4.2: Causes

The arterial \( pCO_2 \) is normally maintained at a level of about 40 mmHg by a balance between production of \( CO_2 \) by the body and its removal by alveolar ventilation. If the inspired gas contains no \( CO_2 \) then this relationship can be expressed by:

\[
paCO_2 \text{ is proportional to } \left( \frac{V_{CO_2}}{V_A} \right)
\]

where:
- \( V_{CO_2} \) is \( CO_2 \) production by the body
- \( V_A \) is Alveolar ventilation

An increase in arterial \( pCO_2 \) can occur by one of three possible mechanisms:

- Presence of excess \( CO_2 \) in the inspired gas
- Decreased alveolar ventilation
- Increased production of \( CO_2 \) by the body

\( CO_2 \) gas can be added to the inspired gas or it may be present because of rebreathing. Anaesthetists are familiar with both these mechanisms. In these situations, hypercapnia can be induced even in the presence of normal alveolar ventilation and normal carbon dioxide production by the body.

An adult at rest produces about 200mls of \( CO_2 \) per minute: this is excreted via the lungs and the arterial \( pCO_2 \) remains constant. An increased production of \( CO_2 \) would lead to a respiratory acidosis if ventilation remained constant. The system controlling arterial \( pCO_2 \) is very efficient (ie rapid and effective) and any increase in \( pCO_2 \) very promptly results in a large increase in ventilation. The result is that increased \( CO_2 \) production almost never results in respiratory acidosis.
It is only in situations where ventilation is fixed that increased production will cause respiratory acidosis. Examples of this would be a ventilated patient who develops acute malignant hyperthermia: the arterial pCO$_2$ will rise unless the alveolar ventilation is substantially increased.

Most cases of respiratory acidosis are due to decreased alveolar ventilation.

The defect leading to this can occur at any level in the respiratory control mechanism. This provides a convenient way to classify causes that is used in the following table.

Alveolar hypoventilation may impair oxygen uptake.

The degree of arterial hypoxaemia will be related to the amount of hypoventilation. Increasing the percent of oxygen in the inspired gas can completely correct the hypoxaemia if hypoventilation is the only factor involved. If pulmonary disease leading to shunt or ventilation-perfusion mismatch is present, then the hypoxaemia will not be so easily corrected. The following list classifies causes by the mechanism or site causing the respiratory acidosis.

<table>
<thead>
<tr>
<th>Causes of Respiratory Acidosis (classified by Mechanism)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A: Inadequate Alveolar Ventilation</strong></td>
</tr>
<tr>
<td><strong>Central Respiratory Depression &amp; Other CNS Problems</strong></td>
</tr>
<tr>
<td>• Drug depression of resp. center (eg by opiates, sedatives, anaesthetics)</td>
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<tr>
<td>• CNS trauma, infarct, haemorrhage or tumour</td>
</tr>
<tr>
<td>• Hypoventilation of obesity (eg Pickwickian syndrome)</td>
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<tr>
<td>• Cervical cord trauma or lesions (at or above C4 level)</td>
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<tr>
<td>• High central neural blockade</td>
</tr>
<tr>
<td>• Poliomyelitis</td>
</tr>
<tr>
<td>• Tetanus</td>
</tr>
<tr>
<td>• Cardiac arrest with cerebral hypoxia</td>
</tr>
<tr>
<td><strong>Nerve or Muscle Disorders</strong></td>
</tr>
<tr>
<td>• Guillain-Barre syndrome</td>
</tr>
<tr>
<td>• Myasthenia gravis</td>
</tr>
<tr>
<td>• Muscle relaxant drugs</td>
</tr>
<tr>
<td>• Toxins eg organophosphates, snake venom</td>
</tr>
</tbody>
</table>
- Various myopathies

**Lung or Chest Wall Defects**

- Acute on COAD
- Chest trauma - flail chest, contusion, haemothorax
- Pneumothorax
- Diaphragmatic paralysis or splinting
- Pulmonary oedema
- Adult respiratory distress syndrome
- Restrictive lung disease
- Aspiration

**Airway Disorders**

- Upper Airway obstruction
- Laryngospasm
- Bronchospasm/Asthma

**External Factors**

- Inadequate mechanical ventilation

**B: Over-production of Carbon Dioxide**

**Hypercatabolic Disorders**

- Malignant Hyperthermia

**C: Increased Intake of Carbon Dioxide**
Rebreathing of CO₂-containing expired gas

Addition of CO₂ to inspired gas

Insufflation of CO₂ into body cavity (eg for laparoscopic surgery)

The generalisation made in this section is that though there are three possible distinct mechanisms that can result in a respiratory acidosis, in clinical practice, nearly all cases are due to inadequate alveolar ventilation. This is a very important point. Nevertheless the rare causes should be considered especially in Anaesthetic and Intensive Care practice where patients are often intubated and connected to circuits. Particular issues here include:

- Malignant hyperthermia (MH) is an extremely rare but potentially fatal condition which occurs almost exclusively in Anaesthetised patients exposed to certain drugs
- Various circuit misconnections & malfunctions, or soda lime exhaustion, can result in significant rebreathing of expired carbon dioxide
- Patients who are paralysed and on controlled ventilation cannot increase their alveolar ventilation to excrete any increased amounts of CO₂ produced by the body (eg in hypercatabolic states such as sepsis or MH)
- Exogenous carbon dioxide is introduced into the body in certain procedures (eg laparoscopy) and this increases the amount of carbon dioxide to be excreted by the lungs
- Adding CO₂ to the inspired gas as a respiratory stimulant has resulted, albeit rarely, in adverse outcomes in the past. (This practice is now abandoned in modern Anaesthetic practice)

Continuous capnography monitoring is now mandatory in Anaesthetic practice.