4.7: Assessment

The arterial pCO₂ value is used to quantify the magnitude of the alteration in alveolar ventilation (assuming CO₂ production is constant and inspired pCO₂ is negligible). The arterial pCO₂ alone is not satisfactory for assessing the magnitude of a respiratory acidosis in some cases. In particular, coexisting metabolic acid-base disorders cause compensatory changes in pCO₂ and these must be accounted for.

The best available quantitative index of the magnitude of a respiratory acidosis is the difference between the 'actual' pCO₂ and the 'expected' pCO₂.

Definition of Terms

- Actual pCO₂ - the measured value obtained from arterial blood gas analysis.
- Expected pCO₂ - the value of pCO₂ that we calculate would be present taking into account the presence of any metabolic acid-base disorder. If there is no metabolic acid-base disorder then a pCO₂ of 40 mmHg is taken as the reference point - ie we would use 40mmHg as the expected pCO₂.

The reason we have to allow for a metabolic acid-base disorder is that the pCO₂ value changes from 40mmHg due solely to the body's compensatory ventilatory response to a metabolic acidosis or alkalosis so just using a value of 40mmHg as normal would be wrong and lead us to incorrect conclusions.

With an acute metabolic acidosis, the body responds by increasing alveolar ventilation. This response is compensatory because hyperventilation results in a decrease in arterial pCO₂ which tends to return the arterial pH towards 7.4 partially correcting the acute deviation of plasma pH from normal. The value of pCO₂ at maximal compensation can be predicted...
using a simple bedside ‘rule of thumb’ and this calculated value is the ‘expected’ pCO₂ which we use to compare with the ‘actual’(measured) pCO₂ value.

If a metabolic disorder is present, we can calculate (using a simple formula) a new reference value of pCO₂ (the expected pCO₂) that we would expect that would be present with typical levels of respiratory compensation. We use this calculated ‘expected value’ to compare with the actual measured value.

You will now note as a consequence of this approach something that you might think to be rather odd: that is, it is possible for a patient to have a significant respiratory acidosis and yet be hypocapnic! This seems counter-intuitive if you wrongly considered that the terms ‘respiratory acidosis’ and ‘hypercapnia’ to be synonomous.

Example 4.7.1

Consider a patient with diabetic ketoacidosis who has a bicarbonate level of 8 mmol/l - clearly a severe metabolic acidosis - and a measured arterial pCO₂ of 40mmHg.

Using the formula in Section 5.5, we calculate (and so predict) that if the metabolic acidosis was the only acid-base disorder present, then:

Patient's expected CO₂= \((1.5 \times 8) + 8 = 20 \text{ mmHg})\)

But the ’actual’ arterial pCO₂ is 40mmHg then, as this is much higher than the expected value, we would decide that our original assumption that this was the only acid-base disorder present was wrong. In this example, a co-existing respiratory acidosis was present. The pH in this patient with a mixed acidosis would be much lower than it would be if only the metabolic acidosis was present.

As an exercise, use the Henderson-Hasselbalch equation to calculate the pH for both values of pCO₂). If we just accepted a pCO₂ of 40mmHg as 'normal' then we would have missed this significant second acid-base disorder. Of course, the term 'respiratory acidosis' is not just words to explain a number - there must be some problem present which would explain the relative hypoventilation in this patient. For respiratory disorders one tends to think of the lung first, but such disorders are frequently caused by an abnormality at another parts of the respiratory control pathway (eg muscle weakness, coma, airway obstruction)

A final point: There is a widespread use of the term 'respiratory alkalosis' to refer to the compensatory hyperventilation that occurs with a metabolic acidosis but this term is quite wrong in this situation. The terms 'acidosis' & 'alkalosis' refer to primary abnormal processes (by definition) and should never be used to refer to compensatory processes. (Refer to Section 3.1 for definitions & discussion).