Anaesthetic Monitoring. Will it wake up? Will it die?

This talk is given to a group of Zoological Veterinary Nurses and focuses on principles and situations encountered when dealing with exotic species. The emphasis is on safe anaesthesia which becomes more and more difficult the smaller the animal, so the talk highlights factors that particularly affect the smaller animals. With such a vast array of animal types it is impossible to cover all differences and variations encountered in each type. Therefore the talk aims to put forward good-practice principles that can be applied to any species. Where species differences are important they will be highlighted.

Will it wake up? Will it die?
From a practical standpoint this is the most fundamental question we can ask ourselves during any anaesthetic we are monitoring. And this is a question that you should strive to be able to answer at any time during an anaesthetic procedure.

Anaesthesia is not an exact science. If it were then any anaesthetic could be conducted by simply delivering a calculated dose of anaesthetic agent to a patient to produce the necessary depth of anaesthesia and for the required time. It would be easy.
But, anaesthesia is far from an ***exact*** science. However, it is a science. Unfortunately we do not yet know or understand all of the many factors that are involved during anaesthesia to be able to make accurate predictions about responses and behaviour during anaesthesia. For that reason we fly by the seat of our pants using all the knowledge we can to make anaesthesia as predictable as possible.

The purpose of this talk is to look at the factors that influence our choices before and during anaesthesia and to look at how we use monitoring methods to help us make those choices.
Many of the talks given in the past have dealt with understanding how different monitoring methods work. During this talk that understanding is assumed and the focus will be on using the information from the monitors, both manual and electronic to support our decisions.

Before looking at details of anaesthetic monitoring some distinction must be made between the species that we deal with. Basically there are 3; Mammals, birds and reptiles.
Mammalian physiological behaviour follows what you are taught in college and what most people are familiar with. Avian behaviour is a little like mammalian in that they are warm-blooded, respiration is driven by CO_2, stretch reflexes and to some extent oxygen. Reptiles are very different because they are cold-blooded, respiration is much more dominated by oxygen than CO_2 and they have a unique ability to divert blood away from the lungs. This can make them unpredictable in terms of their response to inhalation anaesthetics while conscious.

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Will it wake up? Will it die?
How do you know? You will only be able to answer this question if you know what normal physiological values are. This means being prepared before the anaesthetic starts so that you can react to the information being presented to you.

What do you need to keep an eye on during anaesthesia to keep your patient alive?
This is a difficult question, but let's look at the problem from another perspective..
How would you plan to kill a patient under anaesthesia? Imagine you are a human anaesthetist and your arch enemy appears on the operating table in front of you. What could you do to send them to the pearly gates?

Now some of the answers become more obvious:

1. Inhibit their breathing
2. Excessive use of anaesthetic
3. Deprive them of oxygen
4. Induce blood loss
5. Interfere with heart function
6. Induce excessive cooling or excessive heating

And the reasons, in order:

1. Suffocation - respiratory or mechanical. Absence of oxygen. Increase in CO2
2. Respiratory depression - gaseous, injectable
3. Hypoxaemia/Hypoxia
4. Hypovolaemia
5. Excessive pre-load, excessive afterload, physical restraint
6. Hypothermic shock/Hyperthermia

Inhibit their breathing.
The easiest way to kill your enemy is to suffocate them, particularly if they are anesthetised. Inhibition of breathing leads to failure to deliver Oxygen and failure to remove CO2. If maintained, death is fairly rapid.
This can be done in a couple of ways - restrict the movements of the chest or block the major airway.

Restriction of the chest movement is much more important in animals without a diaphragm because this is the only way for them to breathe. So squeezing the chest of a bird or reptile will kill them.

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Block the major airway

Simply put your thumb over the end of the ET tube and with a cuffed tube that is all you need. Death will be rapid.
But it can be blocked just as easily at the other end too by a mucous plug or even over-distension of the ET tube cuff. Death will be equally rapid but this time you may be totally unaware until it is too late.

So, suffocation stops inflow of oxygen and outflow of CO2.
But what kills them? The high CO2 or the low oxygen? Or something else?
We will come back to this...

Excessive use of anaesthetic

A good way to kill them is to give them too much anaesthetic so that they stop breathing. Chemical suffocation if you like. Once breathing stops it's only a matter of time before they actually die.
Why is this? It might seem obvious but what is it that actually kills them? This is the second time we have said that breathing stops they die. But what actually happens? What is the process that occurs that eventually results in death? I think it is interesting and important to look at what happens when a patient dies from lack of breathing:

With no breathing there is no gas exchange so now it has little to do with the anaesthetic concentration. Changing the vaporiser concentration if they are not breathing will have no effect. There is no new oxygen delivered to the cells and there is no elimination of carbon dioxide. Cells will respire anaerobically as long as they can but in so doing they are producing carbon dioxide and lactate which causes an acidosis. This acidosis if prolonged will lead to cell death and necrosis.
The reservoir of oxygen in the lung will be completely used up by the time the blood has been round the body a few times so after about a minute aerobic cellular respiration will stop and no more oxygen reaches the tissues. The lungs now have no useful function. With the loss of the lungs the two remaining vital organs are the heart and the brain. In humans it is given that the brain can last without oxygen for around 5 minutes. This is so but cell death starts as early as within 1 minute. The heart is a muscle and needs a constant source of energy to continue working. With no delivered oxygen lactate will begin to build up and acidosis will affect muscle contraction. This is the same process as seen in striated muscle and cramp is very severe and very quickly the result of lactate build-up.
Because heart contractility is independent of brain control, the heart will continue to beat even if the brain activity ceases. But eventually the heart runs out of a source of energy and stops. Cardiac arrest. Ultimately everything dies of cardiac arrest.
Once the heart stops it's game over for everything. No surprise therefore that the priority is to keep the heart going. But the preceding shows the chain of events that lead to cardiac arrest. And it needn't be that brutal - there are

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degrees of respiratory depression. Full respiratory arrest will lead to rapid death but marked respiratory depression will also lead to death, it will just take longer. If the oxygen supply to the body is less than oxygen consumption then the inevitable result is eventual cardiac arrest.

So, respiratory depression was the initial factor that led to respiratory arrest and subsequently death. Respiratory depression doesn't have to come from the gaseous anaesthetic, it can be caused by injectable anaesthetics and some other drugs, such as pain-killers (opioids). Be aware of what someone is injecting into your patient, if all drug administration is not by you. Common painkillers such as Temgesic are respiratory depressant.

Oxygen deprivation

If you want to intentionally kill your arch nemesis then just turn the oxygen flow off. But you might get caught so be more subtle, increase the dead space. Now the patient doesn't get as much or enough oxygen so they will inevitably die. Or use a small ET tube that allows breathing around the tube. That way they'll dilute their own supply down to a minimum of 21%. If they are old and have lung disease or other concomitant disease that will probably be enough - oxygen demand will exceed supply and hypoxia will lead to death.

An excessive dead space is effective suffocation - so, be VERY careful of dead space. Understand what it means and take all steps to reduce it as much as possible.
What is Dead Space? It is the volume occupied by gas common to inspiration **AND** expiration. Workshop session on Sunday will highlight this. If the dead space approaches tidal volume (only 1ml for a budgie) then they will be suffocated.

So, it doesn't matter if the lungs are working if they are unable to deliver sufficient oxygen to the body. What results is hypoxaemia and or tissue hypoxia. Normally one follows the other so if they are hypoxaemic they will be hypoxic too. Deprivation of oxygen will obviously kill your patient but it doesn't have to be intentional. If they are spontaneously breathing but their respiratory rate is too low and they are breathing room air then they are oxygen deprived. - Important during recovery where we want them to breathe and we want CO2 to build up so we wait. Every minute you should give them a lung full of oxygen. This period in recovery is where you really need a pulse-ox because these situations can lead to gradual hypoxia.

If oxygen delivery is less than oxygen consumption they will die !

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In the three situations above a pulse-oximeter will have been your early warning signal of ***hypoxaemia***. Don't trust them? Ok, look at the blood (there's nearly always some around). Is it bright red? If it is the pulse-ox is probably wrong. Re-position the probe. Try it on yourself to confirm it is working. Keep stray light out as much as possible especially if the patient is small or the signal is weak. Look at the mmbs - true you can't see cyanosis until about 80% but if the Pulse-Ox says 78% and they are bright pink then I would question the pulse-ox reading.
A capnograph would have also been useful in all cases except for the respiratory arrest and the reduced FiO2

Induce blood loss
Cutting your enemies throat or a major vessel will kill your patient. But how? Blood loss will lead to failure to carry oxygen, a fall in blood pressure, a failure to perfuse the major organs including the brain and therefore death.
Repeatedly drawing blood from your patient may be sufficient to induce a hypoxaemia, particularly if you simultaneously expand plasma volume with fluids. At some point your patient will die if you reduce the oxygen carrying capacity enough even if you maintain circulating volume.
Small patients have a surprisingly small amount of blood, so taking blood for sampling can be detrimental (body blood content is approximately 75ml/kg, so a 50g bird only has about 3.75mls) as can surgical site haemorrhage.
If you have enough oxygen but not enough blood then the result is the same - tissue hypoxia followed by all the events leading up to cardiac arrest.
Blood loss doesn't have to be actual haemorrhage of volume it can be functional in that there either just aren't enough blood cells to go round (anaemia). Either will lead to tissue hypoxia. A shocked haemorrhaging patient may be haemo-concentrated and surviving well. Pumping in fluids can lead to Hb dilution and effective anaemia. In this instance the pulse-ox will not warn you because all the blood is 100% saturated, there just may not be enough of it. But the pulse-ox signal will be very weak (the bargraph movement will be small) and the patient will have pale mucous membranes.

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Interfere with heart function

A subtle way to kill you enemy would be to inject a volume of air equal to at least about one tidal volume into the chest cavity thus creating a pneumothorax. A pneumothorax is a very special case and these patients may do better under spontaneous breathing than IPPV - as will be explained. If ventilated the added air would squeeze the heart on every breath and hinder venous return, both of which would reduce cardiac filling and thereby cardiac output.

Interference with heart function really means reducing the cardiac output as this is key. And during surgery/anaesthesia this is most likely to be as the result of a physical effect on stroke volume. Heart rate is likely to be influenced more by chemical effects such as alpha-2 agonists like medetomidine, which cause a fall in heart rate (bradycardia and biphasic BP response - initial BP increase (Periph VC) followed by prolonged BP reduction) medetomidine also causes hypothermia, av-block, resp depression

Reduce cardiac filling and thereby reduce cardiac output

If it can't draw enough in then it definitely can't pump it out. Reducing venous return does exactly that - it reduces the amount that the heart can draw in. So how can you influence venous return? In mammals (functional diaphragm) you can ventilate the patient and hold the inspiratory pressure too long. This puts pressure in the thoracic cavity but not the abdominal cavity so blood return is hindered. If you do this too much then cardiac output will fall to a point where oxygen delivery is not sufficient to support life - cardiac arrest. This isn't an issue in birds and reptiles because they don't have a diaphragm so you can't pressurise just one compartment. But one thing you do have to be careful with is insufflation during endoscopy. The pressure in the coelomic cavity will tend to reduce the size of all the vessels in that cavity (arteries and veins) and increase the back pressure against which the pump will have to work. This will reduce Cardiac Output. The insufflation pressure will need to be quite high to have an effect but if you're not measuring it or it goes high un-noticed then this could be a real problem.

Prolonged inspiratory time in mammals will reduce venous return as outlined above, so we all try to keep our I:E ratio at better than 1:2 so there is twice as long for blood to flow un-hindered compared to blood flow against increased pressure. But there is one other relatively common situation where ***normal*** (i.e. > 1:2 I:E ratio) ventilation will kill your patient very quickly. This happens when there is air in the space between the lungs and the chest wall, i.e. in the pleural space - a pneumothorax. During inspiration when air is pushed into the lungs by the ventilator the lungs expand and the pressure rises. The air in the chest is compressed and matches the pressure in the lungs. Because this air is round the heart this effectively squeezes the heart at the same pressure reducing its ability to function. You will only see this with a pneumo-thorax

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(either traumatic or iatrogenic) but you MUST be aware of the consequences of IPPV in these situations.

Hypothermia

The effects of hypothermia (effects start at 2.5 degrees below normal body temperature) are many. There is brain dysfunction, a reduction in heart rate (after initial increase), a reduction in respiratory minute volume and diuresis. At the extreme there is a marked decline in cerebral blood flow leading to coma and loss of brain activity. There is a fall in blood pressure and cardiac output, an increased likelihood of dysrhythmias and eventual asystole.
These effects are well recorded in mammals and warm-blooded animals. The effects are not so clear in reptiles/cold-blooded animals where normal body temperature is at ambient. However even in these species, excessive cooling by additional means such as evaporative cooling from surgical site preparation will reduce metabolic rate and effect the behaviour and performance of drugs.

Hyperthermia

The effects of hyperthermia are even more extreme. Dogs left in hot cars that reach body temperatures of 108 degrees Fahrenheit (42.5 Centigrade) rarely survive. The pathology behind this is more difficult to determine but it involves multi-organ failure and brain and cardiac effects.
Hyperthermia is becoming increasingly recognised since the introduction of more efficient heating methods for animals during anaesthesia. Core temperature (oesophageal) is the preferred measurement although peripheral temperature (rectal) will be a good guide but may lag some minutes behind changes in core temperature.
Smaller animals are more prone to both hypothermia and hyperthermia because their smaller body mass means that less energy has to be gained or lost to change their body temperature.

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Always aim to keep reptiles at or near their preferred body temperature PBT:

Species	PBT (^{0}C)	Scientific Name
Childrens Python	30 - 33	Anatresia childreni
Coastal Carpet Python	30 - 32	Morelia spilota variegata
Diamond Python	29 - 30	Morelia spilota spilota
Bearded Dragon	35 - 39	Pogona sp.
Eastern Snake-Necked Turtle (long neck)	26	Chelodina longicollis
Murray River Turtle (short neck)	26	Emydura macquarii
Blue Tongue Lizard	28 - 32	Tiliqua Scincoides

So, we have looked at several ways in which we could despatch our enemy to an early grave. Knowing how this could be done is a major step in understanding how to keep your patient alive. Now we can begin to look at the steps needed and the tools required to accomplish this.

From the previous discussion we can see that to maximise our chances of keeping our patient alive we need to :

- Facilitate their breathing
- Minimise respiratory depression
- Ensure a good supply of oxygen
- Minimise functional blood loss
- Minimise interference with heart function
- Maintain normothermia

How can you achieve the above and what tools can you use to help you?

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Facilitate their breathing
Keep anaesthetic levels under constant supervision
Physically & Functionally

Physically
Don't put anaesthetised spontaneously breathing birds on their sternums, don't put reptiles and birds in tight lateral supports - no diaphragm so can't create any negative pressure if chest motion is restricted.

Functionally: Minimise dead space. Why? Dead space is so important. How large can it be? Aim for less than 10% of tidal volume, preferably 5%. This is easy with the larger animals. But what are the volumes of your connectors?:

Item	Dead Space Volume (max)
ET Connector	0.5 - 3.3 mls (non LDS size 5-13)
ET Connector (LDS)	0.15 - 0.25mls (size 2.5 - 3.5)
15mm Y-connector	6.0 mls
15mm-15mm connector with oxygen feed	8.0mls
25mm of No.3 ET Tube	0.1mls
Luer hub (female)	0.1mls

From the above:
Don't use a 15-15mm connector for CO2 sampling on anything less than 5-6kg.
Even with the smallest ET LDS connector the smallest animal should be no less than 125g
Below 100g use catheters and e.g. LDS kit

So, how do you know if you have minimised the dead space?
Look at **inspired** CO2 levels? Maybe, but it can be misleading because it will depend on depth of respiration. Show video here of rebreathing with 1.5m x 22mm tube, 40cm 22mm tube and no tube

What equipment do we need to be able to monitor this?

Eyes - assessment of tidal volume
Stethoscope - confirmation of breathing over all of the chest
Capnograph - efficiency of breathing, degree of FiCO2 and therefore dead space, duration of expiration curve (elongated with rebreathing)

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Minimise respiratory depression

Anaesthetics reduce respiratory drive so if the animal is spontaneously breathing you will see a fall in rate and depth of respiration. This reduces respiratory minute volume so you will see a rise in end-tidal CO2. However many will be ventilated, so how do you determine respiratory depression?

Strictly you can't, but respiratory depression is only one of the effects of the anaesthetic agent. Other effects, that occur in parallel are :
Decreased level of consciousness (affect on reflexes)
Decreased level of muscle tone (affect on limb stiffness, chest compliance)
Fall in blood pressure (esp isoflurane)
Increased incidence of arrythmias

Use the following as a guide to depth of anaesthesia:

- Muscle movement - spontaneous or in response to deep pain stimulation
- Jaw tone
- Eye position (rotated = normal depth), degree of lachrymation (dry = deep)
- Palpebral/corneal reflex (loss of corneal
- Amount of chest movement during ventilation
- Heart rate (usually decreased if too deep)
- Response to surgical stimuli

If the animal is ventilated then why worry about overdoing it with the isofurane? As long as it's asleep why does it matter if the animal is too deep. The ventilator will keep it alive.
Whilst this may be true, the other effects of excessive agent are undesirable such as effects on blood pressure (hypotension), the tendency to induce arrythmias and CNS depression.

So, we'll aim for a good depth of anaesthesia where there is little or no jaw/limb tone, some downward rotation of the eye (which is still moist), some slight response to deep pain stimuli and a normal steady heart rate, close to resting normal.

What equipment do we need to be able to monitor this?

Eyes - look at eye position, chest movements, other movements
Fingers - jaw one, muscle tone, pinch reflex
Stethoscope - how many people auscultate during IPPV? **DO IT!** Then you will know immediately when the ET tube gets blocked.
ECG Monitor - must be able to cope with rates as low as 6 and as high as 400. Look for a steady rate.
Capnograph - If not ventilated then get info on Minute Volume & respiratory drive. If ventilated then get info on adequacy of IPPV.

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Ensure good oxygen delivery
Deliver 100% oxygen (how do you know?) - FiO2, Pulse-oximetry. Look at the waveform and the bargraph. Look at the colour of the blood. Reduce stray light on the probe. Find the biggest artery you can (even the heart)
Ensure a tight-fitting tube
Ensure that the chest movements are not compromised
Cannot have good oxygen delivery if minute volume is too low. If minute volume is too low et co2 will be high. If et co2 is high, ventilate and/or lighten anaesthetic. If oxygen is low and CO2 is OK can improve PaO2 by increased ventilation depth, i.e. increased TV
Cannot have good oxygen delivery if cardiac output is too low. Cardiac output is a function of heart rate and stroke volume. Determination of cardiac output is not easy, even in medical hospitals, so measurement of your bearded dragon's cardiac output is going to be very difficult. But you can't have a good Stroke Volume if the pulse is weak, since the pulse will reflect the SV. So feel for a good pulse or use a Doppler for a subjective assessment of peripheral flow.

What equipment do we need to be able to monitor this?

Eyes - colour of blood, colour of mucous membranes.
Fingers - pulse strength
Stethoscope - strength, rate and rhythm
Pulse-Oximeter for oxygen saturation (but there has to be enough blood)
Capnopgraph to check minute volume
Doppler - subjective assessment of pulse pressure and thereby SV (and thereby CO)
Oxygen analyser e.g. ICON - FiO2

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Minimise blood loss

Reduce frank haemorrhage.
Be careful with blood sampling. Know the approximate circulating blood volume of your patient - 75ml/kg
Be careful with fluid administration to anaemic or haemorrhaging patients as this may simply dilute the blood. Deliver sufficient volume to maintain perfusion pressure.
The aim is to maintain sufficient circulating blood volume at sufficient pressure to perfuse all the major organs. Use of some sort of blood pressure monitor would be helpful where possible and where physical confirmation and size permits.

What equipment do we need to be able to monitor this?

Fingers - feel the peripheral pulse.
ECG - heart rate will increase in the presence of reduced blood volume as the heart attempts to maintain cardiac output and blood pressure
Doppler - subjective assessment of SV and thereby CO. A good pulse will not be maintained with low circulating blood volume and HR will be high.

Minimise interference on heart function

When ventilating keep I:E ratio less than 1:2 especially in mammals.
Be aware of the effects of a pneumothorax either of traumatic or iatrogenic origin.
Be aware of the effects of body cavity insufflation for endoscopy.
Be aware of the effects of alpha-2 agonists on heart rate, cardiac conductivity and peripheral blood pressure.

What equipment do we need to be able to monitor this?

Know the I:E ratio if you are ventilating.
Minimise PIP during IPPV.
ECG - keep an eye on the heart rate. A continually rising heart rate to 2-3 times its normal value is indicative of a struggling heart. Similarly a falling heart rate to bradycardic levels is also a bad indicator. Also look at the character of the ECG for changes in appearance indicative of problems

Doppler - subjective assessment of SV and thereby CO. A good pulse will not be maintained with low circulating blood volume and HR will be high.

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Maintain normothermia

Studies have shown that maintaining normothermia is much easier than trying to restore normothermia to a hypothermic patient. Pre-warming has been shown to be very beneficial in humans and rats and prevents the hypothermic tendency seen with non pre-warmed animals. The essence of the information is this: keep animals for surgery warm by active methods **before** induction. So hold the small animals in warmed towels, or use a pre-heated anaesthetic chamber (heat pad underneath). Once anaesthetised keep them warm by heating to but not above their PBT.
Use gloves filled with warm water to act as local heating on e.g. limbs or main body mass. If possible use core body temperature as the guide to control of heating/cooling procedures.

Beware of hyperthermia. Use fans or water sprays to cool a patient.

What equipment do we need to be able to monitor this?

Surface thermometer for e.g. heat pads and other heating aids to ensure that they don't get too hot
Rectal thermometer for essentially peripheral body temperature
Oesophageal thermometer for essentially core body temperature

Reptiles & Oxygen/CO2

Blood shunted away from the lungs in response to low oxygen levels
Drive for respiration is more oxygen-driven that CO2-driven.

Conscious breath-holding --> low o2 levels --> blood diverted away from the lungs --> no response to volatile agents

But CO2 is still a by-product of cellular respiration and so is still a valid parameter to measure as an indicator of adequate minute volume ventilation

Other signs of well-being
ECG - steady rhythm with little variation. Normal looking complexes.
Pulse palpation - good strong pulse (doesn't indicate BP but still helpful)

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Further notes:

Know what the ventilator sounds like with a blocked et tube - it's so easy to do. Do it before you start the procedure, then you'll definitely know.

SOP :
Test the ventilator for leaks
Test the ventilator for ET tube blocks
Listen to the patient during IPPV using a stethoscope

Be attentive to any sudden changes in ventilator performance - a blocked tube will have the following symptoms:

Patient chest no longer moving
No lung sounds
Rapid inspiratory phase
No capnogram or reported values

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