Acute Respiratory Failure
Learning objectives

At the end of the session you’d be able to:

• Undertake rapid and concise assessment of respiratory system
• Initiate basic treatment for patient in acute respiratory distress
• Understand basic pathophysiology of respiratory system
Respiratory assessment – big ticket items
Single most important thing to do before interpreting CXR
ABG - the ‘GOLD’ standard

Hypoxia kills!
Acute Hypercarbia = Exhaustion
Acute Hypocarbia = working hard
Base excess
Lactate
A-a gradient
P/F index
Rule of thumb

In a healthy individual breathing air normal PaO₂ is over 75 mmhg

IE 3.5 to 5 times the inspired oxygen concentration

Eg at 40% FiO₂ your PaO₂ should be 140-200 mmhg
The partial pressure of oxygen in the lung alveoli depends on the **partial pressure of inspired oxygen** which is the percentage of inspired oxygen \( \times \) atmospheric pressure AND the patient's ventilation rate and effectiveness.

- **The Alveolar Gas Equation:**

\[
P_{A\text{O}_2} = P_{i\text{O}_2} - \frac{0.8}{0.8}
\]

- **Inspired Oxygen Pressure:**

\[
P_{i\text{O}_2} = F_i\text{O}_2 (P_b - P_{H2O})
\]

At sea level breathing room air (\( F_i\text{O}_2 0.21 \)):

\[
P_{A\text{O}_2} = 0.21(760-47) - 40/0.8 \approx 100 \text{ mmHg}
\]

If they are on 40\% O2 = 0.4 (713)-50 = 235
• When taking ABG’s do not take off the oxygen mask!
• Know the inspired \( O_2 \) and factor it into the alveolar gas equation
• Never abruptly remove oxygen
All critically ill patients require oxygen!

- High inspired oxygen concentrations *do not* depress ventilation in patients with acute respiratory failure.
- Rising CO$_2$ in these patients indicates fatigue, and a need for ventilatory support.
“Failure to correct hypoxia for fear of causing hypoventilation and CO₂ retention is unacceptable clinical practice.”

• Bateman & Leach, BMJ 2001
Judicious O₂ delivery......

except for the patient in extremis.
Work of breathing

- Airway resistance + chest wall elastance
- Increased resistance to breathing can only be sustained for a short time
- Muscle fatigue
Acute/exacerbation of Asthma

Clinical findings
- Tachypnoea, SOB, positioning, WOB, accessory muscle usage, fatigue, $\text{SaO}_2$
- Auscultation: wheezes, silent, speech
- Agitated, confused, combative
- Tachycardia, ?BP, warm, flushed,
- **PACE or ALS?**

Management
- Assessment (ABCDEFG)
- **Oxygen**
- Salbutamol (delivery gas?), atrovent, steroids,
- ABG/CXR
- Are they improving?
- Differentials?
  - Pneumothorax (T), PE, LVF
<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
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<tbody>
<tr>
<td>Near-fatal asthma</td>
<td>Raised PaCO₂ and/or requiring mechanical ventilation with raised inflation pressures(^{391-393})</td>
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<tr>
<td>Life threatening asthma</td>
<td>Any one of the following in a patient with severe asthma:</td>
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<tr>
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<td>- PEF &lt; 33% best or predicted</td>
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<td>- SpO₂ &lt; 92%</td>
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<td></td>
<td>- PaO₂ &lt; 8kPa</td>
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<td></td>
<td>- normal PaCO₂ (4.6 – 6.0 kPa)</td>
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<td>- silent chest</td>
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<td>- cyanosis</td>
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<td>- feeble respiratory effort</td>
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<td>- bradycardia</td>
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<td>- arrhythmia</td>
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<td>- hypotension</td>
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<td>- exhaustion</td>
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<td>- confusion</td>
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<tr>
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<td>- coma</td>
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<tr>
<td>Acute severe asthma</td>
<td>Any one of:</td>
</tr>
<tr>
<td></td>
<td>- PEF 33-50% best or predicted</td>
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<td></td>
<td>- respiratory rate ≥ 25/min</td>
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<td></td>
<td>- heart rate ≥ 110/min</td>
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<td>- inability to complete sentences in one breath</td>
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<td>Moderate asthma exacerbation</td>
<td>- Increasing symptoms</td>
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<tr>
<td></td>
<td>- PEF &gt; 50-75% best or predicted</td>
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<td></td>
<td>- no features of acute severe asthma</td>
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<tr>
<td>Brittle asthma</td>
<td>- Type 1: wide PEF variability (&gt;40% diurnal variation for &gt; 50% of the time over a period &gt; 150 days) despite intense therapy</td>
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<td></td>
<td>- Type 2: sudden severe attacks on a background of apparently well controlled asthma</td>
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</table>
Signs of impending arrest

- Exhaustion
- Unable to speak
- Confusion, agitation, reduced level of consciousness
- Rapid shallow breathing
- Feeble respiratory effort
- Silent chest
- Mottled skin
Severe asthma

• Adjunctive therapies (you *need* ICU)
  • CPAP ?
  • Magnesium
  • Ketamine
  • Heliox
  • Volatile anaesthetic agents
COPD
Severe COPD

- Tachypnoea
- Marked dyspnoea
- Pursed lip breathing
- Use of accessory muscles at rest
- Acute confusion
- New onset cyanosis
- New onset peripheral oedema
- Marked reduction in ADL’s
Why Hypoventilation Leads to Hypoxaemia

• The Alveolar Gas Equation:

\[
P_AO_2 = PiO_2 - \frac{0.8}{0.8}
\]

\[
PiO_2 = FiO_2 (P_b - P_{H2O})
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Oxygen therapy in COPD

• Usually don’t need high FiO₂ to correct hypoxia in acute exacerbation

• If fails, consider:
  • CCF, myocardial ischaemia
  • PE
  • pneumonia, aspiration, PTx
COPD

• Up to 25% of patients with a severe unexplained exacerbation of COPD will have a co-existing PE.

  • Tillie-Lebland, Marquette et al, Annals of Internal Medicine 2006
Oxygen therapy in COPD

• If CO$_2$ rises, or LOC decreases...

• Do not remove the oxygen!
  • severe hypoxia may result
Management of Hypoxia and Breathlessness

- ABCDEFG
- Give Oxygen
- Sit the patient up
- Reassure
- Consider bronchodilators
- Consider reversing opiates
- Consider need to call for help for ventilation support