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#### Declaration of interest

Keith Simpson is managing director of Vetronic Services LTD, a company involved with design, manufacture and sale of ventilators and monitoring equipment. All information presented in this article relating to equipment is non-specific and does not favour Vetronic Services products over any others. Any photographs in this series of articles in which the Vetronic name appears are used to illustrate a physiological principle rather than for commercial gain.

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# Capnography for veterinary nurses

## Part Three: Interpretation

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### Introduction

This is the part that you have probably been keen to get to: how to get the information out of the capnograph. The previous articles have been about using the equipment properly to ensure that the information you get is reliable.

Interpretation of a capnogram should be made in a similar manner to interpretation of an ECG or an X-ray. There should be a logical, step-wise process in which each step can only be considered when the previous step has been successfully completed. Here is a suggested step-wise approach to capnogram interpretation:

- Step 1 – Is the waveform suitable for analysis? Yes/No?
- Step 2 – Are the values in normal range Yes/No?
- Step 3 – If the answer to Step 1 is yes, interpret the profile of the waveform in the light of the values seen in Step 2

### Step 1 - Is the waveform suitable for interpretation?

How do we know if the waveform is suitable for interpretation? Looking back at our previous discussions on the waveform profile, we know that we should be able to identify the four phases of the waveform. Now we know that Phase 0 can be indistinct and that Phase I doesn't really impart much information, so we will limit our criteria to Phases II and III.

For the waveform to be suitable you should be able to identify the following criteria:

1. a distinct plateau form to Phase III
2. a steady rise in Phase II

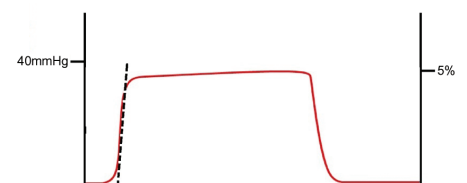
3. a definite 'knee' where Phase II changes to Phase III

If there is a good plateau at Phase III you know your sampling is good and you can proceed to Step 2. Sometimes there isn't an identifiable Phase III because it may be very short. In that case you must look at criteria 2 and 3 for waveform suitability. The following will make this clearer.

Check the profile of Phase II (**Figure 1**). Does it rise steadily in a linear fashion? The dashed line has been placed against Phase II and follows it closely. There is no suggestion that the line of Phase II is rounded. This is helpful in cases of patients with rapid respiratory rates, since there is often no time for a prolonged plateau phase and the capnogram appears as a series of fairly spikey waveforms.

**Figure 2** shows a capnogram from a rabbit breathing at 40 bpm. There is almost no time for an identifiable Phase III, but look at the nature of Phase II. It is still a sharp rise with only the beginning of a curve at the top of the waveform where it changes to Phase III. The trace is a faithful representation of what is going on in the airway.

Now look at the waveform in **Figure 3**. Again, there is no identifiable Phase III, but this time the rise appears to be a curve from the bottom of Phase II



**Figure 1.** The dashed line has been placed against Phase II and follows it closely: there is no suggestion that the line of Phase II is rounded. © Keith Simpson 2014. All Rights Reserved

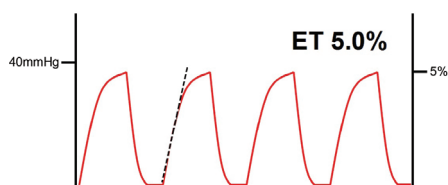


Figure 2. A capnogram from a rabbit breathing at 40 bpm: there is almost no time for an identifiable Phase III, but look at the nature of Phase II. © Keith Simpson 2014. All Rights Reserved

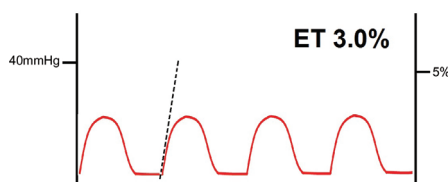


Figure 3. Again, there is no identifiable Phase III, but this time the rise appears to be a curve from the bottom of Phase II to the top. © Keith Simpson 2014. All Rights Reserved

to the top. It doesn't stay as a straight line for very long. Not only that, but it appears to fall in the same manner, so the waveform has the appearance more of a mole-hill than of a square wave. This is not a true representation of what is happening in the patient's airway. We know from the way the gas leaves the lungs that it can't have such a profile, so the profile must be an artefact. What you are seeing here is a dilution effect seen where the dead space causes dilution of the breath sample. This can be seen in mainstream and sidestream capnographs.

Figure 4 shows an example of a diluted capnogram recorded on a sidestream monitor. Because of the dilution effect, the reported end-tidal value will often be well below the actual end-tidal value, but you won't know how far below. Also, the waveform will often not return to zero because of the fact that the dead space is causing re-breathing.

Although the sampling system can effectively dilute and reduce CO<sub>2</sub>, it cannot add CO<sub>2</sub>, so if you see a reported inspired CO<sub>2</sub>, this must be taken as indicating re-breathing.

In summary, look for a plateau phase. If there is one, then the waveform is fine. If the respiratory rate is so fast that there is no distinct plateau phase, then use criteria 2 and 3 to establish that no dilution is occurring. If there is dilution then steps should be taken to improve the sampling system and or anaesthetic setup before continuing.

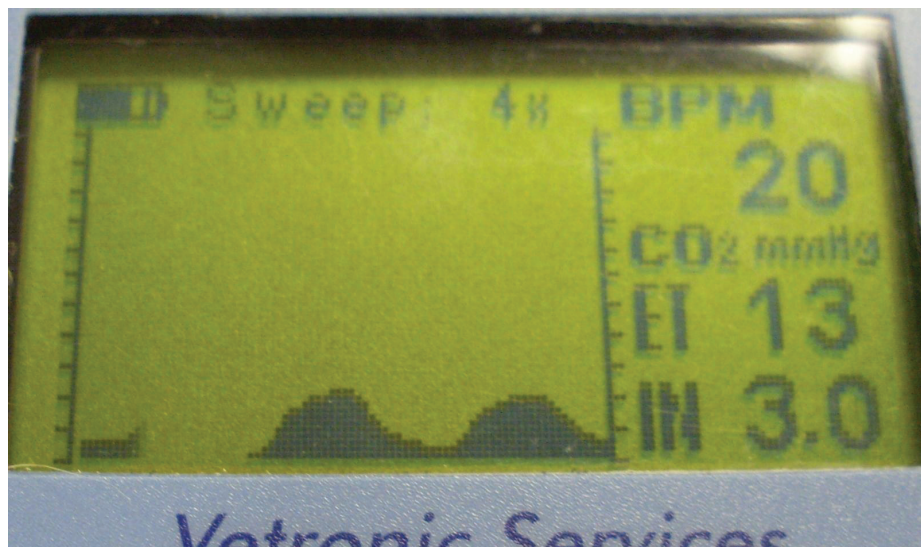


Figure 4. A capnogram on a sidestream capnograph showing the dilution effect. © Keith Simpson 2014. All Rights Reserved

Assuming that there is no dilution, proceed to Step 2.

## Step 2 - Are the reported values in the normal range?

So far we haven't discussed the reported values at all. In mammals it is generally accepted that a normal end-tidal CO<sub>2</sub> value should lie in the region of 35–40 mmHg, or 4.5–5.0%. In addition, we would expect an inspired value of, at or around zero. Often, for sampling and positioning reasons, the inspired value may be 1–2 mmHg (0.1–0.2%) but not more than that.

If the end-tidal values fall within that normal range the animal is in a state of *normocapnia*. End-tidal values above the normal range equate to a state of *hypercapnia* whilst values below the normal range indicate that the patient is in a state of *hypocapnia*.

Figures 5, 6 and 7 are typical capnograms showing the three different states.

### Normocapnia (Figure 5)

Here there is a clear Phase II, a clear transition to Phase III and a nice return to zero at the start of the next breath. The end-tidal CO<sub>2</sub> value can be seen to be around 40 mmHg or 5%.

### Hypercapnia (Figure 6)

This is still a clear capnogram that conforms to all of our profile criteria. The only difference here is that the end-tidal CO<sub>2</sub> value is clearly well in excess of 40 mmHg or 5%. It is approaching

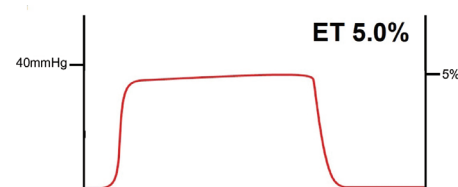


Figure 5. Normocapnia. © Keith Simpson 2014. All Rights Reserved

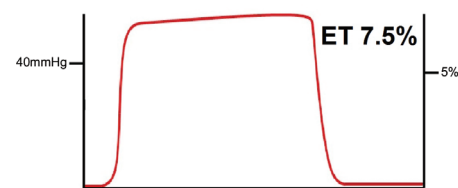


Figure 6. Hypercapnia. © Keith Simpson 2014. All Rights Reserved



Figure 7. Hypocapnia. © Keith Simpson 2014. All Rights Reserved

60 mmHg which is too high. This patient has a respiratory minute volume which is too low. The simple remedy here is to increase the minute volume by increasing the respiratory rate. If the animal is not being ventilated then some manual breaths should be given, or the patient should be started on a ventilator.

As discussed in Part One, the generation of CO<sub>2</sub> is constant for 99% of animals asleep under anaesthesia and on that basis we took our first rule of capnography – **CO<sub>2</sub> production is constant**. The only

time this may not be the case is if the animal has a changing metabolic rate due to fever, and, in particular, to an unusual condition called *malignant hyperthermia*. An increased body temperature raises the metabolic rate, so you should also expect the patient that has heat stroke to have an elevated CO<sub>2</sub> level. Malignant hyperthermia is a rare condition but can be associated with inhalant anaesthetics and stress. It has been reported in dogs (Greyhounds) as well as cats, particularly in well-muscled individuals.

The analysis of the curve in **Figure 6** is simple: the level in the arterial blood and hence lungs is high. Our patient is asleep so CO<sub>2</sub> production is constant. Therefore the only explanation for the hypercapnia is that the rate of removal from the lungs is not sufficient.

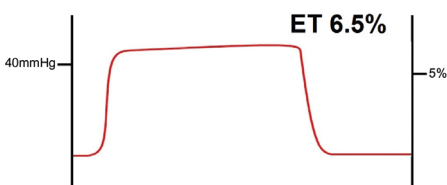
**Hypocapnia (Figure 7)**

Again, this is a nice clear capnogram with all three criteria that we look for during our assessment - it has a sharp rise to Phase II, a clear knee transition to Phase III and a clear plateau form to Phase III. However, the end-tidal value is well below the normal level and this patient is hypocapnic. Hypocapnia is unusual in our patients and is usually due to over-ventilation, most commonly as a result of intermittent positive pressure ventilation (IPPV). The minute volume needs to be reduced and this is most simply achieved by reducing the respiratory rate.

Again, the analysis is straightforward: the level of CO<sub>2</sub> in the arterial blood and hence the lungs is low. Our patient is asleep and so the CO<sub>2</sub> production rate is constant. Therefore, the rate of removal of CO<sub>2</sub> from the lungs is excessive.

**Rebreathing**

We have discussed how rebreathing can occur because of excessive dead space in the breathing system. How does this show up on the capnograph? There will be two indicators: first, the trace of the capnogram will not return to the zero line; second, the reported inspired CO<sub>2</sub> level will not be zero (**Figure 8**).



**Figure 8.** Rebreathing. © Keith Simpson 2014. All Rights Reserved



**Figure 9.** A good example of cardiogenic oscillation. © Keith Simpson 2014. All Rights Reserved

The capnogram itself may look quite normal, have normal slopes to Phase II and III but just not return to the zero line. What this means is that when the patient breathes in it takes in a portion of the previously expired CO<sub>2</sub>.

The inspired level of CO<sub>2</sub> here is about 1.5% or 12 mmHg, which is excessive. Notice that the end-tidal value has also been increased by a similar amount as the offset of the base line and will be reading 6.5% on the capnograph. Here efforts should be made to tackle the cause of the rebreathing as the end-tidal value will reduce to normal once that is done.

Common causes of rebreathing are:

- exhausted soda-lime in a circle system
- excessive dead space
- insufficient fresh gas flow (FGF) in a non-rebreathing system
- failing/missing expiratory valve in a rebreathing system

**Cardiogenic oscillations**

Cardiogenic oscillations are not uncommon and can be a normal occurrence in a lot of animals, especially deep-chested ones such as Greyhounds or Lurchers. The variations are small changes in CO<sub>2</sub> levels as the heart pushes against the lung as it fills and empties. This acts like a little mechanical ventilator on a small part of the lung which therefore adds its CO<sub>2</sub> with

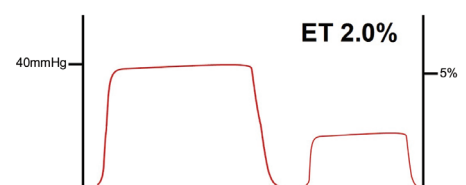
every little compression. What you are seeing is a very small part of the lung ventilating at a higher rate than the rest, and superimposing its waveform on the main waveform. Due to the variable nature of this effect, it will change with posture and little can be inferred from the waveform other than to acknowledge that it represents a normal effect.

We saw a nice example of cardiogenic oscillations in the Colobus monkey in Part One (**Figure 9**).

**Abrupt fall in CO<sub>2</sub> levels**

This is something to watch out for, as it can be an indicator of a serious problem. The capnogram shows a marked change in levels between successive breaths (**Figure 10**). This is not just hypocapnia, because the preceding breath was of a significantly higher value. So what has happened here?

If we analyse the capnogram we see that the form of the second waveform is quite reasonable. It has a good sharp rise to Phase II and a good plateau phase, so it would appear that it accurately



**Figure 10.** An abrupt fall in CO<sub>2</sub> level. © Keith Simpson 2014. All Rights Reserved

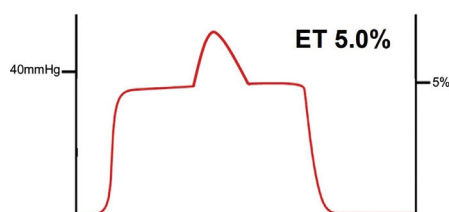
represents the exhaled breath. The height of the plateau phase at the end is an accurate reflection of arterial  $\text{CO}_2$  so it appears that arterial  $\text{CO}_2$  has suddenly fallen. Because our patient is anaesthetised and static, the drop in  $\text{CO}_2$  cannot be due to a fall in production, since that will remain constant. The fall must be due to a sudden drop in delivery to the lungs.

Such a capnogram is therefore seen when you get a sudden fall in cardiac output. This can be due to a surgeon pressing on the pulmonary artery or heart itself. It can also occur with iatrogenic pneumothorax when suturing the diaphragm during a ruptured diaphragm repair. Continued ventilation with the chest full of air causes the heart to be squeezed, with a dramatic fall in cardiac output as a result. If the animal is on a ventilator, a capnogram like the one in **Figure 10** could also mean that the patient has died and that you are seeing the last remnants of  $\text{CO}_2$  being washed out of the lungs by repeated ventilation.

So whenever you see this act quickly. Listen to the heart to make sure it is beating. If not, proceed to your cardiac arrest procedure. If it is still beating, try to determine quickly why the cardiac output should have fallen so suddenly. If this is at the end of a chest closure, make sure all air has been aspirated from the chest. If this is during an open-chest procedure, check that there isn't pressure on the heart or pulmonary artery from the surgeon or packing material.

## Superimposed spike or bump during Phase III

The final capnogram to discuss has many different forms, but they all follow the same pattern. What you see is a normal capnogram with a superimposed spike or bump along the plateau phase (**Figure 11**).



▣ **Figure 11.** A sharp spike in Phase III.  
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Analysing it we see that the waveform has a good Phase II and a sharp transition from Phase II to Phase III, so it is a good waveform. There is, however, a marked spike of  $\text{CO}_2$  during the plateau phase or Phase III. This must mean that during expiration a portion of the breath contained a higher level of  $\text{CO}_2$  than the rest of the breath. This higher level of  $\text{CO}_2$  will be found in areas of lung that are well perfused but underventilated and this will commonly happen with anything that causes gas trapping. It can be seen with foreign bodies or mucous blebs in smaller airways – anything that tends to hold gas in an area of lung.

### Single-lung intubation

Imagine also the effect of single-lung intubation, a situation not uncommon in smaller breeds intubated with relatively long Endotracheal tubes (ET). The ET tube can be advanced so far down the trachea that it reaches the bifurcation of the trachea and then advances into one side or the other. The seal on the intubated side is unlikely to be perfect, so as the patient breathes out, gas is entrained from the non-intubated lung and is pulled up the airway into the ET tube. It is unlikely that the intubation in one lung would be far enough advanced to allow cuff inflation in just that lung. If it were then that lung would be sealed off and there could be no spike on the capnogram. More likely is that just the tip of the ET tube enters one lung and the cuffed portion is still in the trachea. Because there is a poor seal around the tip of the ET tube, air passing up the ET tube creates a venturi-like effect, pulling in air from the other non-intubated lung. The gas in the non-intubated lung will be rich in  $\text{CO}_2$  at a higher level than normal. Depending on the degree of occlusion, this 'stagnant' gas may enter the ET tube at any time, and when it does, you will see a large step in the profile of Phase III.

Whether that rise returns to normal end-tidal values quickly and you see a spike, or it is maintained at the higher level until the next breath will depend on many factors, such as degree of occlusion, exhalation flow rate and whether the non-intubated lung is lowermost or not. It is not necessary to analyse the spike or step change that you see, only to recognise it for what it is – a release of high-level  $\text{CO}_2$  gas from one part of the lung.

Steps should then be taken to resolve it: either withdraw the ET tube slightly



▣ **Figure 12.** A spike in Phase 0. © Keith Simpson 2014. All Rights Reserved

or remove the ET tube and clean it of mucous, consider the possibility of a foreign body, change body posture to help reduce mucous accumulation in lower airways.

## Spike in Phase 0

There is a variant of this waveform that may look similar but in fact has a very different cause. This capnogram looks like the one just described, with one subtle difference. The upturn in  $\text{CO}_2$  is seen after Phase III has finished, in other words during Phase 0 (**Figure 12**). But Phase 0 is inspiration, so how can there be an increase in  $\text{CO}_2$ ?

Because this peak of  $\text{CO}_2$  is seen in inspiration, it must arise from the breathing system and not from the patient. In fact, this type of capnogram is seen in cases where there is a failure in the inspiratory valve to close properly, which results in partial rebreathing and allows a bolus of  $\text{CO}_2$  back into the ET tube. This will therefore not be seen on a non-rebreathing system, where there are no valves and the FGF is high. The capnogram can have different appearances depending on how the valve behaves. If the valve were leaking completely, there would be a capnogram showing rebreathing in a manner similar to what is seen in **Figure 8**.

## Conclusion

These are the most common capnograms that you will encounter in practice. If you come across something that doesn't match the profile of one of the above, try and analyse it in the way we have done here. Remember, your patient is asleep so it will have a steady  $\text{CO}_2$  production. Look at the profile of the waveform to see if it has the characteristics that you expect. Is the end-tidal value high or low? If the clinical circumstances allow and you are unsure of what the capnogram means, take a picture of it on your mobile phone and ask someone about it later.