Chapter 2: respiratory failure
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Introduction
Respiratory failure is when the lung fails to achieve its gas exchange functions; to oxygenate and remove carbon dioxide. In this chapter we will talk about the types of respiratory failure, potential causes and therapy.

Causes of respiratory failure
It is often described that there are two types of respiratory failure; hypoxic (type I) and hypercarbic (type II). In reality, in most pathologies both of these concomitantly occur. However for the ease of classifying and understanding, we will divide them into the two categories.

Hypoxic respiratory failure (type I)
This is where the main pathology is hypoxia. Pathologically the reason for hypoxic respiratory failure is due to 3 mechanisms: Ventilation/perfusion mismatch, shunt and diffusion defects.

Ventilation perfusion mismatch (V/Q mismatch)
This is where alveolus of the lungs are perfused with blood through the pulmonary capillaries, but the alveolus has poor ventilation such that the blood is not oxygenated (1). There is a limit to how much oxygen blood can carry, therefore blood that has low oxygen content from poorly ventilated lungs cannot be compensated by blood that has high oxygen content as it passed through a well ventilated area of lung.

V/Q mismatch can occur when there is inflammation, mucus/pus, fluid and poor ventilation of the lung. Common causes of this within the ICU include:

- Pneumonia
- Pulmonary oedema
- Pulmonary embolism/fat embolism
- ARDS
- Atelectasis
- Bronchiectasis
- Pleural effusion
- Pneumothorax
- Causes of type II respiratory failure. Remember that poor ventilation will eventually lead to ventilation perfusion mismatch and therefore cause hypoxia.
Shunts
A shunt can be thought of an extreme form of V/Q mismatch, where there is no gas exchange of the blood as it flows from the right side of the heart to systemic circulation. Most things that cause V/Q mismatch can cause shunts, however an important cause of hypoxia that has not been mentioned is intracardiac lesions where there is right to left shunt. This is particularly important to be aware of in paediatric patients.

Diffusion defects
This is a less common cause of hypoxia in the ICU setting. Basically, the pathology is where something impedes the oxygen from being transferred from the alveolus to the haemoglobin. The potential pathologies that cause this include:

- Interstitial lung disease/fibrosis
- Pulmonary vasculitis
- Pulmonary hypertention
- Pulmonary oedema

Hypercarbic respiratory failure (type II)
This is where the problem is ventilation, rather than oxygenation. There is 2 main causes of this: central or peripheral. Central is where the problems lies with the respiratory centre in the brain stem, which controls ventilation by detecting PaCO2. Peripheral causes originate from outside of the control centre.

Central causes of hypercarbia
These are pathologies involving the CNS and include:

- Intracranial causes
  - Acute:
    - Bleeds
    - Infarcts
    - Infection
    - Trauma
  - Chronic
    - Obesity
    - Chronic lung disease (chronic hypercarbia)
- Extracranial causes
  - Drugs
  - Metabolic e.g. hypoglycaemia

Peripheral causes of hypercarbia
- Peripheral nerve lesions
  - Guillain Barre syndrome
  - Botulism
  - Critical illness myoneuropathy
Myasthenia gravis

• Chest wall
  - Eschar of the chest wall post burns

• Spine
  - Scoliosis

• Abdomen
  - Abdominal compartment syndrome

• Pleura
  - Pneumothorax
  - Haemorthorax
  - Pleural effusions

• Airway obstruction

• Lung
  - Asthma/COPD
  - Pulmonary oedema
  - Pulmonary embolus
  - Pneumonia
  - Severe shock

Therapies for respiratory failure
The first priority of all medical emergency is Airway, Breathing and circulation. Ensure the airway is secure, then proceed to manage breathing and circulation. In the first instance, administration of oxygen is your first line therapy, following which more advanced respiratory support can be instituted.

Wall oxygen
Standard wall oxygen is limited to the delivery 15L/min+ of 100% oxygen. This can be delivered to the patient through standard nasal prongs, at which a max rate of 4L/min can be delivered for prolonged duration, otherwise the nasal mucosa dries out rapidly. If more oxygen required, then a Hudson mask is used. This can be delivered at a minimal rate of 4-6L/min, otherwise end expiratory CO2 is not cleared and can be rebreathed. The actual percentage of oxygen delivered depends on the minute volume of the patient, but in a sick patient with high minute volume this may be as low as 30%, even when delivered at maximum 15L/min. This is because the peak inspiration flow of the patient will exceed 15L/min, causing air (which is 21% Oxygen) to be entrained, diluting down the oxygen concentration. A reservoir bag has been used in the form of non-rebreather masks, and these can increased inspired O2 to 60%+.

An ambubag bag can used attached to the wall oxygen supply. It contains a reservoir bag and if a good seal is achieved with the mask to patient, close to 100% oxygen can be applied. It also allows you to ventilate the patient if required and apply positive end expiratory pressure with the addition of a PEEP valve. It is portable and simple to use and therefore will be your “go to” device to support the ventilation and oxygenation of the patient if they fail the Hudson mask, and as you prepare for more advanced respiratory support, such as intubation and mechanical ventilation.
High flow humidified oxygen.
High flow circuits deliver O2 flows of 30L/min+ which is humidified, and there can deliver oxygen flow rates which exceeds the patient’s peak inspiratory flow. This provides three advantages:

1. The percentage of Oxygen delivered is controlled, and close to 100% can be delivered
2. When applied in the form of nasal cannula, small amounts of positive end expiratory pressure (also known as PEEP) can be applied to the patient
3. The oxygen is humidified, and therefore it is both more comfortable for the patients and also prevents drying of the respiratory system

Non-invasive ventilation
This involves applying positive pressure ventilation without an endotracheal tube. This requires a seal to be obtained between the mask (there are various types of masks) and the patient. There are specific indications for non-invasive ventilation, but it is also used in patients where invasive ventilation is inappropriate due to futility and triage.

NIV indicated in
1. COPD exacerbation
2. Pulmonary oedema (2) (3)
3. Obstructive sleep apnoeas

NIV can be considered in
1. Asthma (4)
2. Post-op atelectasis
3. Pneumonia in the immunosuppressed
4. Peripheral nerve pathologies (e.g. Mysthenia gravis)
5. Respiratory failure in patients not suitable for invasive ventilation due to futility

NIV is contraindicated in
1. Unconscious patients
2. Uncooperative patients
3. Mask seal cannot be obtained
4. Pneumothorax
5. High aspiration risk
6. Significant shock
7. Recent trauma or surgery involving the cribiform plate

Invasive ventilation
This section will be described in the next chapter.
References


