The highs and lows of ETCO₂

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THE PHYSIOLOGY OF VENTILATION

The rhythmic pattern of breathing is controlled by nuclei in the pons and medulla (the respiratory centres) but the output can be overridden to a certain extent by the cortex, for example, during periods of stress. These nuclei receive input from central and peripheral chemoreceptors.

Central chemoreceptors are situated near the ventral surface of the medulla and are sensitive to the partial pressure of CO₂ (PCO₂) of blood. They respond to the changes in the pH of extracellular fluid (ECF) or cerebrospinal fluid (CSF) that are caused by changes in PCO₂ as CO₂ diffuses from cerebral capillaries. Peripheral chemoreceptors are situated in the carotid and aortic body. They respond to decreased PO₂ and increased H⁺ (decreased pH) and PCO₂.

PCO₂ of blood is the primary factor controlling ventilation under normal circumstances. The peripheral chemoreceptor response is more rapid but less marked than the central chemoreceptor response. The hypoxic response (response to low PO₂) is small and is not really apparent until the patient is severely hypoxaemic.

Normal PaCO₂ in the conscious healthy patient is between 35 mmHg and 45 mmHg — this can be considered as normocapnia. Normocapnia is maintained by minute ventilation (minute volume) and thus by the respiratory frequency and tidal volume of the patient. A number of patient or disease factors can lead to hypoventilation (elevated PCO₂ or hypercapnia) in the conscious patient including: neurological disorders, obstructive upper airway disease, pleural space disease and severe pulmonary disease. These factors will also cause hypoventilation in an anaesthetized patient but even a healthy patient will hypoventilate to a certain degree under anaesthesia because almost all anaesthetic agents have respiratory depressant effects. This is likely to be due to depression of the respiratory centres via the GABA_A receptor effects of most anaesthetic agents. These effects are generally dose dependent and can compromise the animal's ability to adequately ventilate their lungs leading to an increase in PCO₂.

MONITORING VENTILATION

As CO₂ diffuses so rapidly from the blood into the alveoli it can be used as a measure of the adequacy of alveolar

ventilation. To get a true indication of how well an animal is ventilating, an arterial blood gas is required for the measurement of the partial pressure of arterial CO₂ (PaCO₂). However arterial CO₂ levels are approximately equal to alveolar CO₂ levels which can be more easily measured. Alveolar gases are the last gases to be expelled during expiration and therefore end-tidal CO₂ (ETCO₂) monitoring via capnography will give a breath to breath approximation of PaCO₂ and therefore the adequacy of ventilation. Hypoventilation can be classified as PaCO₂ (or ETCO₂) over 45 mmHg and some anaesthetists would instigate intermittent positive-pressure ventilation (IPPV) for any patient whose PaCO₂ rose above this level. Other anaesthetists would argue that the adverse effects of IPPV are worse than the adverse effects of mild hypoventilation, and the subsequent hypercapnia, so would instigate IPPV at a PaCO₂ (or ETCO₂) of 50–55 mmHg. This is termed permissive hypercapnia.

This lecture will cover the physiology of ventilation and how to use capnography in practice including types of capnography and troubleshooting.

KEY LEARNING OBJECTIVES

- Understand the physiology of ventilation
- Understand the use of capnography for the assessment of ventilation status in small animals
- Understand the types of capnography available and troubleshooting of the capnograph

MULTIPLE CHOICE QUESTIONS

1. Which of the following monitors is most important when monitoring ventilation?
(A) A suitably trained veterinary nurse
(B) Capnograph
(C) Pulse oximeter
(D) Arterial blood gases
2. What is considered to be normal ETCO₂?
(A) 35–45 cmH₂O
(B) 15–30 mmHg
(C) 35–45 mmHg
(D) 15–30 cmH₂O
3. What can hypoventilation be defined as?
(A) ETCO₂ <35 mmHg
(B) ETCO₂ >45 mmHg
(C) ETCO₂ <45 mmHg
(D) ETCO₂ >60 mmHg