cpdsolutions
transferring knowledge through excellence in training

Local Anaesthetic Techniques Mini Series

Session 1: Getting Started with Local Anaesthesia

Matt Gurney BVSc CertVA PgCertVBM DipECVAA MRCVS
RCVS & European Specialist in Veterinary Anaesthesia & Analgesia

Local Anaesthetic Techniques in Small Animal Practice
Matt Gurney BVSc CertVA DipECVAA MRCVS

Introduction

The first reported use of local anaesthetic for surgical purposes was by a Peruvian army surgeon, that drug being cocaine, derived from *Erythroxylon coca*, an Andean shrub. In 1884 cocaine was used by Koeller to anaesthetise the eye by instillation into the conjunctival sac. In the 21st century local anaesthesia remains a key modality in the prevention of pain. Advances in our knowledge of techniques has been facilitated by technology, namely nerve locators and ultrasound guidance.

Benefits to the animal

- Better post operative pain control
- Potential to reduce the development of chronic pain

Benefits to the anaesthetist

- Simple and quick to perform
- Smoother anaesthetic
- Less reliance on other drugs which may have side effects
- Cheap

Current analgesic use

In 2013 Murrell et al surveyed veterinary practitioners regarding their analgesic use. This was compared to the previous survey in 1999.

- 1999 – only 50% prescribed analgesia for routine sx
- 2013 – all 720 have opioid & NSAID available
- 98% give opioids & NSAIDs for sx
- 90.5% give dogs opioids, 81.8% to cats
- 75.1% give dogs NSAIDs, 33.4% to cats

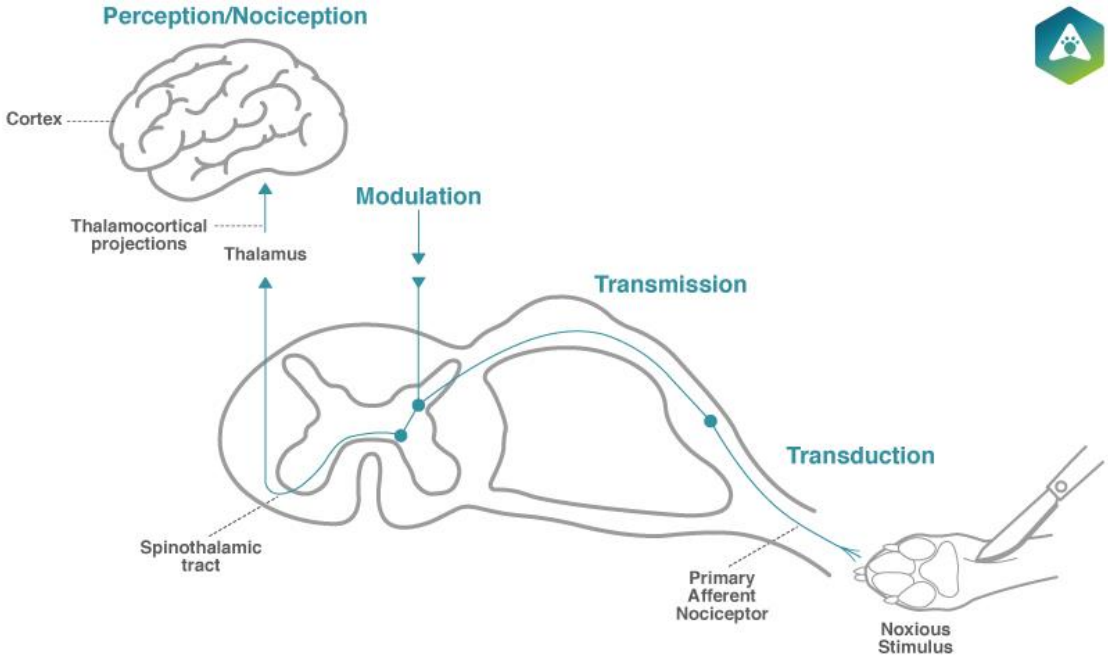
These numbers are still too low and we should consider carefully our use of multimodal analgesia in every case.

Acute pain management

An analgesic is a drug which is used to relieve pain. Analgesic comes from the Greek which means without pain.
The International Association for the Study of Pain (IASP) defines pain as '*an unpleasant sensory or emotional experience associated with actual or potential tissue damage or described in terms of such damage. The inability to communicate verbally does not negate the possibility that the individual cannot feel pain.'* Of course this is particularly relevant to our veterinary patients.

Basic pain physiology

The first stage is **nociception**. The initial insult to the body is detected by nociceptors located in the periphery. The second stage is **transmission** and involves the relay of the painful stimulus via a **peripheral nerve** into the central nervous system. The nerve fibres transmitting pain can be divided into **A fibres** which transmit sharp, mechanical stimuli and **C fibres** which transmit dull or burning pain. These nerve fibres connect to the spinal cord in a region called the **dorsal horn**. From here the impulse can follow several pathways. It is relayed up the spinal cord via the brainstem and thalamus to the cortex where it is perceived as pain. Areas of the brain responsible for pain perception are the **thalamus** and **prefrontal cortex**. The impulse may also form part of a withdrawal reflex or reflex arc at a spinal level.
However this is not just a one-way system. The brain also acts to try to suppress the noxious input, a process known as **descending inhibition**. This is also thought to be a protective process, which for example allows an injured animal to escape a predator. Drugs such as tramadol mediate this system, as do the alpha 2 agonists as well as acupuncture.



Can pain be useful?
Pain can be further divided into **pathological** and **physiological** pain. Physiological pain is the process involved in the detection of the initial input. The pain level perceived is proportional to the noxious stimulus. This type of pain can be seen to play a protective role, which if we relate to an example of a cat with the thorn in its paw, it tells the cat to pull its paw away from the thorn. Chronic pain serves no useful purpose.

What are the detrimental effects of pain?
Pathological pain occurs when the pain perceived has outlasted the initial noxious stimulus. If for example the thorn in the cat's paw causes inflammation, then the pain will persist beyond the removal of the thorn. Likewise in an animal with a fracture where there is tissue damage there will be continued inflammation.
Pain causes changes in the central nervous system by a process known as **plasticity**. The initial painful stimulus causes changes in the brain and spinal cord which result in a heightened perception of pain after the initial period of pain. This is one of the main reasons for using analgesics when pain is anticipated such as prior to surgery and underpins the concept of pre-emptive analgesia. The key receptor responsible here is the **NMDA receptor**, hence this is a target for analgesics.

Multimodal Analgesia
The mainstays of multimodal analgesia (MMA) are the opioids, NSAIDS and local anaesthetics. MMA targets the processes of transduction, transmission, modulation & perception at multiple levels of the neuraxis thereby optimizing not only the provision of analgesia in the acute phase but also minimising the likelihood that chronic pain will develop. With the focus moving towards a preventive approach to analgesia our attention must be directed to assessment and treatment of pain both before surgery and during the rehabilitation stage.

Pre-emptive Analgesia

Pre-emptive analgesia has been defined as '*an antinociceptive treatment that prevents establishment of altered central processing of afferent input from injuries'* (Kelly and others 2001a), which practically put means that applying an analgesic technique before the incision results in better pain control after the operation than applying the same technique after the incision. In practical terms, simply administering an analgesic prior to surgery does not tick the pre-emptive analgesia box without consideration given to pharmacology of the drug. The establishment of an effective level of analgesia is paramount and an inadequate antinociceptive pre-operative intervention should not be regarded as pre-emptive analgesia (Kelly and others 2001a). For example, buprenorphine is slow to associate with its receptor and therefore has a prolonged onset time of 30-45 minutes (Slingsby & Waterman Pearson 1998, Flecknell & Roughan, 2002, Murrell 2007), so clearly a surgical stimulus during this period may fail to achieve the goal of pre-emptive analgesia. Similarly, the time to achieve maximal plasma concentration following subcutaneous administration of commonly used NSAIDs varies from 0.5-2.5 hours (Busch and others 1998, Jung 2009), representing a potentially significant analgesic gap.

The concept of pre-emptive analgesia is much debated amongst anaesthetists and pain management specialists however there is agreement that neuraxial (epidural or spinal) opioids and systemic or epidural NMDA antagonists (such as ketamine) appear to show the most promising pre-emptive effect (Kelly, Ahmad, & Brull, 2001) whilst for other analgesic interventions the evidence is limited. Work in dogs has shown a pre-emptive effect of both pethidine (Lascelles and others 1997) and carprofen(Lascelles, Cripps, Jones, & Waterman Pearson, 1998), which is supported by recent work examining buprenorphine in dogs (Slingsby, Taylor, & Murrell, 2011). None of this debate should deter us from implementing pre-emptive analgesia into our daily practice.

Effective pre-emptive analgesic techniques require multimodal interception of nociceptive input, increasing the threshold for nociception, and blocking or decreasing nociceptor activation (Kelly et al., 2001). Pain is best controlled using several analgesic agents, each of which acts on a specific site along the pain pathway, which lessens the reliance on one particular agent or mechanism, and the resulting synergism, whilst augmenting analgesia, may avoid side effects associated with high doses of individual agents. Synergism has been documented in dogs and cats between alpha-2-agonists and opioids (Grimm, Tranquilli, Thurmon, & Benson, 2000; Slingsby, Murrell, & Taylor, 2010) and NSAIDs and opioids (Shih, Robertson, Isaza, Pablo, & Davies, 2008; Slingsby & Waterman Pearson, 2001; Steagall et al., 2009). An essential aspect in practicing preventive analgesia is that the analgesic intervention should be continued for an long as the sensitizing pain stimulus lasts (Dahl, 2004), highlighting the need to accurately assess pain. There is a key role here for loco regional techniques.

Pre-emptive or Preventive analgesia?

The concept of pre-emptive analgesia refers to the timing of analgesic administration – either before or after the incision. This classic pre versus post approach assumes that intra-operative factors contribute most to generation of a sensitized state. This concept has been broadened in light of increasing knowledge to consider the influence of multiple factors on the generation of central sensitization, with the aim of attenuating the impact of noxious pre, intra and post-operative stimuli and is termed preventive analgesia. Of the three broad peri-operative periods as yet it is unclear the extent to which each period contributes to central sensitization and post-operative pain although there are studies which document beneficial effects of post-operative versus intra-operative nociceptive blockade (Gordon and others 2002). Other studies have demonstrated more effective relief of post-operative pain by targeting pre-operative pain (Klasen and others 2005). With the current state of knowledge we should aim to incorporate excellent pain management at all stages of the peri-operative period based on the 'Anticipate, Assess, Alleviate' approach.

Preventive analgesia is demonstrated when post-operative pain and/or analgesic use are reduced beyond the duration of action of the target drug, defined as 5.5 half-lives of the target drug (Katz, Clarke, & Seltzer, 2011) and the aims of preventive analgesia are to minimize sensitisation induced by noxious perioperative stimuli including those arising pre-operatively, intra-operatively, and postoperatively. With this revised concept the challenge will now be to document preventive analgesia in our veterinary patients.

Pain Assessment
When considering whether an animal is in pain there is a multitude of information to take into account when deciding the best management strategy for that patient. Unless you look for something, you will never find it, so a simple starting point is that all patients should be evaluated for signs of pain after surgery at appropriate intervals.
There are several pain scales to assess pain in dogs, but often your overall impression and clinical experience tells you the most. If you consider an animal to be in pain, then that animal should receive analgesia. The patient can then be reassessed at a time frame appropriate to the drug used. It is best to analgese a patient than leave them in pain. If the patient improves following analgesia, then the pain hypothesis holds up. On the converse side, if animals are repeatedly medicated with drugs without being in pain, then they may suffer detrimental effects from the drugs.
A **visual analogue scale** is a measure used widely in humans. The patient is asked to put a mark on the line where they consider their pain to be. The line is a scale from 0-100. Zero indicates no pain at all and 100 represents the worst pain imaginable. The distance from the no pain end to the patient mark is their pain score. This has been validated in cats for intervention and the authors suggest analgesia should be administered at a pain scale of 30/100.
Similar to this is a **numerical rating scale** which is numbered from 0-10 and the patient marks the number which correlates to their level of pain.
Both scales are very easy to use and can be applied to everyday practice with little extra work. Ideally the same person should score the pain each time to give the best representation of how the patient changes over time. Validation of this scale suggests we administer analgesia at 4/10.
A **simple descriptive scale** will have several expressions used to describe pain and the patient has to select the description best fitting their pain. These scales have been used in veterinary studies evaluating analgesia.

There are several pain scales designed for dogs, which are **composite descriptive scales**. They have several categories, each with a list of descriptors. For example, first you look at the dog from outside the kennel and assess its behaviour. You would then put on a lead, if appropriate, and lead the dog from the kennel. Next you apply pressure around the wound to assess the dog's reaction. Finally you make an overall assessment of your impression of the dog's pain levels. Each descriptor is assigned a score, so the total of all descriptors is the dog's pain score. The most widely used in the Short Form of the Glasgow Composite Pain Scale (search to download).
For cats there are three options. The Colorado State Pain Scale (not validated), the Botacatu pain scale (Brondani et al. 2011) and recently a Glasgow pain scale for cats has been reported (Calvo et al 2014) and refined (Reid et al 2017).

So what signs should you look for?
One of the most consistent signs of pain in animals is abnormal behaviour for that animal. This though may be difficult to truly assess if you have never seen the animal before. Physiological signs such as changes in heart rate, respiratory rate and cortisol levels have been shown to be inconsistent guides to whether pain is present as they can be influenced by many factors other than pain (although many studies still use these parameters).
Dogs will often react when the painful area is touched – this may be displayed as aggression, flinching or as a change in facial expression. An adapted posture may be a reaction to pain and this might be seen as altered behaviour by the owner. Biting or chewing at an area is an obvious sign and should be considered especially post operatively with dressings. Is the injury painful or is the dressing too tight?
A cat in obvious pain can be easy to spot, but sometimes hard to differentiate from the cat who hates coming to the vet. Cats will often not vocalise if in pain. Watch the cat when placed in a kennel. A comfortable cat will sit in sternal recumbency with its paws tucked in. Extremely painful cats will lie flat out. An elevated respiratory rate can be a sign of pain in cats.

How can signs be misinterpreted?
Removing an animal from its normal environment will affect their display of pain behaviour. In the clinic a cat which just curled up at home may become very scared or inquisitive or wary. The dog who hates coming to the vets may exhibit flight behaviour.

An animal's response to pain depends upon their temperament. Some breeds of dogs are very stoic whereas others will show a marked response to a seemingly minor stimulus, for example a greyhound's reaction to a subcutaneous injection.
It is much easier to separate dogs and cats into two groups because the two species react very differently to pain; consider how many cats will hide away after a road accident compared to the dog which screams in pain.
Experience of various procedures or injuries will give you an idea how painful the animal might be before you see the patient. This should be borne in mind when assessing pain, especially if you have never seen the animal before. In cases of chronic pain the history given by the owner is very important.
Signs of extreme pain in dogs can be very obvious in some patients and include vocalisation although some dogs may become very quiet. The degree of pain experienced between two dogs or two cats may be the same, but their displays of pain can be very different.

Analgesic Drugs
The analgesic drugs we use all act at different levels of the pain pathway.
The most common drugs used every day in practice are the **non-steroidal anti-inflammatory drugs, the opioids and the local anaesthetics.** These drugs comprise the temple of analgesia. These three classes of drugs form the pillars of the temple and should be considered wherever possible in patients in pain.

Opioids
Opioids are the mainstay of analgesia in veterinary practice and should be considered as first line for all animals in pain. Traditionally all opioids are compared to the gold standard which is morphine. Opioids act in the central nervous system at spinal levels and higher centres where they modulate the signal being transmitted to the brain and alter perception. This does not mean that the signal is totally blocked. The painful signal still reaches the brain but it is not perceived as pain because of the opioid altering the motivational affective component of the pain.
At doses used clinically there is no reason to avoid opioids for fear of side effects.
Opioids act on opioid receptors in the spinal cord and brain. These receptors are known as mu, kappa and delta. The most effective analgesics act as agonists as mu receptors.

Opioid Doses for Analgesia& Common Side Effects in Dogs & Cats

Opioid	Bradycardia	Analgesia	Resp Depr	Use
Methadone	**++ expect HR 60-80**	**+++ μ agonist& NMDA antagonist**	**+panting**	**Excellent analgesia 0.1-0.3mg/kg q4-6hrs CRI 0.1mg/kg/hr**
Morphine	**+ low doses may be sympatho-mimetic**	**+++ μ agonist**	**+**	**Excellent analgesia 0.1-0.3mg/kg q4-6hrs CRI 0.1mg/kg/hr Emesis**
Buprenorphine	**+/-**	**++ partial μ agonist**	**+/-**	**Good analgesia 0.02mg/kg q5-6hrs Oral transmucosal use in cats**
Butorphanol	**+/-**	**+ Kappa agonist μ antagonist**	**+/-**	**Excellent sedation w ACP or alpha 2's 0.1-0.4mg/kg**
Fentanyl	**+++**	**+++ μ agonist**	**+++ (dose dependent)**	**Excellent analgesia Intra-op CRI 5-20μg/kg/hr Post-op CRI 2-5μg/kg/hr Transdermal patch 2-5μg/kg/hr Interventional analgesia 1-2μg/kg IV**
Pethidine	**Low doses vagolytic**	**+**	**-**	**For mild sedation. Excellent analgesia. 2-5mg/kg IM q90mins**

Drug	Licensed dose	Commonly used doses	Licensed route
Methadone	Dog 0.5-1.0mg/kg Cat 0.3-0.6mg/kg	0.1-0.5 mg/kg	IV, IM, SC
Buprenorphine	Dog 0.01-0.02mg/kg Cat 0.01-0.02mg/kg	0.01-0.02mg kg	IM, IV
Fentanyl	Dog 5-10mcg/kg IV bolus 6-10mcg/kg/hr CRI	2-20mcg/kg/hr	IV
Butorphanol	Dog 0.2-0.3mg/kg Cat 0.4mg/kg	0.1-0.4mg kg	IM, IV, SC
Pethidine	Dog 3.3mg/kg Cat 3.3mg/kg	2-4mg/kg	IM – never IV

The table above is taken from the presentation slides and details the licensed doses of opioids along with commonly used clinical doses.

Methadone has a similar profile to morphine although does not cause vomiting or histamine release. In addition, it acts at NMDA receptors and therefore has a role in preventing chronic pain. Licensed in dogs and cats. My first choice opioid in dogs and cats. **Note the dose used here versus the higher SPC dose.** Whilst some sources suggest not using methadone as a CRI due to its long duration of action, such use was recently evaluated at an RSPCA clinic in dogs and cats. Rate used was 0.1mg/kg/hr. Side effects were minimal in dogs and mostly dysphoria in cats.
Morphine is a full mu receptor agonist and produces excellent analgesia. It can make animals feel nauseous and vomit. If given IV it should be given slowly as it may cause histamine release. Duration of analgesia is around 4 hours. Not licensed in dogs and cats and therefore difficult to justify its use anymore.
Buprenorphine is the most common opioid used in small animal practice in the UK. It acts on mu receptors but unlike morphine and methadone it is a partial agonist. This means it binds well to the receptor but cannot produce analgesia as good as that provided by methadone (Hunt et al, 2013). It is licensed for use in both dogs and cats. Studies have shown that the sublingual route in cats is as effective as the IV route (note: 0.02mg/kg) which makes it a good choice for hospitalised cats requiring ongoing pain management. Its duration of action is 5-6 hours.
Pethidine also acts on mu receptors providing good analgesia. It has a rapid onset of action around 5 minutes and duration of action is around 90 minutes. This means surgical patients need frequent re-dosing to maintain pain control. Pethidine cannot be given IV because like morphine it causes histamine release which may lead to anaphylactic reaction.
Butorphanol is useful because it provides excellent sedation in combination with acepromazine or medetomidine. The duration of action is controversial with some studies proving a similar efficacy and duration to other opioids. Pure mu agonists provide a much better level of analgesia and so butorphanol does not really has a place in provision of analgesia. The licensed analgesic dose is 0.4mg/kg. Work by Camargo et al (2011) documents that firocoxib provides better analgesia than butorphanol post spay. This paper also reviews other studies documenting butorphanol to be not a particularly good analgesic.
Fentanyl is a pure mu agonist which provides good analgesia but is of a very short duration. It is commonly used as a constant rate infusion in an ICU setting. However when formulated as a slow release patch analgesia can be provided continuously for up to 3 days in dogs and 5 days in cats. Fentanyl patches are applied to clipped skin of the patient in a location where they cannot be tampered with. The onset is slow – normally 18-24 hours in dogs and 6-12 hours in cats. This means analgesia must be provided using another opioid whilst the patch starts to work. Patches can be sent home on a patient provided they do not have young children who may potentially ingest the patch.
Recuvyra (topical fentanyl solution) is no longer marketed in the UK.
Fentanyl is licensed in dogs as Fentadon (Dechra).
Codeine use for analgesia is anecdotal and not supported by the scientific literature. It is included in Pardale V but is extensively metabolised and only 4% bioavailable.

Side-effects
The main effects described for the opioids are respiratory depression, cardiovascular depression and slowed gastrointestinal motility. The degree of respiratory depression varies according to the opioid but of the opioids used in clinical practice is rarely of significance to cause concern. Intravenous fentanyl will cause respiratory depression in most patients when used at high doses so in an ICU setting it is used at low doses which provide analgesia without respiratory depression. Methadone causes panting and whining in dogs. Cardiovascular depression is usually manifest as bradycardia and depends upon the opioid and the route of administration – examples of this occurring would be IV fentanyl or methadone. If a bradycardia is induced by the opioid, which then affects blood pressure then the bradycardia should be treated with an anticholinergic or the opioid dose lowered for subsequent use.

Non-steroidal anti-inflammatories (NSAIDS)
NSAIDs are often a first line treatment for painful conditions as most are available in an injectable preparation and have a long duration of action of around 24 hours. NSAIDS reduce the cardinal signs of inflammation such as heat, redness, swelling and pain.

Examples of injectable non-steroidal anti-inflammatories are meloxicam, carprofen, robenacoxib, firocoxib, cimicoxib, mavacoxib. They act in the periphery where inflammation is present. Inflammation occurs following tissue damage and disruption of cell membranes. Components of the cell membrane, such as phospholipids are degraded by certain groups of enzymes to produce inflammatory mediators. Inflammatory mediators act on the nociceptors and cause them to fire, thus initiating an action potential which conveys the pain stimulus. NSAIDS inhibit the cyclooxygenase enzyme group and therefore prevent the production of inflammatory mediators. The main inflammatory mediators of concern are prostaglandins and thromboxane A2. There is increasing evidence of a central effect through inhibition of phospholipase A2 in the spinal cord.

Which is the best NSAID?
There are no studies demonstrating superiority with regard to analgesia or side effect profile in dogs. Recent work in cats demonstrates superiority of robenacoxib over meloxicam. (Kamata, King, Seewald, Sakakibara, & al, 2012). NSAID choice is often based on formulation and frequency of dosing.

NMDA antagonists
The most common NMDA antagonist we use in every day practice is ketamine. Ketamine is a modulator. The NMDA receptor is located in the dorsal horn of the spinal cord and becomes activated in cases of extreme or persistent pain. This is one reason why there is such an emphasis on pre-emptive analgesia especially for elective surgical cases where pain can be anticipated. For many of our painful cases we are unable to anticipate pain as the animal is already in pain upon presentation. In these cases we must aim to use analgesics, such as methadone and ketamine which are known to act on the NMDA receptor, but also not to forget to continue analgesia as long as the animal is painful so there is no gap in the provision of pain care.
In a critical patient in need of good pain control, ketamine is used as a constant rate infusion in conjunction with opioids.
I commonly use ketamine as a CRI 10mcg/kg/min during surgery, dropping to half this rate for recovery. Suitable cases would be amputations, spinal surgery, orthopaedic surgery, TECA's. It should not be used as a sole analgesic, always against an opioid background as part of a multimodal approach.
For cats, add 75mg to 500ml Hartmann's solution and run at 2ml/kg/hr.

Paracetamol
Paracetamol is considered a NSAID by some and not by others. This is due to uncertainty about its exact mechanism. Paracetamol is licensed in dogs as Pardale V for 5 days. The dose in Pardale V works out at 33mg/kg. The listed does in most formularies is 10mg/kg BID-TID.
Paracetamol is available as an injectable for IV use (Perfalgan). I use it at 10mg/kg BID-TID in dogs perioperatively but will use the labelled Pardale dose for chronic pain cases.
A recent retrospective study compared paracetamol 10mg/kg TID to meloxicam 0.2mg/kg in dogs undergoing stifle surgery. This study showed that pain scores were similar between groups although the paracetamol dogs received less methadone post op based on the opinion of the person pain scoring. A prospective study documented similar findings. Paracetamol is useful as an alternative to NSAIDs in dogs that will not tolerate an NSAID. It should never be used in cats.

Constant (continuous) Rate Infusions
CRI's are useful in hospitalised patients to provide analgesia. The use of a CRI avoids the peaks and troughs of plasma levels associated with bolus dosing of drugs. Drugs suited to use as CRI's should be short acting otherwise these drugs will cumulate. Of the opioids, fentanyl is best suited to use as a CRI although more is also used. Other drugs used as CRIs include lidocaine and ketamine.

Drug	Rationale	Dose	Caution
Fentanyl	Excellent analgesia	2-5µg/kg/hr Higher rates used during surgery 10-20µg/kg/hr) – may necessitate IPPV.	Respiratory depression at high doses (20µg/kg/hr)
Lidocaine	Analgesia, anti-inflammatory, anti-oxidant, pro-motility	30-100µg/kg/min 2mg/kg IV loading dose in dogs.	Highly protein bound. Caution w cats. ? anorexia?
Methadone	Excellent analgesia	0.1mg/kg/hr	Cumulative after ~24hrs Stop CRI if dog excessively sedated or cat dysphoric.
Ketamine	Excellent analgesia	10µg/kg/min during sx, 2µg/kg/min post op. 0.5mg/kg loading dose IV or 1mg/kg IM.	Use in combination w opioids not on its own
Dexmedetomidine	Analgesia & sedation	1-2µg/kg/hr	Only if CV system stable

Lidocaine
As well as acting on neurones to block conduction, local anaesthetics also act on pathways used by other pain related mediators. Actions at G protein couple receptors is well documented as well as inhibition of substance P and endothelin receptors (both GPCRs). At low doses lidocaine suppresses the activation of sensory neurons by bradykinin. These inflammatory components are known to contribute to post-incisional pain.

Rescue Analgesia
In situations where a local technique has not completely worked or cannot be used, it is useful to consider rescue analgesia. Remember that simply turning up the vaporiser does nothing to prevent transmission of the painful stimulus.
By taking a baseline HR, RR, BP you can gauge the animal's response to surgery. If these parameters increase 20% above baseline in response to surgery then further analgesia is required.
Options:
Fentanyl 1-2mcg/kg IV
Ketamine 0.5mg/kg IV diluted or 1mg/kg IM
Dex/medetomidine 1-5mcg/kg IV
Repeat the opioid used in the premed.

Nursing care & pain management?
Good nursing care of veterinary in-patients plays a crucial role in overall pain management. All patients should have a comfortable, clean bed at an ambient temperature. The opportunity to toilet outside should be considered for individual cases as appropriate. Stroking and grooming are good ways to interact with patients. Naturally cats and dogs should be kept apart. Any dressings should be checked regularly to ensure they are clean and comfortable.

Local Anaesthetics
An action potential is propagated down a nerve because of activity in sodium channels. Local anaesthetics block sodium channels and therefore block transmission of the painful stimulus via the A and C fibres. Local anaesthetics act on nerves in the periphery, depending on where they are injected.
Local anaesthetics reversibly block sodium channels in the nerve membrane which are necessary for membrane depolarisation. The conduction of the action potential is halted and thus transmission of the stimulus is prevented.

Ideal properties of a local anaesthetic
- Rapid in onset
- Long duration
- Highly potent
- Small volume
- Low toxicity
- No pain upon injection
- Cost effective

Loco-regional anaesthesia is well detailed in the BSAVA Manual of Anaesthesia & Analgesia 3rd edition and following notes will detail commonly used techniques.
Benefits of loco-regional techniques are well summarised by Campoy and others (2008).
'*Local anaesthesia prevents nociceptive impulse transmission therefore minimises central sensitisation which reduces the requirement for post-operative systemic analgesics.*'
The focus of current research is on developing new veterinary techniques, which are often modifications of techniques in humans, documenting new approaches to well-established techniques and improving the efficacy of techniques.
The basic principle of regional anaesthesia was stated in an editorial in the British Journal of Anaesthesia:
'*Regional anaesthesia always works – provided you put the right dose of the right drug in the right place'*(Denny & Harrop-Griffiths, 2005).
Techniques to improve accuracy and therefore efficacy include ultrasound guidance and the use of a nerve locator, both of which are reported for blockade of nerves innervating the pelvic limb in dogs (Campoy, Martin-Flores, Ludders, Erb, & Gleed, 2012; Shilo et al., 2010).
Alternatives to extradural (epidural) anaesthesia include femoral and sciatic nerve blocks. These blocks have been documented as safe and efficacious (Vettorato et al, 2012) and to have an opioid-sparing effect in dogs when compared to a morphine/bupivacaine extradural technique (Campoy et al., 2012). Extradural anaesthesia has been comprehensively reviewed (Valverde 2008). Pelvic limb peripheral nerve blocks has been reviewed by Gurney & Leece (2014), a copy of which is included with these notes.

How can local anaesthetics be used?
The most common use of local anaesthetics is in nerve blocks whereby local anaesthetic is infiltrated around the nerve in anaesthetised patients. This is particularly useful for dentistry as the nerve blocks are easy and quick to perform and the patient is much more comfortable after surgery. Evidence from human studies document better patient satisfaction where first day pain is more effectively controlled.
Local anaesthetic techniques should be considered in all cases undergoing a surgical procedure
- Reduce volatile agent requirements & associated cardiopulmonary depression
- Superior pain control
- Decrease requirement for other analgesics
- Key to multimodal analgesia

Local anaesthetics are commonly incorporated into epidurals to provide a complete absence of sensation from the caudal half of the animal.

What are the benefits of using local anaesthesia?
With practice and a basic knowledge of anatomy local analgesic techniques are quick and simple to perform and add greatly to balanced anaesthesia. The benefits to the animal are reduced post-operative pain and reduced intra-operative pain.

As a result of this the anaesthetic will be much smoother and the requirements for maintenance agents will be much reduced.

Which agents are available in practice?
Lidocaine, bupivacaine, levobupivacaine and ropivacaine are all (available) in preparations suitable for infiltration and are most commonly used in small animal practice. Neither bupivacaine, levobupi nor ropivacaine are licensed for veterinary use. Lidocaine without adrenaline should be used.

The ideal properties of a local anaesthetic
The ideal local anaesthetic should be rapid in onset so that it is working by the time the surgeon incises but should have a long duration so that it is working for the duration of the surgery and well into the post op period. Ideally the chosen drug would be a small volume that does not cause pain on injection. Individual properties of local anaesthetics are listed here.

	Lidocaine	**Bupivacaine**	**Levobupivacaine**	**Ropivacaine**
Onset	fast 5-10mins	moderate 20-30mins	moderate 20-30	moderate 20-30mins
Duration	short 1-2hrs	long 4-6 (10)hrs	long 4-6hrs	long 4-6hrs
Potency	+	+++	+++	+++
Toxicity	+	+++	++	++
Therapeutic Doses	4mg/kg	1-2mg/kg	1-2mg/kg	2-3mg/kg
Toxic Doses	>20mg/kg	>2mg/kg	>3mg/kg	>3mg/kg
Trade Name	Various	Marcain®	Chirocaine®	Naropin®

Unfortunately none of these properties are found in one preparation, however can be partly achieved by combining products. Lidocaine and bupivacaine combination is thought to provide a rapid onset with a good duration of action whereas lidocaine/ropivacaine gives a similar duration of action with less risk of cardio-toxicity: this however has not been evaluated in dogs or cats.

Additives
Various additions have been reported. Adrenaline causes vasoconstriction and keeps the local anaesthetic near the site – or also prevents diffusion away and absorption. Bicarbonate alters the pH and increases the speed of onset.
Of most interest is the addition of dexmedetomidine to the local anaesthetic to prolong action. Exact dose required is yet to be determined in dogs.

Extended action
A liposome encapsulated bupivacaine is currently marketed in people and may be released with a vet license later this year in the UK. The lipid bilayer of the liposomes releases over time to give an anaesthetic effect up to 96hrs (Lascelles & Kirkby 2016).

Toxicity
Local anaesthetics reversibly block sodium channels in excitable tissues which as well as nerves includes the myocardium. This blockade affects the cardiac action potential. Bupivacaine is ten times slower to dissociate away from sodium channels than either lidocaine or ropivacaine and this may lead to arrhythmias and ventricular fibrillation. For this reason neither bupivacaine nor ropivacaine should be used IV.
Levobupivacaine, an isomer of bupivacaine has a better CV side effect profile than bupivacaine however should still not be administered IV. The dose for bupivacaine and levobupivacaine is the same at 1-2mg/kg.

There is a risk of inadvertent lidocaine overdose in small cats and kittens when using lidocaine sprays for desensitising the larynx. One spray contains 2-4mg of lidocaine. An alternative technique for small patients is to draw up the calculated dose of 1% lidocaine into a syringe and apply directly onto the larynx. If local anaesthetic techniques are also performed in these patients this should be borne in mind when calculating doses.

Central nervous system effects may be seen in cases of overdose starting with muscle twitches. Inhibitory interneurones are blocked which causes excitation followed by convulsions. If all central neurones are depressed this will eventually lead to coma and respiratory depression before cardiac side effects are seen.

In a practice where a large number of local anaesthetic techniques is being carried out it is wise to stock a lipid emulsion solution. This solution acts as a lipid sink and when injected IV it binds the lipid soluble local anaesthetic and prevents it from acting. It is the same lipid rescue used for permethrin toxicity.
View the video demonstrating this here http://lifeinthefastlane.com/intralipid-myth-or-miracle/
In humans a dose of 1.5ml/kg lipid rescue is administered IV with CPR starting, plus an infusion of 0.25ml/kg/hr. Studies in dogs have shown successful resuscitation with 4ml/kg and 30ml/kg/hr CRI.

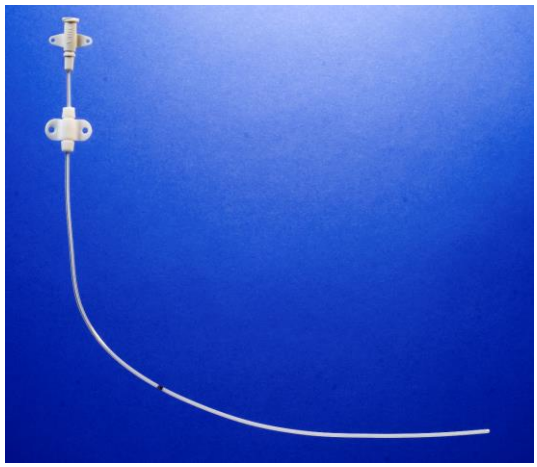
The key to reducing toxic potential is to use only as much local anaesthetic as is needed. In the human field with an awake patient they can be asked when sensation disappears, however we do not have this luxury in our patients! In some situations the use of a peripheral nerve stimulator allows for more accurate infiltration. A needle is used that a current can be passed down to stimulate the motor nerve being blocked. Following infiltration the nerve is again stimulated to check correct deposition of local. In veterinary patients this technique may be used for a brachial plexus nerve block, however would be ineffective for a maxillary nerve block because the maxillary nerve contains only sensory fibres in this region.

Contraindications to local anaesthesia

- Infection
- Neoplasia

Wound catheters

Potential advantages of continuous infiltration anaesthesia via wound catheters has attracted interest amongst veterinary anaesthetists. In humans where there is a significant body of evidence supporting lower pain scores, reduced opioid consumption and shorter hospital stays (Macintyre and others 2010, Gupta and others 2011). In cats undergoing fibrosarcoma resection the use of wound catheters for analgesia significantly reduced the length of hospitalisation (Davis, Hardie, Martin, Zhu, & Brownie, 2007). Further studies documenting analgesic benefits are required in veterinary patients (Abelson et al., 2009). I use bupivacaine 1-2mg/kg every 6-8 hours in cases such as fibrosarcoma resection and limb amputation for 24 hours post surgery. I do not use a CRI method for delivering the local but prefer an intermittent method.



If you are placing a wound drain as well as a local infusion catheter ensure you drain the wound first and then instill the local.
The distributor for these is Mila. Lengths range from 2 to 9 inches.

Nerve Location
Electrical nerve locators are used to increase the accuracy and safety of local anaesthetic techniques. This relies on the nerve being stimulated having a motor component – stimulation of the nerve produces a response in the corresponding muscle. An electrical nerve locator (ENL) will stimulate muscular twitching at a close distance to the nerve without actually touching it; hence, providing greater accuracy for local anaesthetic deposition. Needles have an insulated shaft so the current comes from the tip. The needle is connected to the current meter and a syringe.

The further the needle is from the nerve the greater the current required to stimulate (Coulomb's Law). However, if the needle is in the nerve, a twitch may not be seen – only when the needle is moved away does a twitch appear. The aim is to get the needle as close as possible to the nerve without actually touching the nerve. The ENL is essentially a current generator – the output can be varied. For most blocks start at a current of 1mA. Once the nerve is located step down to 0.2mA in increments of 0.2mA– at this current no stimulation should occur. If stimulation does occur at 0.2mA the needle is too close to the nerve sheath and at this point injection could cause nerve damage. Reposition the needle in this case.

Once you are at a current of 0.2mA with no stimulus, go back up to 0.4mA at which point you should see the twitch resume. Aspirate & start the injection –the stimulation will cease – this is not the local working but the nerve being separated from the stimulating needle by the fluid injected.

Practical Tips
Move the needle forwards and backwards and not laterally. This may push the needle towards the nerve, but there may still be tissue in between the needle and the nerve.
Investigate the area in an arc when searching for the nerve.
Aspirate before every injection.

Nerve locators are available from Vygon.

Ultrasound-guided nerve location
Ultrasound in regional anaesthesia offers a new standard in nerve location and identification, allowing real-time imaging of nerves and direct needle guidance.
All needles show up equally well on ultrasound, but it is imperative that the needle tip is in the field of vision at all times.
The aim is not to touch the nerve but place the needle close to it; nerve identification can be confirmed by the combined use of peripheral nerve stimulation.
A successful block is one in which the local anaesthetic is seen to spread around the nerve under direct vision – referred to as the donut sign.
Ultrasound location offers the opportunity to improve success, reduce complications, and enhance teaching of regional anaesthesia.
Blocks in dogs and cats where this technique is used are the brachial plexus block (axillary approach), and the femoral-sciatic block. (Campoy et al., 2010).