4.4: Metabolic Effects

4.4.1: Depression of Intracellular Metabolism

As CO$_2$ rapidly and easily crosses lipid barriers, a respiratory acidosis has rapid & generally depressing effects on intracellular metabolism.

Hypercapnia will rapidly cause an intracellular acidosis in all cells in the body. The clinical picture will be affected by the arterial hypoxaemia that is usually present. The effects described below are the metabolic effects of hypercapnia rather than respiratory acidosis. Patients with respiratory acidosis can be hypocapnic if a severe metabolic acidosis is also present.

**Important effects of Hypercapnia**

- Stimulation of ventilation via both central and peripheral chemoreceptors
- Cerebral vasodilation increasing cerebral blood flow and intracranial pressure
- Stimulation of the sympathetic nervous system resulting in tachycardia, peripheral vasoconstriction and sweating
- Peripheral vasodilation by direct effect on vessels
- Central depression at very high levels of pCO$_2$

4.4.2: Importance of Cerebral Effects
The cerebral effects of hypercapnia are usually the most important.

These effects are:

- increased cerebral blood flow,
- increased intracranial pressure, &
- potent stimulation of ventilation.

This can result in dyspnoea, disorientation, acute confusion, headache, mental obtundation or even focal neurologic signs. Patients with marked elevations of arterial pCO$_2$ may be comatose but several factors contribute to this:

- Anaesthetic effects of very high arterial pCO$_2$ (eg > 100mmHg)
- Arterial hypoxaemia
- Increased intracranial pressure

As a practical clinical example, the rise in intracranial pressure due to hypercapnia may be particularly marked in patients with intracranial pathology (eg tumours, head injury) as the usual compensatory mechanism of CSF translocation may be readily exhausted. Any associated hypoxaemia will contribute to an adverse outcome.

4.4.3: Effects on Cardiovascular System

The effects on the cardiovascular system are a balance between the direct and indirect effects.

Typically, the patient is warm, flushed, sweaty, tachycardic and has a bouncing pulse.

The clinical picture may be modified by effects of hypoxaemia, other illnesses and the patient's medication. Arrhythmias may be present particularly if significant hypoxaemia is present or sympathomimetics have been used.

Acutely the acidosis will cause a right shift of the oxygen dissociation curve. If the acidosis persists, a decrease in red cell 2,3 DPG occurs which shifts the curve back to the left.

An arterial pCO$_2$ in excess of about 90 mmHg is not compatible with life in patients breathing room air.

Why?

This is because of the obligatorily associated severe hypoxaemia. The alveolar gas equation predicts an alveolar pO$_2$ of 37mmHg (and the arterial pO$_2$ would be lower than this) when the pCO$_2$ is 90mmHg:

$$ p_{A}O_{2} = [0.21 \times (760-47)] - \frac {90} {0.8} = 37mmHg \$$

Higher values of paCO$_2$ have been recorded in patients breathing an increased inspired oxygen concentration which prevents the hypoxaemia. Values up to about 260mmHg have been recorded with inadvertent administration of high
inspired pCO$_2$ but this is Guinness Book of Records stuff! High pCO$_2$ levels also have an anaesthetic effect.

**Hypercapnia -vs- Respiratory acidosis?**

Note that 'hypercapnia' and 'respiratory acidosis' are not synonymous as, for example, a patient with a severe metabolic acidosis and a concomitant respiratory acidosis could have an arterial pCO$_2$ less than 40mmHg.

However, most of the discussion of 'metabolic effects' on this page is more correctly the 'metabolic effects of hypercapnia' rather than respiratory acidosis per se. Despite this, even in the mixed disorder just mentioned, the effects of an elevated arterial pCO$_2$ are linear, so compared to the situation of a severe metabolic acidosis alone, the metabolic effects of the higher pCO$_2$ of the mixed acid-base disorder (ie with the concomitant respiratory acidosis) are mostly still relatively correct.