

# What are you going to do about the base deficit?

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The management of elevated blood acid (metabolic acidosis) is poorly understood and standard therapies are often initiated without consideration of the underlying pathophysiology.

## WHAT IS BASE DEFICIT?

The concept of base excess/base deficit was introduced by Astrup and Siggard-Andersen in 1958 (Astrup and Severinghaus, 1986). The base deficit is the amount of strong base (in mEq/litre) that must be added to whole blood in vitro at 37 °C to achieve a pH of 7.4 at a pCO<sub>2</sub> of 5.3 Kpa. It is now commonly used as an index of the metabolic component of acid–base disturbances (normal range between –2 to +2 mEq/litre). A base deficit (negative base excess) occurs with metabolic acidosis (base needs to be added to neutralize whole blood) whereas a positive base excess indicates metabolic alkalosis.

The base deficit can be used to calculate the amount of bicarbonate required to neutralize a metabolic acidosis by using the equation: NaHCO<sub>3</sub><sup>-</sup> dose = (base deficit) x (weight in kg) x 0.3. However, although potentially conceptually useful, in many cases this is not the appropriate treatment (Sirker et al, 2002).

## METABOLIC ACIDOSIS

Metabolic acidosis indicates either an inability of the body to excrete the generated acid load (excess production or inadequate excretion) or loss of bicarbonate from the body. Classical acid–base theory based on the

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Henderson–Hasselbalch equation describes the inter-relationship between CO<sub>2</sub>, HCO<sub>3</sub><sup>-</sup> and H<sup>+</sup>. Additional calculation of the anion gap ((Na<sup>+</sup> + K<sup>+</sup>) – (HCO<sub>3</sub><sup>-</sup> + Cl<sup>-</sup>); normal range = 11–19 mmol/litre) allows identification of unmeasured anions which may be contributing to acidosis (e.g. ketones).

An alternative conceptual framework proposed by Stewart (1981) involves calculation of the three independent variables dictating pH: pCO<sub>2</sub>, strong ion difference (SID) and weak acids (predominantly albumin and phosphate). SID refers to the mathematical difference in charge between the strong ions which are virtually fully dissociated in solution (Na<sup>+</sup>, Cl<sup>-</sup>, K<sup>+</sup>, Ca<sup>2+</sup>, Mg<sup>2+</sup>, and for the purposes of this model lactate) and is normally between 38 and 42 mEq/litre (Sirker et al, 2002).

## CLINICAL RELEVANCE

Metabolic acidosis with an increased anion gap or increased SID occurs with accumulation of unmeasured anions. This is seen in failure of renal secretion of acid (e.g. renal dysfunction), increased endogenous acid production (e.g. lactic acidosis, diabetic ketoacidosis) administration of exogenous acids (e.g. alcohol, salicylate) and hyperalbuminaemia. Normal anion gap metabolic acidosis (normal SID) occurs with loss of bicarbonate and is associated with hyperchloraemia (e.g. diarrhoea, uretero-enteric diversion, renal tubular acidosis) and with acidifying agents containing measurable anion (e.g. sodium chloride, ammonium chloride). It is important to realize that infusion of 0.9% NaCl may result in metabolic acidosis and this may be associated with adverse clinical outcomes (Wilkes et al, 2001).

In critical care and anaesthesia occult hypovolaemia is common and increased base excess is frequently the result of tissue hypoperfusion, sepsis or systemic inflammatory response syndrome. Other causes, e.g. renal impairment or diabetic ketoacidosis, should always be considered, and measurement of lactate may help to confirm the diagnosis. Appropriate therapy is restoration of tissue perfusion by resuscitation to achieve adequate blood flow and pressure.

Administration of bicarbonate has been unpopular since Graf and Arieff (1986) reported that this worsened the clinical picture in canine lactic acidosis. They suggested that HCO<sub>3</sub><sup>-</sup> metabolism in the blood releases CO<sub>2</sub> which diffuses freely into cells thus worsening intracellular metabolic acidosis. Conversely in the case of normal anion gap metabolic acidosis administration of bicarbonate may be appropriate. Excess sodium administration leading to hypernatraemia should be avoided. In the stable or improving patient the possibility of doing nothing should be considered. **HM**

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