Hypoxia in Surgical Patients

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Introduction

This article discusses basic physiology of oxygen delivery, pathophysiology and mechanisms of hypoxia, the most common causes of hypoxia in surgical patients and principles of management. The aim is not to provide a detailed overview, but structure to enable early recognition, diagnosis and treatment.

Hypoxia is impaired tissue oxygenation. It is one of the most common post-operative complications but often not recognised because it is not looked for, e.g. post-operative confusion can often be secondary to hypoxia. Patients who are critically ill usually have increased oxygen demands; oxygen delivery is therefore fundamental to managing sick patients.

Physiology of Oxygen Transport

Oxygen Delivery = oxygen content x cardiac output, where

Oxygen content = (Hb x 1.34 x SaO2) + (0.0032 x PaO2). Fully saturated haemoglobin carries 1.34 ml oxygen/gram of Hb, but the constant can vary slightly. The maximum amount of oxygen that can dissolve in blood is 0.0032 ml/dl/mmHg PaO2. At Hb=15 and SaO2=98 blood carries 198ml O2/litre, of which 195 ml is carried as oxygenated haemoglobin.

Cardiac output depends on stroke volume and heart rate (CO = SV x HR); stroke volume is dependent on cardiac preload, contractility and afterload. Heart rate increases early with hypoxia. Peripheral perfusion and tissue oxygen delivery depend on cardiac output and peripheral resistance (BP = CO x PR).
Oxygen delivery therefore depends on:

- A patent and open Airway (see February 2012 review).
- Effective Ventilation (see March 2012 review):
  - Central drive, volume, rate, Functional Residual Capacity (FRC).
- Oxygen availability:
  - Percentage oxygen in inspired air (FIO2), oxygen pressure in the air and alveoli (pAO2), pulmonary capillaries (paO2).
- Oxygen transport:
  - Haemoglobin level (Hb), Cardiac output, Peripheral resistance. Each haemoglobin molecule can bind four oxygen molecules; binding of each molecule facilitates binding of the next until Hb is fully saturated, i.e. the affinity for the 4th oxygen molecule is much higher than for the 1st. This is the biochemical basis for the sigmoid shape of the Oxygen-Haemoglobin dissociation curve.
- Tissue factors:
  - Oxygen release, Diffusion, Utilisation. Oxygen release is enhanced by shifting the oxygen-haemoglobin curve to the right by a lower pH and higher temperature in active tissue (e.g. contracting muscles) and by higher levels of 2,3-DPG (raised by exercise, higher altitude).

Pathophysiology of Hypoxia

The following factors need to be maintained to prevent tissue hypoxia:

1. Patent airway
2. Effective ventilation
3. Effective gas interchange
4. Arterial oxygen saturation (SaO2) and pressure (PaO2)
5. Effective systemic and capillary circulation
6. Haemoglobin concentration and integrity
7. Effective oxygen release from Hb
8. Unhindered extracellular diffusion
Physiologically hypoxia is usually classified into four groups:\(^1\textsuperscript{2}\)

(a) Hypoxic hypoxia, when the pressure of oxygen in arterial blood (PaO\textsubscript{2}) is reduced, accompanied by decreased SaO\textsubscript{2} of haemoglobin. This is caused by inefficient gas exchange (when PAO\textsubscript{2} would be maintained) or decreased PAO\textsubscript{2}, e.g. at high altitude or suffocation due to airway obstruction or breathing in a closed space with loss of oxygen.

(b) Anaemic hypoxia, due to decreased oxygen carrying capacity in blood, e.g. due to loss of red blood cells (RBCs), inadequate Hb within RBCs or carbon monoxide (CO) poisoning. Oxygen binding sites on Hb have higher affinity for CO than O\textsubscript{2} which prevents oxygenation and patients do not show clinical symptoms and signs of hypoxia. In sickle cell anaemia the O\textsubscript{2}-Hb dissociation curve shifts to the right so that oxygen is released in the tissues more easily, compensating for a Hb of 6-8 g/l.

(c) Circulatory hypoxia, also known as stagnant or ischemic hypoxia, when too little oxygenated blood is delivered to the tissues. This can be localised, e.g. with acute arterial insufficiency, or general, e.g. with circulatory shock or cardiac failure.

(d) Histotoxic hypoxia, which means that oxygen delivery is normal but tissues cannot use O\textsubscript{2} due to toxins affecting cellular respiration, e.g. with cyanide poisoning. Methylene blue can be used in cyanide poisoning to bind cyanide molecules but this forms met-haemoglobin (where iron is reduced to Fe\textsuperscript{3+}), which has a much lower affinity for O\textsubscript{2}, limiting oxygen delivery.

In critical care the following mechanisms provide a practical mnemonic to think of potential causes of hypoxia:

1. ↓pAO\textsubscript{2}
   Alveolar PO\textsubscript{2} can drop significantly at altitude. In practice this is of importance when transferring patients with e.g. chest injuries, acute blood loss, shock or anaemia in unpressurised aircraft at high altitude. This is relevant in regions already at high altitude or crossing mountain ranges e.g. in parts of Ethiopia, the central great lakes states and South Africa.

2. ∆FiO\textsubscript{2}
   Patients on ventilators have their FiO\textsubscript{2} controlled artificially.
3. ↓V
Decreased ventilation will primarily cause CO2 accumulation. In normal patients this will increase the ventilation rate via chemoreceptors, but patients who are on artificial ventilation or are centrally depressed (e.g. due to opiate overdose) cannot mount this response. Hypoxia occurs late and can rapidly progress to cardiac arrest.

4. ∆V/∆Q
This means that there is discrepancy between ventilated alveoli and alveolar capillary perfusion, and there are two categories:
(a) Shunt: when alveoli are perfused but not ventilated (e.g. atelectasis, pneumonia), or oxygen diffusion is limited (e.g. in ARDS), right to left shunting is increased, i.e. more non-oxygenated blood reaches the systemic circulation. This is a significant cause of hypoxia in critically ill patients.
(b) Ventilation-perfusion mismatch: when alveoli are ventilated but not perfused, e.g. with pulmonary embolism (PE).

5. ↓CO
Hypovolaemic, cardiogenic or obstructive circulatory shock or congestive cardiac failure (CCF) can cause significant enough hypoxia to cause rapid death. Patients on artificial ventilation who are in CCF will usually not come off the ventilator unless cardiac function is supported (e.g. with inotropes).

Assessment of the hypoxic post-operative patient

Clinical Assessment
Hypoxia is the inability to effectively oxygenate the tissues and is a threat to life.
This may result from pathology of the airway, breathing or circulation.
Prompt responses are crucial, allowing accurate diagnosis and effective treatment.
A five-step, structured, sequential set of responses to hypoxia is:

1 Review: The primary assessment is a rapid, targeted clinical examination of airway, breathing, circulation and disability. This is conducted in correct order, with immediate management of a life threatening problem when discovered.
The most important skills at this stage are the use of the trained human senses to “look, listen and feel”, gathering important clinical information, informing diagnosis and acting
when necessary. Monitoring devices are useful when available, providing additional information. This information is only helpful to the patient when it is accurate, timely and used to guide effective treatment. Cutaneous pulse oximetry gives real-time data on the oxygenation of haemoglobin (saturation) and peripheral pulse rate. These signs may be lost with severe vasoconstriction, as in severe shock. Arterial blood gas analysis gives information on arterial oxygen tension (which is not “saturation”), carbon dioxide tension and blood acid-base status. Chest radiography and electrocardiography may provide additional data, but may detract from immediate, time critical interventions.

When critical hypoxia is revealed by the primary assessment, the next response, *Resuscitation*, is started without delay. Otherwise, the review phase may continue with the *secondary assessment*, aimed at gathering more information. A patient history is taken with attention to the acute and chronic aspects of the patient’s condition, with identification of specific symptoms and risks, such as asthma, smoking, heart disease etc. Review of current and past medication (including missed doses) is necessary. A clinical examination is done to identify signs of organ and system dysfunction. Chart review is crucial. Changes in vital signs can inform diagnosis and treatment. There is a further, *tertiary assessment*, but this is done upon completion of patient treatment. This is a review of the clinical process, identifying strengths and weaknesses, aimed at improving future care. This can be done in an educational setting and should be conducted in a supportive, positive way.

2 **Resuscitation:** This is started when the review phase shows an immediate threat to life. The resuscitation is also sequential and structured, aimed at restoration and maintenance of oxygen to the tissues, especially the vital organs. The stepwise approach is part of the Basic and Advanced Life support guidelines. Patients with hypoxia need oxygen. Resuscitation responses to common hypoxic problems are outlined later in this review.

3 **Request** HELP. Management of postoperative hypoxia can be complex and demanding. The chance of a successful outcome is increased when skilled help is sought and available to help manage the situation. A team approach requires good clinical leadership, situational awareness with effective task allocation. A key factor is concise, effective communication.
An “SBAR” communication format is helpful. A clinical record should be kept: when possible a team member can be given this task.

4 **Reassess** the situation regularly. The clinical picture will most likely change both with time and treatment and this is only apparent with reassessment. New information may be elicited or become available from other sources, which may alter further management.

5 **Resource:** your situation. Identify what is needed to improve the chance of a good outcome. This may involve acquisition of drugs, equipment or people to help with any of steps listed above.

**Pulse Oximetry**

Pulse oximetry is a valuable adjunct in the rapid assessment of peripheral oxygenation. It gives *an estimate* of percentage saturation on oxygen binding sites on Hb. It is related to PaO2 through the sigmoid shaped O2-Hb dissociation curve but should not be interpreted as direct substitute for PaO2.³

![Oxygen dissociation curve](image)

**Remember:**

Normal arterial blood has a saturation of only 97-98% due to physiologic shunt, but 95%-100% is normal on pulse oximetry for a patient on supplementary oxygen. A value <93% can be a warning and one should ask “Why?”. Unless there is a significant shift in the Hb-O2 dissociation curve, a PaO2 >8 kPa with a SaO2 > 90% usually means that the oxygen saturation is still on the plateau part of the curve. With a value of <90% the patient is in
serious trouble because the paO2-SaO2 ratio is now on the steep part of the curve and saturation will drop rapidly with a minor decrease in PaO2.

*Double check that you distinguish the SaO2 from the pulse rate when looking at the monitor.*

Error readings in pulse oximetry can occur due to:

- Low cardiac output
- Vasoconstriction
- SaO2 <70%
- Poor positioning
- Movement
- Hypothermia (often in trauma patients)
- Abnormal Hb (COHb, MetHb)
- Hyperthermic limb
- Dirty probe
- Black, blue or green nail polish
- External light

**Arterial blood gas analysis (ABGs)**

ABG analysis can be useful in the diagnosis and management of critical illness and injury, but waiting for results should not delay immediate management of potential hypoxia. The following account is a traditional interpretation. Another analysis, Stewart’s “Strong Ion Difference” approach is an alternative.

The ABG analyser measures:

- Hydrogen ion concentration, reported as either hydrogen ion concentration [H⁺] or pH (-log₁₀[H⁺]). A lower pH value is more acidotic
- Oxygen tension (PₐO₂), reported in kilopascals (kPa) or mmHg.
- Carbon dioxide tension (PₐCO₂) (kPa or mmHg)

Other values such as bicarbonate [HC0₃⁻] expressed in mmol l⁻¹ and Base Excess/Deficit (BE/D), are calculated. Base Deficit is the amount of base that would be needed to correct the pH of the sample to 7.4. Base excess is the amount of acid needed to correct to pH 7.4.
Normal Ranges\(\text{(SI units are preferred,\(\)) i.e not mmHg)\)}:

\[\text{[H}^+\text{]} \quad 40 +/- 4 \text{nmol l}^{-1} \quad \text{pH 7.4 +/- 0.04 (pH has no units)}\]

\(P_aO_2\) (breathing air, \(F_{O_2} 0.21\)) about 13.3 kPa (less with healthy ageing)

\(P_aCO_2\) 5.1 +/- 1 kPa = 40 +/- 5 mmHg (remains constant with healthy ageing)

\([HCO_3^-]\) 22 +/- 2 mmol l\(^{-1}\)

\(BD/BE\) +/- 2

\([H^+]\) gives the overall acid/base state for the patient. High \([H^+]\) is acidosis (acidaemia), low \([H^+]\) is alkalosis (alkalaemia) These states result from respiratory or metabolic causes, or a mixed pattern.

\(P_aO_2\) is a measure of arterial oxygen, a balance between oxygen delivery (a function of the cardiorespiratory system) and uptake by the tissues (aerobic metabolism). This varies normally with age and living at altitude, abnormally in cardio-respiratory disease.

The level of \(P_aCO_2\) is a balance between production (cellular aerobic metabolism) and clearance. \(\text{CO}_2\) is cleared in two ways. First, by ventilation (acute adaption over seconds) and second, by metabolic compensation (renal excretion) after conversion to \(HCO_3^-\) (chronic, over hours and days). \([HCO_3^-]\) level indicates the adaptive responses to acidosis or alkalosis. Low \([HCO_3^-]\) indicates acidosis, high alkalosis.

**Four Step Interpretation of ABG**

1. What is the \([H^+]\) (or pH)?

Acidosis if above normal, Alkalosis if below normal

2. What is the \(P_aCO_2\)?

If high, this is hypoventilation. If associated with a high \([H^+]\), it is respiratory acidosis.
If low, this is hyperventilation. If associated with a low \([H^+]\), it is respiratory alkalosis.

3. What is the base deficit or excess?

A base excess indicates metabolic alkalosis. A base deficit indicates metabolic acidosis.

4. What is the \(P_aO_2\)? If higher than 13kPa, additional oxygen is being given.
**Chest X-Ray**

Chest X-Ray is of no value in diagnosing hypoxia and should not delay immediate treatment, but can help with the diagnosis of specific conditions that cause hypoxia, especially of lung parenchymal disease and pleural or bony abnormalities after trauma (See section below and March 2012 review on chest trauma).

**Causes of hypoxia in post-operative patients:**

*Patients at risk of hypoxia*

- Pre-op hypoxia
  - Smokers, COPD
- Reduced FRC
  - Elderly, Obesity, Diabetes, General Anaesthetic
- Surgical pathology
  - Restricted ventilation, SIRS
- Post-op Sedation
- Hypothermia
- Fluid overload

*Common causes of post-operative hypoxia*

- Pulmonary oedema
- Bronchopneumonia
- Lobar pneumonia
- Pre-existent COPD
- Atelectasis with hypoventilation
- Pulmonary embolism
- ARDS

*Mechanisms of becoming hypoxic*

Hypoxia can occur via interference of physiological mechanisms of oxygenation at different levels, as discussed above. To keep things practical the causes of post-operative hypoxia are discussed in the following categories:

1. Lack of Alveolar Ventilation.
Adequate oxygen levels are prevented from entering the alveoli to facilitate gas exchange.

2. Lack of Alveolar Perfusion.
   - Inadequate levels of blood are supplied to the lung to facilitate gas exchange.

3. Decreased alveolar diffusion.
   - Adequate levels of both blood and oxygen are available in the alveoli and pulmonary circulation but alveolar pathology prevents gas exchange occurring.

**Lack of Alveolar Ventilation**

Alveolar ventilation represents the volume of gas available to the alveolar surface area per unit time. Lack of alveolar ventilation can therefore occur due to numerous mechanisms in the post-operative surgical patient, all of which should be considered during the assessment.

**Upper Airway Obstruction**

Airway obstruction must always be excluded as part of ABC assessment of the patient. Immediate clearance of an identifiable upper airway obstruction is necessary if identified.

**Clinical Features:** Features of upper airway obstruction are noted during the initial airway assessment. The airway may be noted to be blocked by a bolus. A patient with reduced consciousness may not be supporting his/her own airway and the soft tissues of the oropharynx impair airway efficiency.

Very rarely anaphylaxis will present post-operatively, with upper airway swelling and obstruction. In such a scenario the patient will be acutely unwell with additional bronchospasm and cardiovascular instability.

**Management:** Any identifiable obstruction should be immediately cleared if possible. Post-operative patients who become unwell with reduced level of consciousness may not be able to support their own airway effectively. A Guedel airway should then be inserted immediately. Further emergency assessment should be performed and the patient may require intubation and ventilation.
If anaphylaxis is suspected adrenaline should be administered immediately along with airway support, supplemental oxygen, intravenous hydrocortisone and intravenous fluid therapy. 7

**Respiratory Depression: Opiates and Carbon Dioxide Narcosis**

An often encountered complication in post-operative patients that can present as hypoxia is reduced ventilation secondary to reduced respiratory drive. The commonest causes to consider are impaired consciousness due to drug toxicity (most commonly opiates) and impaired consciousness due to carbon dioxide narcosis.

Opiate toxicity occurs readily in post-operative patients who often have significant analgesic requirements. An opiate ‘overdose’ need not occur with high drug doses. Patients with renal impairment do not excrete opiates effectively and accumulation can occur. The elderly are often also susceptible to such effects. In toxic concentrations respiratory depression occurs, with hypoventilation. 8

Carbon Dioxide narcosis occurs in patients with pre-existing Chronic Obstructive Pulmonary Disease (COPD). Respiration in some patients with COPD is dependent upon ‘hypoxic drive’, rather than hypercapnia. 9 If a patient is given high concentrations of oxygen, their respiratory stimulus is lost and respiratory depression occurs. The patient can quickly develop carbon dioxide retention, respiratory acidosis and reduced consciousness, causing hypoventilation and associated hypoxia.

**Clinical features:** The patient will present with signs of hypoxia, and reduced level of consciousness (GCS). A reduced respiratory rate will be noticed. In opiate toxicity pin-point pupils will be seen. In carbon dioxide narcosis a bounding pulse is often noted and a history of COPD. An arterial blood gas sample can be taken to measure carbon dioxide levels.

**Management:** Initial management is to secure the patients airway and offer respiratory support as necessary. Patients with CO2 narcosis will require non-invasive ventilation (CPAP, BiPAP) if conscious or intubation and ventilation for respiratory support to facilitate ‘blowing off’ excess CO2.

In patients with suspected opiate toxicity management should be similar but naloxone, an opioid antagonist, should be administered immediately. If the patient recovers quickly
always remember that the half life of naloxone is less than morphine, so the effect may wear off before the patient has metabolised enough morphine to prevent recurrent respiratory depression. Further doses may be required.\textsuperscript{10}

**Atelectasis and Lobar Collapse**

Atelectasis is a frequently encountered post-operative complication causing hypoxia. It usually occurs in the first 48 hours following surgery. In abdominal and thoracic surgery the normal mechanisms by which mucus is cleared is impaired by pain, inhibiting deep breathing and coughing. Mucus retention occurs with resorption of alveolar air, leading to alveolar collapse. This normally occurs in the basal lobes. Secondary infection can then occur.\textsuperscript{11}

**Clinical Features:** Patients with atelectasis may report dyspnoea and cough with signs of hypoxia, tachypnoea and have reduced bibasal air entry. Normally this is following abdominal or thoracic surgery. There may be an associated fever, though recent evidence questions the link between fever and atelectasis.\textsuperscript{12}

**Management:** Management of atelectasis includes assessment of degree of respiratory support required. In general supplemental oxygen therapy will be sufficient, but ventilation is required in extreme cases in a deteriorating patient.

An assessment of the patient’s analgesic requirements should be performed and this optimised to ensure they can breathe and cough without inhibition.

Chest physiotherapy is required to ensure clearance of mucus and secretions. It is also helpful in preventing secondary infection.\textsuperscript{13}

Antibiotic treatment is not initially required unless secondary infection is suspected.

**Pneumothorax**

Pneumothorax can occur in post-operative patients either spontaneously or as an iatrogenic complication of a procedure such as insertion of central venous catheter. For detailed assessment and management details see the March 2012 review on chest trauma.
**Bronchospasm**

Bronchospasm is a sudden constriction of bronchiolar muscle. It is stimulated by histamine release and degranulation of mast cells and basophils. Bronchospasm inhibits air entry and exit into the alveoli. It can occur in post-operative patients with pre-existing pulmonary conditions such as asthma or chronic obstructive pulmonary disease with contributing airway hyper-reactivity.\(^\text{14}\) It is also a feature of anaphylaxis.

**Clinical Features:** The main clinical feature of bronchospasm is wheeze on auscultation of the chest. Often a history of COPD or asthma will be identified.

**Management:** Nebulised bronchodilators should be administered to the patient. Nebulised salbutamol or ipratropium bromide are highly effective.\(^\text{15}\)

Very rarely an acute asthma attack will become severe and life-threatening. Under these circumstances immediate input from intensive care is required as the patient may require intubation and ventilation.

Once the patient is stabilised, the patient’s prescription chart should be reviewed. Non-steroidal anti-inflammatory drugs are frequently administered as analgesia post-operatively and may have been the provoking factor for bronchospasm. A new prescription of aspirin could also contribute.\(^\text{16}\) These should be discontinued.

**Lack of Alveolar Perfusion/ Ventilation Perfusion mismatch**

**Pulmonary Embolus**

Pulmonary embolus causes obstruction to the pulmonary vascular tree by an embolus, usually from a deep vein thrombosis of the pelvic or large leg veins. Inadequate pulmonary perfusion to adequately ventilated areas of the lung occurs, impairing gas exchange and causing hypoxia. Immobility, cancer and surgery are significant risk factors.\(^\text{17}\)

The embolus is not always thrombus; air embolism can occur following insertion of central venous catheters and rarely fat embolism can occur in patients who have sustained long bone fractures.
Clinical Features: Pulmonary embolus can present in a variety of fashions. Small, subclinical emboli can occur without any symptoms. In general, the larger the embolus and larger the ventilation/perfusion mismatch, the more profound the symptoms.

A massive pulmonary embolus classically presents with a collapsed, haemodynamically unstable patient who may have just visited the toilet (the straining dislodging the distal thrombus). Smaller emboli can present with dyspnoea, pleuritic chest pain and haemoptysis with signs of hypoxia. Other clinically useful signs are: 

- Tachycardia
- Tachypnoea
- Signs of DVT
- Low grade fever
- New onset arrhythmia

Investigations of use in identifying pulmonary embolus include an ECG which may demonstrate sinus tachycardia, atrial fibrillation, signs of right heart strain or the classic S1Q3T3 pattern. None of these are specific for pulmonary embolus however. A CT Pulmonary Angiogram or Ventilation Perfusion scan are diagnostic if the patient is stable. In the unstable patient with diagnostic doubt, a portable bedside echocardiogram can be performed. Evidence of right heart strain and signs of increased pulmonary artery pressure are indirect signs of pulmonary embolus.

D-dimer testing is not useful in post-operative patients with pulmonary embolus as surgery increases serum levels.

If fat embolism is suspected urine can be sent for microscopy, where fat cells can be identified.

Management: Management of pulmonary embolus is dependent on the patient’s clinical condition. If the patient is collapsed immediate resuscitation is necessary, with airway support, 100% supplemental oxygen and parenteral fluid therapy. If the patient can be stabilised, or in a non-collapsed patient, investigations to confirm the diagnosis should be performed.
Heparinisation under such circumstances is essential. A continuous infusion of unfractionated heparin is currently recommended for massive pulmonary embolus, while low molecular weight heparin is used in other cases. Adequate physiological support should be provided.\(^2\) Thrombolysis has also been recommended in collapsed patients with massive pulmonary embolus. Absolute contraindications to this are active gastrointestinal or intracranial bleeding. Recent surgery is a relative contraindication. The risk of bleeding must be balanced against the risk of cardiovascular instability caused by pulmonary embolus.\(^2\) Long term management in an established diagnosis of pulmonary embolus is anticoagulation therapy with warfarin for a period of 6 months.

The management of fat embolus is supportive therapy. Anticoagulants and thrombolysis have no role.

**Decreased Alveolar Diffusion**

**Pneumonia**

Pneumonia is a disorder marked by inflammation of the lungs, most commonly caused by bacteria in post-operative patients. Lung inflammation prevents adequate gas exchange, despite adequate ventilation and perfusion. Progression can lead to segmental bronchial collapse, reducing oxygenation further by preventing alveolar ventilation.

Colonisation of the lung with pathogenic bacteria due to aspiration of contaminated secretions, combined with relative immunosuppression due to surgery make this a common postoperative complication.

Distinction between hospital acquired and nosocomial pneumonia is essential in guiding antimicrobial therapy. Hospital acquired pneumonia is defined as pneumonia which occurs after 48 hours in hospital.\(^2\)

**Clinical Features:** A working diagnosis of postoperative pneumonia can be made in the presence of 3 or more of the following features without any other obvious cause: cough, sputum production, dyspnoea, chest pain, temperature >38 degrees Celsius and tachycardia.\(^2\)
Management: Initial management is supportive, with administration of oxygen and intravenous fluid therapy. Physiotherapy is essential to allow the patient to clear secretions from the lung.\textsuperscript{25}

Antimicrobial treatment is based upon local sensitivities. Typically postoperative pneumonia is poly-microbial, the majority being caused by gram negative aerobes. The most commonly isolated organisms are \textit{Pseudomonas aeruginosa}, \textit{Enterobacter} species, \textit{Klebsiella pneumoniae}, \textit{Acinetobacter} species, \textit{Serratia} and \textit{Citrobacter} species. In the absence of positive culture penicillin and an aminoglycoside should provide adequate cover.\textsuperscript{23}

The postoperative patient with pneumonia should be frequently reassessed to ensure that no further deterioration occurs. If the patient’s condition worsens despite full supportive management and he/she appears to be tiring (especially with worsening tachypnoea), input from intensive care should be sought as the patient may require ventilatory support.

Pulmonary Oedema

Pulmonary oedema is accumulation of fluid in the lung parenchyma. It prevents effective diffusion of gas between the alveoli and pulmonary circulation, leading to hypoxia.

Pulmonary oedema can occur readily in postoperative patients. It is generally cardiogenic, as a result of:

- Acute deterioration in cardiac function:
  - Myocardial infarction, acute coronary syndrome or arrhythmia.
- Fluid Overload:
  - Excessive parenteral fluid therapy can cause left ventricular dilatation and cardiac failure.
  - With renal failure this can occur more readily.
  - Patients with pre-existing cardiac disease are more susceptible to this complication.

Clinical Features: The patient may appear to be in respiratory distress with dyspnoea, tachypnoea and signs of hypoxia. They may also have tachycardia, and a narrow pulse pressure. The patient may be hypotensive. An elevated Jugular Venous Pulse may be
noted, or an elevated central venous pressure if monitoring is in place. On auscultation of the chest inspiratory crackles will be heard.  

Fluid charts should be assessed for a discrepancy between urine output and volume of parenteral fluid administered. This may give a clue that the patient's deterioration is due to pulmonary oedema.

In acute deterioration, myocardial infarction should be strongly considered as a possible provoking cause. An ECG is essential, along with cardiac enzymes.

A chest x-ray may demonstrate features of interstitial oedema, Kerley B lines, a bats-wing appearance and upper zone diversion.  

**Management:** Initial management requires supportive therapy, sitting the patient upright and administering 100% oxygen.

When the diagnosis is confidently established, frusemide 40 to 80mg can be given intravenously, and diuresis and clinical response monitored.

Intravenous nitrates can also be given if the systolic blood pressure is greater than 90mmHg. These cause peripheral vasodilation and reduce cardiac pre-load, improving function. An isosorbide dinitrate infusion at 2-10mg/h titrated to blood pressure should be given.  

Low dose intravenous morphine (given as intermittent 1-2 mg IV boluses) will decrease pulmonary venous pressure and buy time until other therapies take effect.

The patient should then be reassessed. If there is no clinical improvement, a further bolus of frusemide should be given and either non-invasive ventilation (CPAP or BiPAP) or intubation and ventilation considered.

**ARDS**

Adult Respiratory Distress Syndrome (ARDS) is a condition characterised by inflammation of the lung parenchyma causing impaired gas diffusion. It normally presents as a sequel to severe systemic inflammatory response syndrome (SIRS), when release of pro-inflammatory cytokines causes macrophage activation and neutrophil recruitment in the lung. This causes local microvascular disturbance and parenchymal inflammation and oedema.
The inflamed lungs become ‘stiff’, reducing ventilator capacity and compounding the effect on respiratory function.  

ARDS should not be diagnosed in a post-operative patient who has had uncomplicated elective surgery, but other parenchymal causes of hypoxia should be considered. It is more likely in patients with profound sepsis (who may have had surgery to correct the cause), extensive trauma or massive blood transfusion.

Clinical Features: ARDS occurs within 24 to 48 hours of major injury, sepsis or non-infective SIRS (e.g. pancreatitis). The post-operative patient with ARDS will generally show signs of clinical deterioration with tachycardia, tachypnoea, hypotension and hypoxia or increasing oxygen requirements.

ARDS is characterised by:

- Acute onset and rapid deterioration
- Bilateral infiltrates on Chest X-Ray sparing costophrenic angles
- Pulmonary artery wedge pressure <18mmHg (If Swan-Ganz catheter in situ)
- Clinical evidence of left ventricular dysfunction
- A PaO$_2$: FiO$_2$ ratio of < 200mmHg (the gradient between inspired oxygen level and that detected in blood)
- The absence of cardiac disease

Management: ARDS causes severe respiratory problems. Whilst supportive management with 100% supplemental oxygen and cardiovascular support may temporise the condition, management with intubation and ventilation is almost inevitable. Mechanical ventilation allows for stabilisation of the patients respiratory condition while the systemic cause of ARDS is treated and reversed. Until the systemic cause is addressed, ARDS is likely to persist.

There are little specific treatments for ARDS, although steroids, nitric oxide and surfactant therapy have been investigated in small studies.
Summary

Mild to moderate hypoxia is a common surgical complication, often under-diagnosed. Severe life-threatening hypoxia is fortunately rare but needs rapid action to prevent death. A physiological approach guides rapid diagnosis of the pathophysiological cause of hypoxia and of support of oxygen delivery until a specific diagnosis of causative disease process can be made. Decision for intervention is based mainly on clinical assessment, with discretionary interpretation of chest X-rays, blood gases and pulse oximetry.

References


