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Pathophysiology of acid base balance: The theory practice relationship

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Summary There are many disorders/diseases that lead to changes in acid base balance. These conditions are not rare or uncommon in clinical practice, but everyday occurrences on the ward or in critical care. Conditions such as asthma, chronic obstructive pulmonary disease (bronchitis or emphysema), diabetic ketoacidosis, renal disease or failure, any type of shock (sepsis, anaphylaxis, neurogenic, cardiogenic, hypovolaemia), stress or anxiety which can lead to hyperventilation, and some drugs (sedatives, opioids) leading to reduced ventilation. In addition, some symptoms of disease can cause vomiting and diarrhoea, which effects acid base balance. It is imperative that critical care nurses are aware of changes that occur in relation to altered physiology, leading to an understanding of the changes in patients' condition that are observed, and why the administration of some immediate therapies such as oxygen is imperative.

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Introduction

The implications for practice with regards to acid base physiology are separated into respiratory acidosis and alkalosis, metabolic acidosis and alkalosis, observed in patients with differing aetiologies. By understanding normal physiological principles and how they relate to clinical situations can enhance patient care. A good understanding of

the essential concepts of acid base physiology is necessary so that quick and correct diagnosis can be determined and appropriate treatment implemented.

The homeostatic imbalances of acid base are examined as the body attempts to maintain pH balance within normal parameters.

General principles of acid base balance

The primary function of the respiratory system is to supply an adequate amount of oxygen (O₂) to tissues and remove carbon dioxide (CO₂). The kidneys

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Table 1 The major body buffer systems

Site	Buffer system	Description
Interstitial fluid (ISF)	Bicarbonate Phosphate and protein	For metabolic acids Not important because concentration is too low
Blood	Bicarbonate Haemoglobin Plasma proteins Phosphate	Important for metabolic acids Important for buffering CO ₂ and H ⁺ Minor buffer Concentration too low
Intracellular fluid	Proteins Phosphates	Important buffer of extracellular H ⁺ Important buffer
Urine	Phosphate Ammonia	Responsible for most of titratable acidity Important—formation of NH ₄ ⁺ and hence excretion of H ⁺
Bone	Calcium carbonate	In prolonged metabolic acidosis

will excrete any excess acids or alkali. The respiratory and renal organs together with the buffering effects of blood maintain hydrogen ion (H⁺) concentration. H⁺ concentration is one of the most important aspects of acid base homeostasis. When there is an increase or decrease in acid production, blood bicarbonate (HCO₃⁻), proteins, and phosphate buffer body fluids (Table 1). However, there comes a point in the disease process when these buffers can no longer maintain appropriate concentrations of H⁺. Patients admitted to hospital can have life threatening situations such as diabetic ketoacidosis, asthma, severe vomiting, which alter pH balance and exacerbate their problems.

To maintain homeostasis during stress/strenuous exercise and/or illness/diseased states there is generally an increase in depth and rate of breathing due to stimulation of the sympathetic nervous system (Richardson, 2003). High alveolar ventilation brings more O₂ into the alveoli, increasing O₂, and rapidly eliminating CO₂ from the lungs (for chemical abbreviations see Table 2).

Table 2 Chemical abbreviations

Abbreviation	Interpretation
O ₂	Oxygen
CO ₂	Carbon dioxide
kPa	Kilo pascals
PO ₂	Partial pressure of oxygen
PCO ₂	Partial pressure of carbon dioxide
H ⁺	Hydrogen ions
HCO ₃ ⁻	Bicarbonate ions
H ₂ CO ₃	Carbonic acid
Na ⁺	Sodium
K ⁺	Potassium
Cl ⁻	Chloride
NH ₄ ⁺	Ammonium

Partial pressure of gases

Dalton's law explains the partial pressure of a gas, which is the pressure exerted by a gas within a mixture of gases independent of each gas in the mixture (Marieb, 2004). The partial pressure of each gas is directly proportional to its percentage in the total mixture and in air is determined by atmospheric pressure. Atmospheric pressure is 101 kPa (760 mmHg), 21% of this air is oxygen, and the partial pressure of oxygen (PO₂) in atmospheric air is:

$$\frac{21}{100} \times 101 = 21.2 \text{ kPa}$$

Within the alveoli the PO₂ is different to air because of enrichment in the air passages (dead space) with CO₂ and water vapour. Alveolar air contains much more CO₂ and water vapour and much less O₂ and so makes a greater contribution to the near-atmospheric pressure in the lungs, then they do in air. This is due to:

- gas exchanges occurring in the lungs,
- humidification of air by the conducting passages,
- mixing of gases in the dead space (contains air not involved in gaseous exchange) between the nose and alveoli.

In alveoli, PO₂ averages only 13.2 kPa (100 mmHg). Continuous consumption of O₂ and production of CO₂ in the cells means that there is a partial pressure gradient both in the lungs and at the tissue level ensuring diffusion of oxygen into the blood and CO₂ from it (Waterhouse and Campbell, 2002).

Changes in partial pressures of carbon dioxide (PCO₂) and H⁺ are sensed directly by the respiratory centre central chemoreceptors in the medulla (Guyton and Hall, 2000). In contrast, a reduction

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