1. Is hypercarbia the same as hypercapnia?

Yes! Both terms mean an abnormal elevation of carbon dioxide within the blood. The difference between the terms is in their etymology.

“Hypercapnia” is derived from Greek with “hyper” meaning “above” or “too much” and “kapnos” meaning “smoke”.

While hypercarbia still has the Greek prefix of “hyper”, the suffix, “carbia” is of a Latin derivation of “carbo” which means “charcoal”.

To logophiles, it is often considered improper to combine a Greek prefix and a Latin suffix, so hypercarbia is a less commonly used term. However, in practice the two terms are often used synonymously.

2. In a case of suspected raised intracranial pressure, should we be aiming for an ETCO$_2$ of 30 mmHg (I have always aimed for 35 mmHg)?

Assessment and management of end-tidal carbon dioxide is important when dealing with animals with suspected raised intracranial pressure such as patients with head trauma or probable intra-cranial neoplasia. We use end-tidal carbon dioxide as a surrogate marker for arterial carbon dioxide concentrations.

Arterial partial pressures of carbon dioxide (PaCO$_2$) influence cerebral vascular resistance. A low PaCO$_2$ will result in cerebral vasoconstriction, which reduces cerebral blood flow, while a high PaCO$_2$ results in vasodilation and increased blood flow.
As dictated by the Monroe-Kellie doctrine, when there is an increase in intra-cranial pressure, for example due to a space occupying lesion, if you can reduce cerebral blood flow (via reducing PaCO2 and inducing cerebral vasoconstriction) intracranial pressure may be reduced. Prolonged, severe hyperventilation (PaCO2 < 26 mmHg) should be avoided as it causes detrimental cerebral ischemia in human medicine. However, mild hypocapnia (~30 mmHg), achieved with intermittent positive pressure ventilation (IPPV) for short periods of time while other treatments are instigated, can be life-saving in an emergency setting of increased intracranial pressure (Leece 2016). Hypercapnia (PaCO2 > 45 mmHg) should be avoided in patients with intracranial pathology. If a patient is not showing any clinical signs of raised intracranial pressure, but it is suspected to be a possibility, then maintaining an ETCO2 of 35 mmHg is acceptable. However, if an animal shows signs of neurological decompensation; visualised either via advanced imaging or the clinical signs of bradycardia and a concurrent hypertension consistent with aspects of the Cushing’s triad, it is generally considered that PaCO2 should be maintained 30 – 33 mmHg with the use of IPPV.

3. **What length of time under anaesthesia is it safe to keep a patient with an ETCO2 of 30 mmHg when there are concerns regarding raised intracranial pressure?**

When inducing hyperventilation with positive pressure ventilation, we need to find a balance between the benefits of cerebral vasoconstriction for animals with raised intracranial pressure versus the risk of cerebral ischemia due to reduced cerebral blood flow and perfusion. Therefore, inducing mild-moderate hypocapnia is usually used as a potentially life-saving technique to “buy time” to perform other treatments. Hypocapnia can be induced very quickly and usually quite easily, allowing a time buffer while further treatments are prepared. This may include administration of mannitol or hypertonic saline, which if effective, can reduce the need to perform hypocapnia for a prolonged period of time.

4. **How high can the ETCO2 be for brachycephalics and still be “normal”?**

Control of ventilation (and therefore concentration of carbon dioxide in the body and thus end-tidal gases) occurs through central and peripheral chemoreceptor activation. Central chemoreceptors are highly sensitive to changes in hydrogen ion (and therefore PCO2) concentration. Increases in PaCO2 cause an increase in alveolar ventilation to result in an overall decrease in carbon dioxide concentration. Conversely, decreases in PaCO2 will reduce alveolar ventilation. This is what results in a normal end-tidal carbon dioxide level of 35 – 45 mmHg.
Brachycephalic animals tend to have an increased work of breathing due to their anatomical features. This increased work of breathing results in a reduced response of the central and peripheral chemoreceptors to changes in hydrogen ion (and therefore PCO2) concentration. The result is that these animals often hypoventilate and present with a higher PaCO2 and end-tidal CO2 than what we would typically consider normal. The level that becomes “normal” for that animal will depend upon the individual.

When dealing with a brachycephalic that is otherwise healthy and most importantly does not have intracranial disease, a lot of anaesthetists will be happy with an end-tidal CO2 to be reaching 50 – 55 mmHg before starting to treat the hypercapnia. For anything higher than 55 mmHg, we are likely to start instigating management changes to try and reduce this value. The changes that we make will depend upon the clinical situation. For example, a reduction in anaesthetic depth may be appropriate and result in an increase in minute ventilation. Otherwise, we may have to support the animal’s ventilation by providing positive pressure ventilation.

5. Why do you think anaesthetic mortality is higher in cats?

Studies have shown that the mortality associated with sedating or anaesthetising cats (0.24%) is greater than for dogs (0.17%) (Brodbelt 2006). It is hypothesised that this could be due to factors such as a smaller patient size, greater degree of subclinical co-morbidities (e.g. cardiac disease), increased difficulties surrounding tracheal intubation or increased likelihood of fluid overload due to a smaller blood volume compared to dogs.

Recent guidelines for feline anaesthesia have been published by the American Association of Feline Practitioners (Robertson et al. 2018). These are comprehensive and provide advice regarding all aspects of feline anaesthesia. They can be accessed at http://journals.sagepub.com/doi/abs/10.1177/1098612X18781391 (accessed 03/7/20).

References

