7.5: Compensation

The compensatory response is hypoventilation

It was believed that the peripheral chemoreceptors alone acted as the initial sensor responding to the rise in blood pH but further animal studies have indicated that metabolic acid-base disorders do cause a slow change in brain ISF \([H^+]\) and this change allegedly could be sufficient for account for the change in ventilation that occurs. This view is not accepted by all - see discussion in Section 2.3)

The hypoventilation causes a compensatory rise in arterial pCO₂ but the magnitude of the response has generally been found to be quite variable. More recent studies have almost invariably shown that hypoventilation does reliably occur in metabolic alkalosis.

Why is hypoventilation not always found?

This has been attributed to various problems with some of the older studies which did not account for the presence of conflicting factors, particularly those causing hyperventilation:

- **Hyperventilation due to pain** - in response to the stress of a painful arterial puncture. This could lower the measured pCO₂ during the procedure.

- **Hyperventilation due to pulmonary congestion.** Some patients with metabolic alkalosis due to diuretic use have subclinical pulmonary congestion sufficient to stimulate intrapulmonary receptors and cause tachypnoea and give a sensation of dyspnoea. This slight hyperventilation is sufficient to negate the rise in arterial pCO₂.

- **Hyperventilation due to hypoxaemia.** An associated hypoxaemia will stimulate the peripheral chemoreceptors and cause hyperventilation if the arterial pO₂ is below 50 to 55mmHg. This may not have been considered in early studies.
This common association of metabolic alkalosis with factors causing hyperventilation probably accounts for most of the past findings of variability of the change in arterial pCO$_2$. In effect, this is saying that many of these patients had a co-existent respiratory alkalosis.

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**The arterial pCO$_2$ can be quite high in severe cases**

It was also widely believed that the maximum value of arterial pCO$_2$ due to compensatory hypoventilation was 55 to 60mmHg. There is no doubt that this is wrong.

Arterial pCO$_2$ can rise higher than this and values up to 86mmHg have been reported in severe cases of metabolic alkalosis!

If hypoventilation is sufficient to cause hypoxaemia, this also may stimulate respiration via the peripheral chemoreceptors. As mentioned above, associated hypoxaemia is probably responsible for variability in the measured arterial pCO$_2$ in patients who also have a sufficiently low arterial pO$_2$. Patients who present with hypoxaemia and hypercapnia may be diagnosed with respiratory failure if the association with metabolic alkalosis is not appreciated. It is usually best in these patients to administer oxygen and to avoid intubation and ventilation.

A couple of cautions for severe cases:

- For patients that you do not intubate and ventilate: If significant hypoxaemia was present, its relief can remove the hypoxic respiratory drive with resultant hypoventilation and a rise in arterial pCO$_2$. This reveals the appropriate (in acid-base terms) physiological response but can cause concern.
- For patients that you intubate and ventilate: It is easy to render ventilated patients hypocapnic and this respiratory alkalosis can greatly worsen the alkalemia. Convulsions have occurred in such patients.

The expected pCO$_2$ due to appropriate hypoventilation in simple metabolic alkalosis can be estimated from the following formula:

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\text{Expected } pCO_{2} = 0.7[HCO_3^-] + 20 \text{ mmHg (range: } \pm5) \]

Note the wide variation allowed (ie a 10 mmHg range) because of the conflicting factors that affect ventilation (discussed above). This formula is used to determine if a coexistent respiratory acid-base disorder is present. For example, if pCO$_2$ is much lower than expected, a respiratory alkalosis is also present.