

## Acid-base balance

Blood hydrogen ion concentration lies within the range **pH 7.36—7.44**. The terms acidosis and alkalosis in clinical practice indicate a change or a tendency to a change in the pH of the blood in a particular direction.

In acidosis, there is an accumulation of acid or a loss of a base causing a fall or a tendency to a fall in the pH. The converse occurs in alkalosis.

### buffering systems

The pH of the blood is regulated and controlled by various buffering systems essentially consisting of:

- 1- Weak acids and bases, of which the most important is the **bicarbonate:carbonic acid ratio  $HCO_3:H_2CO_3$** .
- 2- The removal of carbon dioxide by the lungs and
- 3- The excretion of both acids and bases by the kidneys.

The ratio of bicarbonate to carbonic acid is normally 20:1. Alteration in this ratio alters the pH. A decrease in the ratio leads to increased acidity and vice versa.

The bicarbonate level can be altered by metabolic factors, while the carbonic acid level is subject to alteration by respiratory factors. Alteration of one is followed automatically by a compensatory alteration in the other, so that the ratio ( $HCO_3:H_2CO_3$ ) and therefore the pH of the blood remains constant.

### Terms and normal values

**PCO<sub>2</sub>** is a measurement of the tension or partial pressure of carbon dioxide in the blood. The normal arterial PCO<sub>2</sub> is 31—42 mmHg.

**P<sub>O<sub>2</sub></sub>** is a measurement of the tension or partial pressure of oxygen in the blood. The normal arterial P<sub>O<sub>2</sub></sub> is 80—110 mmHg.

Standard bicarbonate is the concentration of the serum bicarbonate. Normal levels are 22—25 mmol/litre.

### Alkalosis

#### **Metabolic alkalosis**

Metabolic alkalosis, a condition of base excess or a deficit of any acid other than  $H_2CO_3$ , *can be caused by:*

- 1) Excessive ingestion of absorbable alkali. This is not uncommon in patients who take proprietary indigestion remedies without medical supervision;
- 2) loss of acid from the stomach by repeated vomiting or aspiration;
- 3) Cortisone excess, usually the result of over-administration of adrenal corticoids, but occasionally due to Cushing's syndrome.

#### **Compensation is effected by:**

- (a) retention of carbon dioxide by the lungs; and
- (b) Excretion of bicarbonate base by the kidneys (alkaline urine).

#### **Clinical features.**

Alkalosis due to loss of acid from the stomach is the most common and most important. In its most typical form, it is seen in patients with pyloric stenosis in whom the loss of acid by repeated vomiting is often accentuated by the taking of medicines containing sodium bicarbonate. The most striking feature of

severe alkalosis is ***Cheyne—Stokes respiration*** with periods of apnoea lasting from 5 to 30 seconds. Tetany sometimes occurs. Subclinical degrees of alkalosis are recognisable only by a raised standard bicarbonate concentration. Severe alkalosis may result in renal epithelial damage and consequent renal insufficiency.

***Treatment.***

Metabolic alkalosis without hypokalaemia seldom requires direct treatment. The cause of the alkalosis should be removed where possible and a high urinary output encouraged.

***Hypokalaemic alkalosis***

Hypokalaemic alkalosis is seen in patients who have lost potassium and acid owing to repeated vomiting from pyloric stenosis. The low serum potassium causes potassium to leave the cell and be replaced by Na<sup>+</sup> and H<sup>+</sup> ions. The shift of H<sup>+</sup> ion into the cell causes intracellular acidosis and increases the cellular acidosis of the kidney cells.

***Treatment***

When hypokalaemia is sufficient to cause a metabolic alkalosis, the losses can be massive (> 1000 mmol). Replacement is a serious undertaking. It can be achieved gradually and relatively safely by supplementing intravenous fluids with 40 mmol/litre of KCl if the urine output is adequate. More rapid replacement (up to 60 mmol/hour) will require intensive monitoring and supervision with continuous ECG monitoring in a high dependency or intensive care unit.

***Respiratory alkalosis***

Respiratory alkalosis, a condition where the arterial PCO<sub>2</sub> is below the normal range of 31—42 mmHg, is caused most commonly in surgical practice by excessive pulmonary ventilation carried out upon an anaesthetised patient. Other causes are hyperventilation occasioned by high altitudes, hyperpyrexia, a lesion of the hypothalamus and hysteria.

Compensation, which depends on increased renal excretion of bicarbonate, usually is inadequate. During anaesthesia alkalosis is accompanied by pallor and a fall in blood pressure. In severe cases respiratory arrest follows.

***Treatment***

Respiratory suppression due to alkalosis is treated by insufflation of carbon dioxide.

***Acidosis***

***Metabolic acidosis***

Metabolic acidosis, a condition where there is a deficit of base or an excess of any acid other than H<sub>2</sub>CO<sub>3</sub>, occurs as a result of:

1-increase in fixed acids due to the formation of ketone bodies as in diabetes or starvation, the retention of metabolites in renal insufficiency, and the rapid increase of lactic and pyruvic acids by anaerobic tissue metabolism, following cardiac arrest.

2-loss of bases such as occurs in sustained diarrhoea, ulcerative colitis, gastrocolic fistula, a high intestinal fistula or prolonged intestinal aspiration.

**Clinical features.**

In severe acidosis, the leading sign is rapid, deep, noisy breathing. The hyperpnoea is due to over stimulation of the respiratory centre by the reduction in pH of the blood, and the physiological purpose of overbreathing is to eliminate as much as possible of the acid substance  $H_2CO_3$ . The urine is strongly acidic.

**Treatment.**

The commonest cause of an acute peroperative metabolic acidosis is tissue hypoxia and the correct treatment is restoration of adequate tissue perfusion. Treatment with bicarbonate solutions will correct the measured metabolic acidosis but not treat the problem. Indeed, as bicarbonate is rapidly converted into carbon dioxide intracellular acidosis may, in fact, get worse. The administration of bicarbonate solutions should be reserved solely for situations where bases have been lost or where the degree of acidosis is so severe that myocardial function is compromised.

The acute acidosis seen in prolonged cardiac arrest may require the infusion of 50 mmol of 8.4 per cent sodium bicarbonate solution.

***Respiratory acidosis***

Respiratory acidosis, a condition where the  $PCO_2$  is above the normal range, is caused by impaired alveolar ventilation.

In practice this problem most commonly occurs when there is inadequate ventilation of the anaesthetised patient, or when the effects of muscle relaxants have not fully reversed at the end of the anaesthetic. There is also a risk of respiratory acidosis when the patient undergoing surgery already has pre-existing pulmonary disease (e.g. chronic bronchitis or emphysema), and this is accentuated by thoracic and upper abdominal incisions.

**The anion gap**

This is a calculated estimation of the undetermined or unmeasured anions in the blood. It is sometimes used to establish the cause of a metabolic acidosis.  $Anion\ gap = (Na + K) - (HCO_3 + Cl)$ . The normal anion gap is 10–16 mmol/litre. An increased anion gap is seen in metabolic acidosis due to ketoacidosis, lactic acidosis, poisoning (salicylates) and renal failure. A normal anion gap is seen in metabolic acidosis due to renal tubular acidosis and loss of alkali due to diarrhoea, intestinal obstruction or intestinal fistula, and in the hyperchloraemia of ureterocolic anastomosis.

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