Recovery from Anaesthesia

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What is anaesthesia ?

General anaesthesia is a reversible dose related intoxication of the central nervous system whereupon a patient is rendered analgesed, immobile and there is a suppression of consciousness. The later usually means the patient is unconscious but the suppression of consciousness maybe described as hypnosis, narcosis, dissociation and/or amnesic depending on the agents anaesthetic used

Balanced anaesthesia is an anaesthetic state produced by a combination of drugs each of which is aimed at achieving one or more goals of anaesthesia, the so called " triad of anaesthesia" (hypnosis, muscle relaxation and analgesia).

To produce the anaesthetic state we often employ several drug types to reduce the side effects of high doses of any single agent which would otherwise be required to produce an anaesthetic state if they were used alone.

Do things go wrong during anaesthesia and recovery?

Yes. We can subdivide problems so:

- Patient mortality
- Patient morbidity (diseased state, disability or poor health due to any cause)
- Patient experience of its anaesthetic visit
- Our own and our staff experience of administering anaesthesia, our possible morbidity, and even mortality

The risk of death:

"The risk of death: the confidential enquiry into perioperative small animal fatalities."
Brodbelt DC, Blissitt KJ, Hammond RA, Neath PJ, Young LE, Pfeiffer DU, Wood JL.
Vet Anaesth Analg. 2008 Sep;35(5):365-73. Epub 2008 May 5.

OBJECTIVE:

To estimate the risks of anaesthetic and sedation-related mortality in companion animals in the UK. (The Confidential Enquiry into Perioperative Small Animal Fatalities, CEPSAF).

STUDY DESIGN:

A prospective cohort study with nested case-control study.

ANIMAL POPULATION:

All small animals anaesthetized and sedated at participating centres between June 2002 and June 2004.

METHODS:

Patient outcomes at 48 hours (alive, dead and killed) were recorded. Anaesthetic and sedation-related death was defined as death where surgical or pre-existing medical causes did not solely cause death. Species-specific risks of anaesthetic-related death and 95% confidence intervals (95% CI) were calculated. Risks were also estimated in the sub-sets of dogs, cats and rabbits that were either healthy or sick (ASA 1-2 and 3-5, respectively).

RESULTS:

One hundred and seventeen veterinary practices participated in the study and 98 036 dogs, 79 178 cats and 8209 rabbits were anaesthetized and sedated. Overall risks of anaesthetic and sedation-related death in dogs were

Species	Dog		Cat		Rabbit	
Overall mortality rate	0.17%	(1 in 601)	0.24%	(1 in 419)	1.39%	(1 in 72)
Healthy animals ASA grade 1 and 2	0.05%	(1 in 1849)	0.11%	(1 in 895)	0.73%	(1 in 137)
Sick animals ASA Grade 3,4 or 5	1.33%	(1 in 75)	1.40%	(1 in 71)	7.37%	(1 in 14)

Postoperative deaths accounted for 47% of deaths in dogs, 61% in cats and 64% in rabbits. Most other small animal species had higher mortality risks.
CONCLUSIONS AND CLINICAL RELEVANCE:
Small animal anaesthesia appears to be increasingly safe compared to the Clark and Hall survey of 1990 (overall mortality rate 1: 679 healthy dogs and cats). Greater patient care in the postoperative period could reduce fatalities.

What are the risks of morbidity and what are the contributing factors?
Incidence of complications in small animal anaesthesia
Gaynor et al JAAHA 1999
University Teaching Hospital over 1 year

	dogs	cats
no. of anaesthetics:	2,556	683
mortality	0.43%	0.43%
anaesth. complication	12%	10.5%
hypotension	7%	8.5%
cardiac dysrhythmias	2.5%	1.8%
hypercapnia	1.3%	<1%
hypoxaemia	0.5%	
transfusions	1.2%	

Risk factors for anaesthetic related death in dogs 2008
"Results of the confidential enquiry into perioperative small animal fatalities regarding risk factors for anesthetic-related death in dogs".
Brodbelt DC, Pfeiffer DU, Young LE, Wood JL.
J Am Vet Med Assoc. 2008 Oct 1;233(7):1096-104.

OBJECTIVE:
To identify major risk factors associated with anesthetic-related death in dogs.
DESIGN: Case-control study.
ANIMALS:
148 dogs that died or were euthanized within 48 hours after undergoing anesthesia or sedation and for which anesthesia could not be reasonably excluded as a contributory factor (cases) and 487 control dogs that did not die within 48 hours after undergoing anesthesia or sedation (controls).
PROCEDURES:

Details of patient characteristics, preoperative evaluation and preparation, procedure, anesthetic and sedative agents used, monitoring, postoperative management, and personnel involved were recorded. Mixed-effects logistic regression modeling was used to identify factors associated with anesthetic-related death.
RESULTS:
An increase in physical status grade, urgency of the procedure, age, or intended duration of the procedure; a decrease in body weight; anesthesia for a major versus a minor procedure; and use of injectable agents for anesthetic induction and halothane for maintenance or use of inhalant anesthetics alone (compared with use of injectable agents for induction and isoflurane for maintenance) were associated with increased odds of anesthetic-related death.
CONCLUSIONS AND CLINICAL RELEVANCE:
The results suggested that specific factors could be associated with increased odds of anesthetic-related death in dogs. Knowledge of these factors should aid the preoperative assessment and perioperative management of dogs undergoing anesthesia and sedation.

Common risk factors identified with veterinary anaesthesia:
Xyalazine
Halothane vs isoflurane
ASA Grade 3,4,5
Species risk dog<cat<horse<rabbit
Increasing age
Extremes of weight
Increasing procedural urgency, complexity and duration
Fluid therapy in cats
Intubation in cats, brachycephlic dogs and rabbits

Response to the issues of anaesthetic deaths and morbidity
Minimise occurrence by:

- Vigilant competent continuous monitoring
- Practice guidelines for anaesthetic incidents, errors, accidents and emergencies
- Prompt recognition of problems and rapid effective response to correct or minimise the consequences of problems

Why monitor?

- To improve safety and survival from anaesthesia, surgery and intensive care
- Anaesthesia greatly alters normal physiology
- Monitor to assess physiological functions and changes induced by anaesthesia, disease or injury

Monitoring :

- Vigilant & well trained staff are essential!
- Monitoring should be continuous
- Keep a record!
- When to monitor: The major factor in most cases of mortality is that the animals are not under direct observation when problems first arise “and the lesson to be learnt is that monitoring is essential from the time the drugs are given until recovery of full consciousness” Clarke & Hall 1990

Good Anaesthesia Practice avoids:

- Hypoxia and hypoxaemia
- Hypercarbia
- Hypotension
- Overdosage

- Hypothermia
- Fear, anxiety & pain
- Hypoglycaemia
- Inattention
- Aspiration
- Injury

Many deaths occur during recovery from anaesthesia

Recovery from Anaesthesia

Human anaesthetists have established minimum standards for monitoring: "Recommendations for monitoring during anaesthesia and recovery" 2007 Association of Anaesthetises of Great Britain and Ireland

Ideal recovery:

- Minimise adverse effects of anaesthesia & surgery
- Maintain normal physiology
- Rapid regain of airway control
- Rapid elimination of anaesthetic drugs
- Prevent the stress response
- Prevent all pain and it deleterious consequences
- Facilitate surgical goals
- Optimise the patients experience

Recovery from Anaesthesia

- Not simply a reversal of induction
- Patient has
    - Altered physiology from anaesthesia
        - Including impaired homeostasis,
        - cognitive and behavioural changes
    - Consequences of the surgery:
        - surgical pain,
        - the stress response has been activated,
        - Surgery associated pathophysiological alterations
    - Drugs present
        - drug effects
        - drug redistribution, metabolism and elimination
        -

Recovery from Anaesthesia: Altered physiology

- Diminished control of airway: partial and complete airway obstruction is common
- Hypoxia
- Hypothermia
- Hypotension
- Hypovolaemia
- Reflux of acidic gastric fluid
- Nausea and vomiting
- Decreased mucociliary airway clearance
- Cognitive and behavioural changes including delayed recovery of consciousness
- Hypoglycaemia
- Stress response is activated

- Cardiac dysrhythmias. Sympathetic nervous system response (pain, hypoxia etc) and stress response both act to increase circulating catecholamines which in turn can cause cardiac dysrhythmias especially in the presence of halogenated anaesthetic agents

During recovery from anaesthesia hypoxia is common. Reasons:

1) Functional Residual Capacity is reduced
2) Ventilation response to carbon dioxide level is dampened
3) Respiratory response to hypoxia is decreased at sub-anaesthetic concentrations of anaesthetic argents (0.1MAC)
4) Hypoxic pulmonary vasoconstriction reflex is depressed by general anaesthesia (this is the major reflex that matches blood flow to the ventilation of each alveoli)
5) Gaseous anaesthetic (mean dose ± SD 0.70 ± 0.33 MAC) significantly depressed carotid body response by 24% ($p = 0.041$). There were no differences between halothane, enflurane, isoflurane
6) CNS depression (residual drugs)
7) Hypoventilation because of reduced strength in the diaphragm and intercostal muscles from residual anaesthetic effects
8) Reduction in compliance as a consequence of reduced FRC and the development of lung tissue atelectasis.
9) CNS depression (residual drugs)
10) Hypotension and decreased cardiac output
11) Hypothermia induced shivering which increases O2 consumption
12) Pain
13) Sympathetic nervous response and stress response both act to increase circulating catecholamines
14) No sigh reflex (collapsed lung)
15) No voluntary changes in body position

Let us look at several of these factors more closely:

Functional Residual Capacity is reduced (or is it?)

Functional Residual Capacity (FRC) is the volume of air present in the lungs at the end of passive expiration

At FRC, the elastic recoil forces of the lungs and chest wall are equal but opposite and there is no exertion by the diaphragm or other respiratory muscles. Because of the weight of the lung during normal conscious respiration the upper areas of the lung remain relatively quite distended and thus stiffer and less compliant whereas the lower areas are more collapsed and compliant. As a result, during inspiration the bulk of the tidal volume enters the lower lung. However with very low lung volumes the lower areas of the lung have become so collapsed they are actually atelectic and can no longer readily open with normal inspiratory effort. So at low lung volumes the distribution of ventilation is reversed, the upper regions ventilate better than the lower regions.

Is Functional Residual Capacity reduced in dogs? Yes.

- Lai et al 1979 investigated the effect of posture and thiopentone anaesthesia on the FRC and Total lung capacity in 7 mongrel dogs (Weighting 13-28kg) with permanent tracheotomies, Anaesthesia produced no significant changes in lung volumes (falls 2%-5.6% not significant), nor did postural change in either the awake of anaesthetised dog
  FRC: prone 42.0±3ml/kg, supine 42.0±2ml/kg, lateral 40.5±3.0ml/kg
- Rozanski et al 2010 investigated effect of body position, chest wrap and sedation on FRC in 6 healthy deep chested dogs (mean weight 31.5±6.9kg)
  Non-sedated FRC: Standing 75.3±23.8ml/kg, Lateral 50.8±13.2 ml/kg

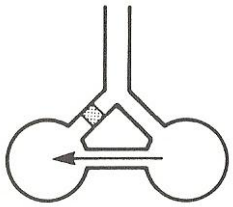
Sedation sternal 52±29.5ml/kg, Sedation + R lat FRC 30.4±10ml/kg

- All other species have ↓ FRC except children with ketamine, young animals have more compliant chests and ketamine is a bronchodilator
- Radiography shows the diaphragm moved cranially in anaesthetised animals

Functional Residual Capacity is reduced and atelectasis ensues

- This leads small airway closure and atelectasis especially of the downside lung
- Absorption atelectasis during anaesthesia if on 100% inspired concentration of O2
- Mucous trapping blocks airways leading to atelectasis
- Low FRC + Post-op shallow breathing to avoid pain leads to atelectasis
- Excessive IV fluids

Note: Atelectasis in dogs and cats is partly decreased by the presence of well developed collateral ventilation



Collateral

Atelectic and poorly ventilated regions of the lung are detrimental as they contribute to hypoxia. These areas are either not ventilated or poorly so, yet still are perfused so the blood flowing through does not undergo gas exchange and this causes venous admixture, shunt and hypoxia.

When ever we anaesthetise an animal, the down side lung tends to collapse. This effect is readily seen when performing a radiographic examination of the chest. If one of the lateral views is taken first and then either the VD or DV view taken next, the cardiac shadow is often well displaced to what was the downside during the recent lateral position. Downside atelectic lung can take minutes to hours or even days to fully reinflate after anaesthesia.

Hypoventilation

What effect does underventilation have on oxygen concentration in the tissues?

- It depends on the partial pressure of oxygen in the alveoli P_AO_2
- At atmospheric pressure, inhaling normal atmospheric air (21% oxygen), underventilation causes hypoxia
- During anaesthesia we know that anaesthetics are respiratory and cardiovascular depressants and we address this problem by providing an oxygen enriched inspired gas mixture (must be > 30%) and IV fluids

Answer: Provide supplemental oxygen

- All anaesthetised patients have depressed ventilation
- Ventilatory depression when breathing air ***will always*** result in hypoxaemia
- Even mild hypoxaemia reduces the margin of patient safety
- A Nasal oxygen catheter flowing 100-200ml O2/kg/min will provide 30%-50% humidified O_2
- A slight increase in the inspired O2 concentration will prevent hypoxia even in the face of moderate to severe hypoventilation

See figure below: The effect of enriching inspired oxygen concentration during hypoventilation in man. From Nunn's Applied Respiratory Physiology 5th Edit Lumb AB

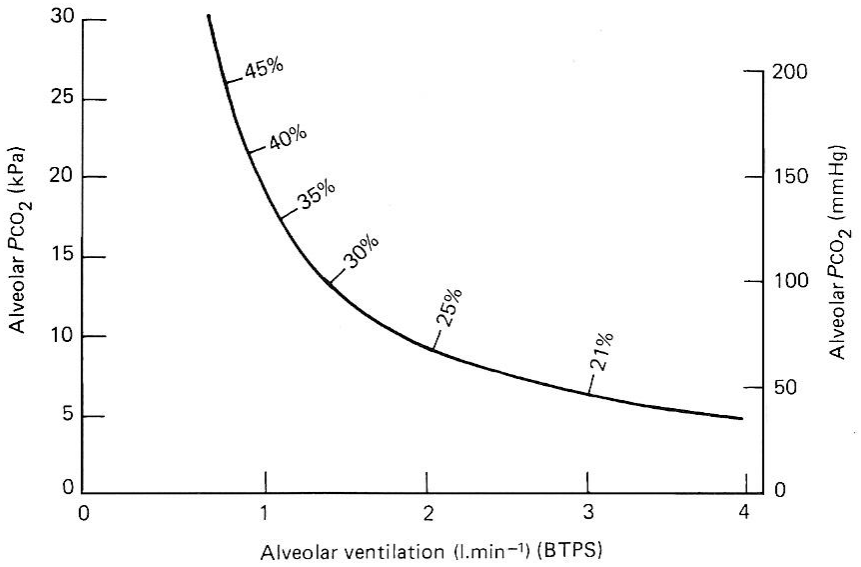


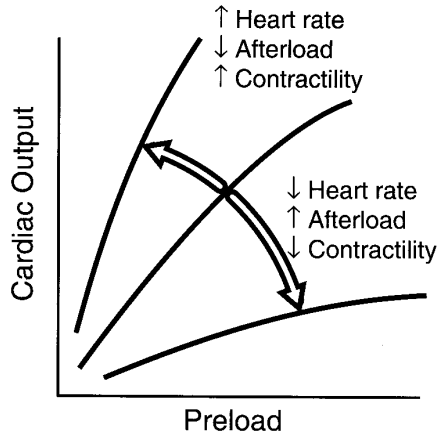
Figure 26.5 Alveolar P_{CO_2} as a function of alveolar ventilation at rest. The percentages indicate the inspired oxygen concentration that is then required to restore normal alveolar P_{O_2}.

Interestingly, because of the progressive increase in the cross sectional area of the airways as air moves into the lungs, the final movement of gas in and out of alveoli is not brought about by the flow of air, rather, by diffusion. Increasing the inspired concentration of Oxygen (FIO2) enhances the diffusion of O2 into the alveoli.

Anaesthetics are cardiovascular depressants

There are several useful models one can use to help understand cardiovascular function:

1) The function of the arterial side of the circulatory system can be understood by a modification of Ohms law: Blood Pressure = Cardiac Output x Peripheral Resistance
2) The function of the venous side of the system by the Frank-Starling Law: Cardiac output is a function of preload



The Frank-Starling law of the heart describes a fundamental principle of cardiac behaviour which states that the force of contraction of the cardiac muscle is proportional to its initial length. The energy set free at each contraction is a simple function of cardiac filling. The more the ventricle is filled with blood during diastole (end-diastolic volume), the greater the volume of ejected blood will be during the resulting systolic contraction (stroke volume).

Methods to minimise the cardiovascular depressant action or anaesthetics

1) Use cardiovascular depressant drugs conservatively, for instance keep the Isoflurance concentration as low as practical. Have a trained staff member monitor every anaesthetic
2) Use adjunctive drugs that decrease the MAC of the inhalant for example opioids, benzodiazepenes, sedatives, ketamine
3) Use adjunctive analgesic/anaesthetic techniques: Epidural, regional nerve blocks, local infiltration. For dogs or cats undergoing hind leg surgery that have received an epidural analgesic, it is common to be able to maintain unconsciousness with an inhaled concentration of isoflurane at 0.5 to 1.0%
4) Provide IV fluids during anaesthesia to improve cardiac output (the Frank-Starling law) and improve tissue perfusion
5) Monitor HR and Non-invasive or direct BP.
   Note: A "PetMAP" is a very useful Non-invasive BP monitor for small animal practice.

Recovery from Anaesthesia: How to extubate

- Inspect the oropharynx. Use a laryngoscope.
- Treat any reflux that may be present
- All necessary treatments done? bandages, nasal oxygen catheter, analgesics, warming, chest drain aspirated, bladder empty??
- Empty the ET tube cuff and undo the ET tube tie
- Check air is still moving
- Move to cage or recovery whilst still asleep (except brachiocephalics)
- Gently slide out the tube so not to stimulate laryngeal and pharyngeal reflexes
- Check for movement of air, not just observation of respiratory efforts!!
- Monitor !

Recovery from Anaesthesia: When do you extubate?

Cats: just before they start to swallow
Dogs: once they start to swallow except brachycephalic breeds

How do you extubate brachycephalics?

- In sternal recumbency
- quiet
- do not stimulate
- leave intubated until they are conscious and moving their head looking around

Recovery from Anaesthesia: Dealing with airway obstruction

- Is the patient moving air?
- History:
  - Does the patient snore when asleep?
  - Does the patient have an URT stridore all the time when conscious?
  - Does the patient develop a stridor with exercise
  - Consider laryngeal paralysis or collapse?
  - Does the patient have an intermittent URT stridor Consider tracheobronchial collapse?
- Stridor = partial airway obstruction.

- No sound = complete airway obstruction
- Length of tongue in a brachiocephalic is the same as if a normal skull length patient. Pull the tongue rostrally to open the pharynx

Avoiding and treating airway obstruction
- Anaesthesia technique that has a fast recovery so patient rapidly regains control of pharyngeal and laryngeal reflexes
- Leave brachycephalic breeds intubated throughout recovery in a quiet environment where the patient is not stimulated (don't check eye reflexes continuously or move the ET tube). Place in sternal recumbency.
- Pull tongue forward (if possible)
- Pharyngeal stent (plastic syringe barrel, case, bandage roll)
- Nasal oxygen
- Re-intubation
- Consider tracheotomy

Reflux
"Gastro-oesophageal reflux during anaesthesia in the dog: the effect of preoperative fasting and premedication". AD Galatos & D. Raptopoulos Vet Record 1995;137:479-483
Lower oesophageal reflux was monitored in 240 anaesthetised dogs.
- The incidence of gastro-oesophageal reflux was 16.3 %
- Most of the reflux episodes occurred shortly after the induction of anaesthesia.
- The refluxate was nearly always acid (pH < 4.0),
- In 10.3 per cent of the cases it was alkaline (pH > 7.5)
- Gastric contents of pH below 2.5 were refluxed on 19 occasions (7.9%)
- Regurgitation occurred in only one dog.
- Prolonging preoperative fasting was associated with an increased incidence of reflux and increased gastric acidity.

Treating Regurgitation:
- Head down tilt
- Check cuff is inflated correctly
- Ideally check pH with urine Dipstix
- Clean pharynx with gauze swabs
- Place oesophageal tube
- Irrigate oesophagus with 0.3M Na Citrate 1-3ml/kg whilst slowly extracting the stomach tube
- Repeat clean pharynx

Anaesthetic recovery of the obese patient: problems
- Undiagnosed disease
    - Diabetes, Hyperadrenocorticism, Hyperthyroidism, liver disease
- Hypoventilation and Hypoxaemia
    - Decreased FRC
    - Decreased chest wall compliance (stiffer)
    - Limited diaphragmatic excursions due to increased abdominal contents
    - Obesity Hypoventilation syndrome (Pickwickian syndrome):
        - ↑ $PaCO_2$ (>45mm Hg) and ↓ PaO_2
        - Hypoxic pulmonary vasoconstriction reflex → pulmonary hypertension → cor pulmonale
        - high PCV

    - Worsened by anaesthesia and during recovery
- Upper airway obstruction from increased mass of pharyngeal tissues and tongue
    - Worsened by sedatives and anaesthetics
- Functional Residual Capacity is reduced

Anaesthetic recovery of the obese patient: recommendations

- Premeds, select those which will enable a prompt recovery
- Once premeds administered, continuously observe for airway obstruction
- Rapid control of airway at induction
- Anaesthetic drugs should be dosed for lean body weight
- IPPV (and PEEP 4-5mm Hg)
- Sigh breaths (30-40cm H_2O)every 20 mins during GA
- Oxygen/air mix (eg F_I 0.5 to 0.8). Nitrogen acts as a splinting gas and slows absorption atelectasis
- During recovery
    - Keep intubated until they no longer will tolerate ET tube
    - Sternal recumbancy
    - Supplementary oxygen
    - Select drugs to enable prompt recovery
- Monitor

Recovery from Anaesthesia: Vocalisation during recovery: pain, emergence delirium, or other?

- What breed is it?
- Pre-emptive appropriate and adequate analgesia and analgesic plan
- Avoid by attending to likely problems prior to recovery eg additional analgesia (opioids IV), bandages, empty bladder, minimise noise, move to recovery whilst still asleep
- Methadone incremental doses 0.1mg/kg IV (then flush), low dose ACP after 2^{nd}-3^{rd} dose (0.01mg/kg)
- Comfort with soothing pats and words. Warmth. Sit with the patient
- Think: Hypoxia? Pain? Hypothermia? Bladder? Hypotension? Abnormal mentation? Ischemic pain (bandages), Anxiety (airway obstruction, fear behaviour), dysphoria from drugs (Zoetil, ketamine in dogs)
- Consider rare problems: acute vestibular disturbance, acute blindness, embolism, burns
- Anxious and fearful dogs: think about behavioural therapy (training, clomipramine, home sedation) prior to routine procedures. Owners present at induction (recovery?).
- If persistent, consider: morphine, morphine+ketamine, morphine+ketamine+lidocaine, morphine+midazolam+Ketamine infusions. Phenobarbitone.

Recovery from Anaesthesia: Treating and preventing hypothermia:

- If you are not looking for it, you are unlikely to notice it
- Must measure temperature, at least pre and immediately post-op
- For every problem missed through lack of knowledge, one hundred are missed through lack of looking!
- Methods for treating and preventing hypothermia:
    - Prevent chilling in the first place
    - Warm woollen blankets
    - Wrap the feet and legs in bubble wrap
    - Hot water bottles (old fluid bags that have a dye added to indicate they are not sterile)
    - Heating pads – can cause burns. Animal needs to have the ability to move off them if they become too hot
    - Radiant heating lamps are very useful during recovery but can cause burns

- Warm air blankets can be used both in surgery and in recovery

Recovery from Anaesthesia: Consequences of the surgery:

- surgical pain,
- the stress response has been activated,
- Surgery associated pathophysiological alterations:
  - Hypovolaemia,
  - Hypothermia (cold table, open body cavities etc)
  - Bleeding,
  - Coaggulopathies,
  - Portal hypertension after porto-systemic shunt surgery,
  - Pneumothorax and atelectasis after thoracotomy
  - Paralytic ilieus (following GI surgery)
  - Sepsis (pyometritis, enterotomies)
  - Systemic inflammatory response (SIRS)
  - Reperfusion injury (eg GDV surgery)
  - Thromboembolism
  - Cardiac rhythm disturbances
  - Hypogylcaemia

Monitoring Recovery

- Airway
- Ventilation
- Oxygenation (sternal recumbency, supplementary oxygen, measure SpO2)
- Check temperature & provide warmth
- Additional analgesia for pain
- Treat vomiting if ongoing (check for hypoxia, drug induced?)
- Treat reflux (sodium citrate 0.3M)
- Measure cardinal signs: HR, RR , temp, MM colour, CRT
- Monitoring Recovery
- IV fluid rate – usually reduced post-op
- Delirium (pain, excitement, old age, hypoxia –treat accordingly)
- Empty bladder
- Critical care: BP, SpO2, ECG, ETCO2 and airway pressure if artificially ventilated, urine output
- Laboratory tests (glucose, PCV, TPP, clotting times, electrolytes, blood gases)
- Additional drugs (infusion rates, compatibilities)
- Offer water once able to walk and stand, food a few hours after

Recovery from Anaesthesia: Summary

- The most important monitor is trained staff: Veterinarian and/or Veterinary nurse.
- Monitor from pre-anaesthetic evaluation to recovery of consciousness & control of normal physiological functions
- Technical monitors to assist: Pulse oximeter, capnograph, oesophageal stethoscope, NIBP, temp, ECG, agent monitoring, direct BP
- Most common underlying reason for death: inattention, human error and hypoxia
- High risk: Alpha 2 agonists, ASA 3,4,5, species (rabbits, horses and cats), urgent, complex or long surgeries, extremes of age
- Provide adequate and safe analgesia
- If any chance or suspicion of hypoxia, provide supplementary oxygen
- IV fluids via fluid pump

- Establish and implement practice guidelines & protocols