WEEK 1

Lecture 1: Assessment and Management of Airway

Recognise causes of airway obstruction

- **Blockage in the airway → partially or totally prevent air from getting into lungs**
- Upper, lower, partial, complete, acute and chronic airway obstructions
  - Foreign body, infection (epiglottis, retropharyngeal abscess, bacterial tracheitis, diptheria, tetanus)
  - Tongue obstruction most common in unconscious patients
  - Immune angioedema, anaphylaxis, tumour, trauma (neck hematoma), poison and toxic exposure, laryngospasm, drug induced

Life threatening airway signs and symptoms
Dyspnoea, laboured respirations, decreased or no air movement, cyanosis, presence of foreign body in airway, trauma to face or neck, breathlessness/agitation/combativeness, unconsciousness

Understand how to manage the obstructed airway effectively while help arrives

Respiratory illness is common but many other conditions may cause respiratory distress

Respiratory assessment

- **History**: onset + duration of symptoms, cough/SOB, triggers

- **Inspection/observation:**
  - overall appearance, alert/orientated, active/hyperactive/drowsy, irritable
  - Colour (centrally and peripherally): pink, flushed, pale, mottled, cyanosed (late sign!), clubbing
  - Respiratory rate (most important vital sign), rhythm and depth (shallow, normal or deep)
  - Respiratory effort (WOB): mild, moderate, severe, inspiratory:expiratory ratio, SOB
  - Use of accessory muscles (UOAM): intercostal/subcostal/suprasternal/supraclavicular/substernal retractions, head bob, nasal flaring
  - Symmetry and shape of chest, tracheal position, tracheal tug, monitor O2 sat
  - Bleeding, vomitus, secretions, oedema

- **Auscultation**: listen for absence/equality of breath sounds, auscultate lung fields for bilateral adventitious noises e.g wheeze, crackles etc → listen for airflow! *Normal breathing is quiet, completely obstructed is silent, partial obstruction is noisy breathing*

- **Palpation**: bilateral symmetry of chest expansion, skin condition (temperature, turgor and moisture), capillary refill (central/peripheral), fremitus (tactile), subcutaneous emphysema

Respiratory management

1. Identify and remove any partial or complete airway obstruction
2. Position airway to maintain patency = insert oropharyngeal or nasopharyngeal airway
3. Protect cervical spine

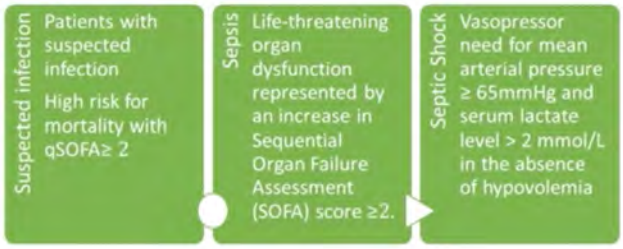
Understand the role of simple techniques and devices for airway management
Understand role of advanced airway management

Indications for airway management
- Apnea, obstruction (tongue, epiglottis, soft pallet, foreign body, vomit, food, laryngeal swelling, spasm)
- GCS <9, unstable mid face trauma, airway injury, respiratory failure, high aspiration risk, inability to maintain airway or oxygenation

Lecture 2: Sepsis

Basic pathophysiology of sepsis

- Organ dysfunction due to infection (medical emerg)
- Sepsis: body's systemic response to infection, can result in multi-organ failure and death → life-threatening organ dysfunction caused by dysregulated host response to infection
- Sources of infection (respiratory, GIT, urinary, hospital acquired), bacteria enter blood, leaking from blood vessels = organ dysfunction



Shock: life threatening condition that occurs when body is not getting enough blood flow meaning cells and organs do not get enough oxygen and nutrients to function properly. Types of shock:

- Hypovolemic: intravascular volume loss, haemorrhagic, fluid loss
- Cardiogenic: arrhythmia, AMI, valve failure, cardiomyopathy, pericarditis/PE
- Distributive: vasodilatory, decrease SVR, septic shock, anaphylaxis, neurogenic shock, drug, addisonian
- Obstructive: tension PTX, tamponade, PE

Identify risk factors, signs and symptoms + management actions

- Presentation is variable → depending on causative agent and portal of entry
- Low threshold of suspicion and early recognition of sepsis are essential for successful outcomes
- Thorough and timely history focuses on symptoms, comorbidities, recent surgery, recent antibiotic use, presence of medical devices, travel
- **At risk** = children/babies, elderly, post surgical, medical history

Signs and symptoms of SEPSIS: infection confirmed or suspected plus
- Temperature > 38.3C or <36C (normal temp does not exclude sepsis)
- Respiratory rate > 20/min
- Heart rate > 90/min
- Acute confusion or decreased level of consciousness
- Hyperglycaemia: blood glucose > 7.7 mmol/L in patient without diabetes
- Oliguria: urine output less than 0.5 mL/kg/hour

Signs and symptoms of SEPTIC SHOCK: infection confirmed or suspected plus
- Mottled or cold peripheries
- Cap refil time > 3 seconds
- Systolic BP < 90 mmHg or MAP < 60 mmHg
- Purpuric rash
- Arterial or venous lactate > 2 mmol/L
- Oliguria (urine output less than 0.5 ml/kg/hour)

Sepsis kills: Sepsis Pathway Assessment

Recognise
- Risk factors: recent surgery, indwelling medical device, immunocompromised, >65Y, fall
- S/S: fever, dysuria/frequency, cough/breathless, line associated infection/redness, abdominal pain, altered cognition

Respond and escalate
- Any red zone observation = call rapid response
- Two+ yellow zone observations (lactate > 2mmol/L in sepsis) = call clinical review
- Look for other common causes of deterioration and treat:
  - New arrhythmia, hypovoleamia/haemorrhage, DVT, atelectasis, AMI, stroke, overdose/over sedation

Intra-arterial monitoring
- Indications?
  - IV vasoactive medications
  - Interventional, pharmacologic or mechanical CV manipulation
  - Frequent ABGs or lab tests
  - Severe hypertension, induced hypotension, severe vasoconstriction or vasodilatation
  - Cardiac or major vascular surgery

Reliable measurements: equipment must be standardised to obtain reliable measurements. Transducers must be levelled and zeroed. Level = phlebostatic axis (4th intercostal space, maxillary line = right atrium)

Trouble shooting: damp tracings/inaccurate readings: is monitor in proper mode/setting? Is transducer at phlebostatic axis? Incorrect insertion? Are all connections tight and all 3 way taps in correct position? Bubbles in line or transducer? Occlusions or kinks in line? Poor patient positioning? Pressure bag maintained at 300mmHg?

Dicrotic notch: sharp rise indicates L ventricular systole. The rounded slope represents peak systolic pressure. Contraction diminishes and pressure drops
- Drip in waveform = dicrotic notch → represents the closure of aortic valve and the ascending slope indicates the beginning of diastole
- Arterial pressure waveform always occurs just after the depolarisation of the ventricles QRS complex

Information obtained:
- Systolic, mean and diastolic arterial pressure
- Myocardial contractility
- Systemic vascular resistance: low dicrotic notch suggests rapid aortic runoff
- Stroke volume: area under ejection phase, upstroke to dicrotic notch is proportional to SV
- HR and haemodynamic effects of alterations in rhythm

Complications:
- Ischaemia, skin necrosis, infection (due to 3 ways taps source), aneurysm/arteriovenous fistula formation
- Thrombosis/embolism, air embolism: major complication → cerebral air embolism (retrograde flow associated with flushing)
- Haemorrhage: keep connections tight!

How can we prevent infection?
- Use aseptic techniques during insertion
- Remove catheter ASAP
- Use non-vented caps on all stopcocks
- Change line components as per policy
- Minimise system entry: use closed blood sampling, use continuous CO system

How can we prevent thrombosis? Use continuous flush device, remove catheter ASAP, use heparin in flush solution, use catheters with heparin coating

Nurse management
- Monitor waveforms, compare values with non-invasive BP
- Maintain system, check site and circulation + alarm limits

Right atrial pressure: CVP

Direct measurement of pressure in right atrium through catheter (normal range 2-8mmHg)
- During diastole, tricuspid valve open between RA and RV. Therefore, pressure reading of RA reflects pressure in RV. Gives us a reflection of RV preload or RV end diastolic pressure

Measurement of CVP: single, double, triple, quadruple lumen CVC. Pulmonary artery catheter. PICC lines. Vascath.

WEEK 11

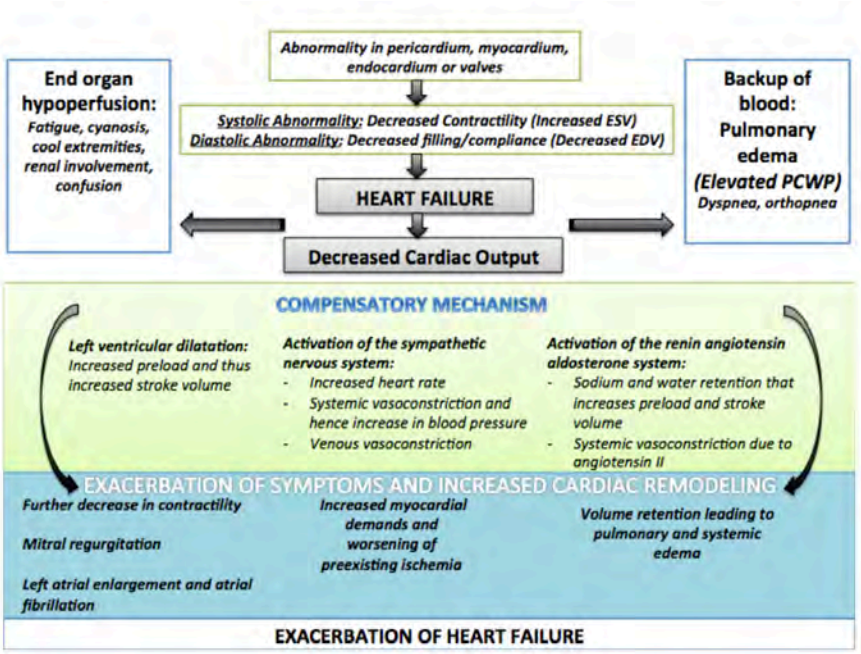
Lecture 1: Heart Failure

Describe the signs and symptoms of heart failure

Systolic HF (HFrEF): most common, weak ability for heart to contract in systole. Coronary artery disease and hypertension = main contributors to systolic HF

Diastolic HF (HFpEF): can occur with or without systolic HF, characterised by impairment of LV to fill during diastole as a result of slow or early relaxation of LV OR as a result of increased stiffening of myocardium which leads to higher filling pressures. Age, hypertrophy and ischemia are main contributors

Acute HF: rapid onset or worsening of symptoms and/or signs of HF. Life threatening medical condition requiring urgent evaluation and treatment = urgent hospital admission.
- May present as first occurrence, or as consequence of acute decompensation of chronic HF
- May be caused by primary cardiac dysfunction or precipitated by extrinsic factors
- Factors triggering AHF: tachyarrythmias, excessive rise in BP, infection, toxic substances/drugs, COPD exacerbation, PE, surgery complications



- Most commonly hypertensive, some hypotensive (poor prognosis when hypoperfusion also present)
- Congestion (L): orthopnoea, paroxysmal nocturnal dyspnoea, peripheral oedema bilaterally
- Congestion (R): gut congestion
- Hypoperfsuion: cold sweated extremities, oliguria, mental conduction, dizzy, narrow PP
- Hypotension, bradycardia, tachycardia, abnormal respiratory effort (>25 UOAM, or <8)
- Low O2 saturation, hypoxaemia, hyperaemic respiratory failure I and II, hypercapnia, acidosis, elevated blood lactate, oliguria

New onset HF: first presentation, acute or slow onset.
Transient HF: symptomatic HF over limited time period, although long-term treatment may be indicated (e.g caused by ishaemia and resolved by revascularisation). Episodic (usually resolved when cause is treated)
Chronic HF (decompensation) most common form: leading to hospital admission, not reversible, debilitating. Persistent. Stable, worsening or decompensated.

HF: clinical syndrome characterised by typical symptoms (breathless, ankle swelling, fatigue) that may be accompanied by signs (elevated JVP, pulmonary crackles, peripheral oedema) caused by structural and/or functional cardiac abnormality = reduced CO and/or elevated intracardiac pressures at rest and during