

Spinal cord

Arterial supply

- SC supplied by 3 arteries derived from post circulation of circle of Willis
- 1. One anterior spinal artery
  - Arises from branches of R + L vertebral artery
  - descends in anterior median sulcus
  - supplies anterior 2/3 of cord
- 2. Two posterior spinal arteries
  - Arise from posterior inferior cerebellar arteries
  - Descend along lateral aspect of spinal cord just medial to dorsal roots
  - Supply posterior 1/3 of cord
- 3. Radicular arteries
  - arise segmentally from local arteries and supply local ares of cord
  - Most important = artery of Adamkiewicz at T8 and L1 (from anterior spinal artery)

Venous drainage

- 3 anterior + 3 posterior spinal veins located in pia mater → anastomose to form venous plexus → drains into epidural venous plexus

Determinants of perfusion:

- cord perfusion pressure = MAP – CSF or spinal vein pressure (whichever is greater)
- where blood flow = perfusion pressure/ vascular resistance
- factors
  - resistance: pressure + metabolic autoregulation; ↑PaCO2 and ↓PaO2 ↓resistance
  - MAP: CO x SVR
  - CSF pressure: ↑brain parenchyma or ↑CSF vol (monro-kellie doctrine); posture; ITP

ACUTE RESPIRATORY FAILURE

Outline the causes of hypoxaemia/ Describe the physiological consequences of hypoxaemia

Hypoxaemia

- hypoxaemia = low partial pressure of O2 in blood
- ↓PaO2 and ↓SpO2 = PaO2 <60
- Classified according to aetiology
  - Hypoventilation
    - Leads to ↓PAO2 → ↓O2 partial pressure gradient across alveolar-capillary barrier → ↓PaO2
    - Leads to ↑PaCO2 (VA and PaCO2 are inversely related)
  - Diffusion limitation
    - ↓alveolar surface area
    - ↑alveolar capillary barrier thickness: pulmonary fibrosis; ARDS
    - NB diffusion limitation is rarely a cause of hypoxaemia
  - Shunt
    - Physiological: anatomical; functional
    - Pathological: intracardial; large communicating vessels; intrapulmonary shunts
    - V/Q mismatch

Effects

- Respiratory:
  - ↑PaCO2 + ↓PaO2 →
    - Detected by peripheral chemoreceptors (carotid + aortic bodies) →
    - stimulate ventilation via action on carotid and aortic body chemoreceptors →
    - stimulates respiratory centre → significantly ↑minute ventilation
    - hyperventilation stimulated at PaO2 <55mmHg, maximal at PaO2 <30mmHg
  - ventilatory response further augmented in presence of hypercapnoea
  - NB severe prolonged hypoxaemia has a depressive effect on the respiratory centre → apnoea
  - HPV
- Neuro:
  - cerebral acidosis (via anaerobic metabolism) → stimulates central pH receptors → ventilation
- Cardiovascular
  - Systemic vasodilation: ↑CO2, ↓MAP
  - Acidosis + ↑2,3DPG → shift HbO2 dissociation curve to right
- acid base changes
  - Hypoxia → anaerobic metabolism → lactate production → base deficit + ↓HCO3
- Renal
  - ↑EPO and Hct in chronic hypoxia

Define respiratory failure and differentiate between type 1 and type 2 respiratory failure

Resp failure

- When the resp system is no longer able to meet the metabolic demands of the body
- a failure of gas exchange resulting in abnormal ABG partial pressures
- PaO2 indicates the severity of resp failure whilst PCO2 indicates type
  - **Type 1 / hypoxaemic:** PaO2 <60mmHg when patient breathing room air + normal or ↓PaCO2
    - Failure of O2 exchange → hypoxic hypoxia
    - Typically 2o V/Q mismatch
  - **Type 2 / hypercapnic:** PaCO2> 50mmHg
    - Failure to remove CO2 →accumulation → resp acidosis
    - Typically 2o hypoventilation; may be extension of decompensated type 1
  - Acute or chronic

Major pathophysiologic causes of respiratory failure

- **low inspired partial pressure of O2**
  - ↓diffusion gradient of O2 across alveolar membrane
  - PaO2 = FiO2 x barometric pressure
  - E.xamples:
    - high altitude
    - hypoxic mixture of gas
- **Hypoventilation**
  - Failure of normal ability to exchange gas in the lungs with atmospheric gas → ↑PaCO2 and ↓PaO2
  - Examples:
    - Resp centre depression: drug injection, anaesthesia, head injury, encephalopathy
    - Disruption of resp signal during transmission along nerves to resp muscles: GBS, SC injury, MND
    - Dysfunction of NMJ: MG, NMBD
    - Dysfunction of muscles of respiration: myopathy, fatigue, malnutrition, dystrophy
    - Chest wall abnormalities: kyphoscoliosis, ank spond, pleural fibrosis
- **V/Q mismatch**
  - Aa gradient
    - Gradient between partial pressure of O2 in alveolus (PAO2) and arterial blood (PaO2)
    - PAO2 is estimated from alveolar gas equation:
      - PAO2 = FiO2 (PB – SVPH2O) – PaCO2 / R + F
      - Where FiO2 = fractional inspired O2 concentration
      - PB = barometric pressure
      - SVPH2O = saturated vapour pressure of water
      - PaCO2 = measured arterial partial pressure CO2
      - R = resp quotient
      - F = correction factor (small and usually ignored)
    - PaO2 is obtained from ABG
    - used to determine whether a shunt or diffusion abnormality is present
    - normal = <20mmHg

        - normal gradient implies that any hypoxaemia can be explained by hypercapniea; an ↑gradient suggests V/Q mismatch (shunt or dead space) or diffusion abnormality is causing the hypoxaemia
    - Causes of V/Q mismatch
        - Shunt
            - Perfused but not ventilated
            - Pathological shunt: alveoli collapsed or filled with pus/ blood/ fluid e.g. Pneumonia, pulmonary oedema, pulmonary haemorrhage, contusion, atelectasis
            - Anatomical shunt: R to L cardiac shunt
        - Dead space
            - Alveoli ventilated but not perfused
            - E.g. PE, hypovolaemia, poor cardiac function, ↑intrathoracic pressures (2° IPPV)
            - NB in practice, MV ↑s and PaCO2 remains relatively constant until patient no longer able to compensate → therefore this form of V/Q mismatch has little effect on PaO2

- **Diffusion abnormality**
    - ↓diffusion of O2 across alveolar-capillary membrane → ↓PaO2
    - less common
    - examples:
        - severe destructive disease of the lung e.g. late fibrosis diseases
        - severe APO
        - ARDS
        - Toxic or inflammatory pneumoinitis

Interpret blood gas analysis in respiratory failure

	Type 1 (acute metabolic acidosis)	Type 2 (acute respiratory acidosis)
pH	↓	↓
pO2	< 60	N or ↓
pCO2	N or ↓ (due to ↑MV)	> 50
HCO3-	↓ (buffering effect)	↑ (renal reabsorption)
Lactate	↑ (due to anaerobic metabolism)	N