6.1: Definition

If there was no compensation and no other acid-base disorder present, then this must necessarily lead to an increase in arterial pH.

If there is no metabolic acid-base disorder present, then the actual measured arterial pCO$_2$ is compared against the standard reference value of 40mmHg.

If there is a co-existing metabolic acidosis, then the expected pCO$_2$ used for comparison is not 40mmHg but a calculated value which adjusts for the amount of change in arterial pCO$_2$ which occurs due to respiratory compensation. (The formula used is discussed in Section 9.3). This decrease in pCO$_2$ that occurs as compensation for a metabolic acidosis is not a respiratory alkalosis as it is not a primary process. For this reason, hypocapnia is not synonymous with respiratory alkalosis.

Key fact: A respiratory alkalosis is ALWAYS due to increased *alveolar* ventilation

Now, consider the following, which are also correct:

- A primary increase in total (or minute) ventilation does NOT always result in a respiratory alkalosis, and:
- Increased alveolar ventilation will NOT always result in a respiratory alkalosis

This may seem a bit confused but consider the following:
Firstly, note the difference between an increased minute ventilation and an increased alveolar ventilation.

Minute (or total) ventilation is the product of respiratory rate and tidal volume. Alveolar ventilation can be defined as the product of respiratory rate and (tidal volume minus physiological dead space volume). If, for example, a person has a large increase in dead space then minute ventilation can be much increased but alveolar ventilation could remain unchanged. It is only the alveolar ventilation that results in excretion of carbon dioxide. Any increased ventilation of dead space is 'wasted ventilation'.

The clinical relevance is that some patients may be clinically hyperventilating or have obvious respiratory distress but yet their arterial pCO$_2$ will not be decreased.

Secondly, hypocapnia does not necessarily mean a respiratory alkalosis.

The two possible situations are:

• hypocapnia (or increased alveolar ventilation) occurring as a primary process -this is a respiratory alkalosis, or:
• hypocapnia occurring as a compensatory response to a metabolic acidosis -this compensatory response is secondary so is not a respiratory alkalosis.

The practical point: If you look at a set of blood gas results and find a low arterial pCO$_2$ (hypocapnia): this indicates increased alveolar ventilation but this may be a compensatory response to a metabolic acidosis and hypocapnia from this cause is not a primary process, and so by definition is not a respiratory alkalosis.

This may sound a bit of a technical quibble but there are adverse effects of the alternative practice. For example, if all compensatory responses were considered an acidosis or an alkalosis then all acid-base disorders would tend to occur in pairs (such as a 'metabolic acidosis' and a 'respiratory alkalosis'). It would also mean that clinically significant diagnoses may be missed in patients with some mixed acid-base disorders. For example, a patient with both a metabolic acidosis and a respiratory acidosis could be interpreted as having a metabolic acidosis alone & the respiratory problem would be missed and lead to quite inappropriate treatment (eg large doses of sodium bicarbonate).