Get the hang of it – Analgesia and anesthesia in small animal practice

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OVERVIEW

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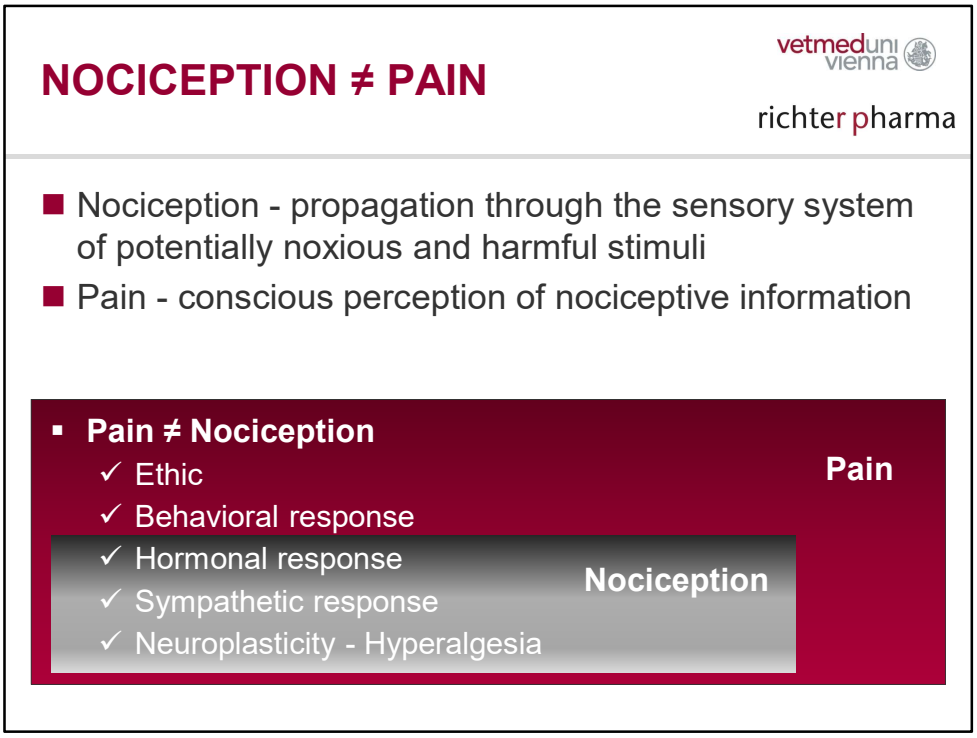
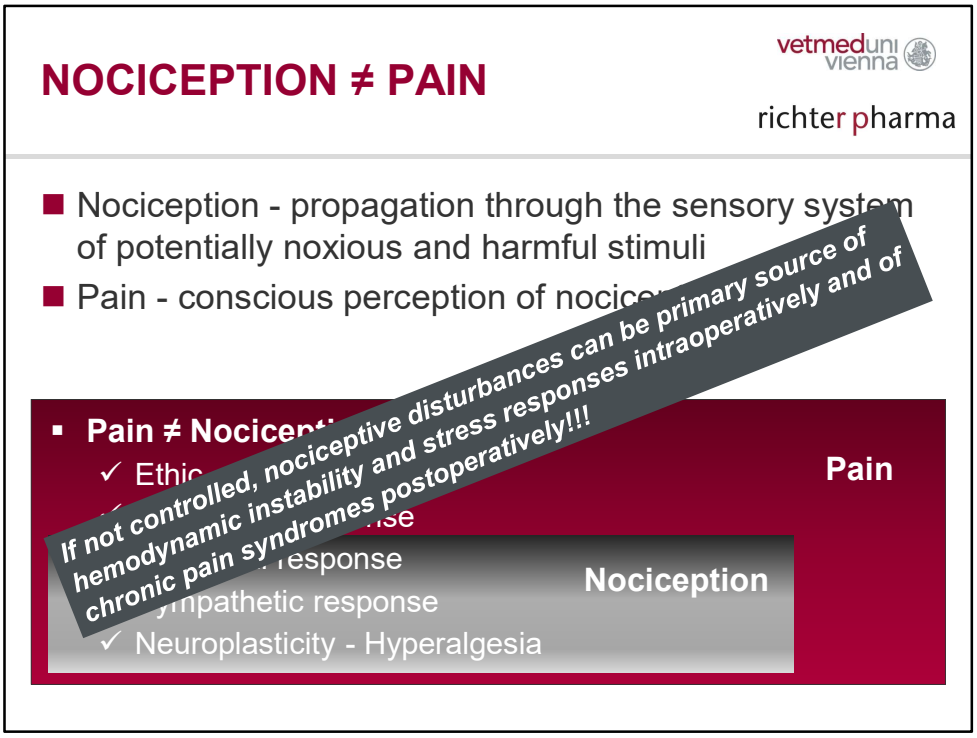
- ✓ Differences between pain and nociception
- ✓ Definition of multimodal analgesia, pre-emptive vs. preventive analgesia
- ✓ Definition of balanced general anesthesia
- ✓ Formulation of a rational strategy for balanced general anesthesia and analgesia management
- ✓ Examples summarizing anesthetic and analgesic management for 2 representative surgeries (fracture repair, exploratory laparotomy)



One needs to distinguish between nociception and pain.
For example, if a patient is unconscious after receiving only propofol and has an increase in heart rate and blood pressure in response to the surgical incision, then this is an example of nociception.
If a surgeon makes an incision to create a dialysis fistula after an inadequate administration of local anesthesia for a field block and the patient says, “Ouch,” then this is pain.
Nociception induced by surgery, due to tearing of tissue and inflammation, is the primary reason for placing a patient in a state of general anesthesia.
“An unpleasant sensory and 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 an individual is experiencing pain and is in need of appropriate pain-relieving treatment.”



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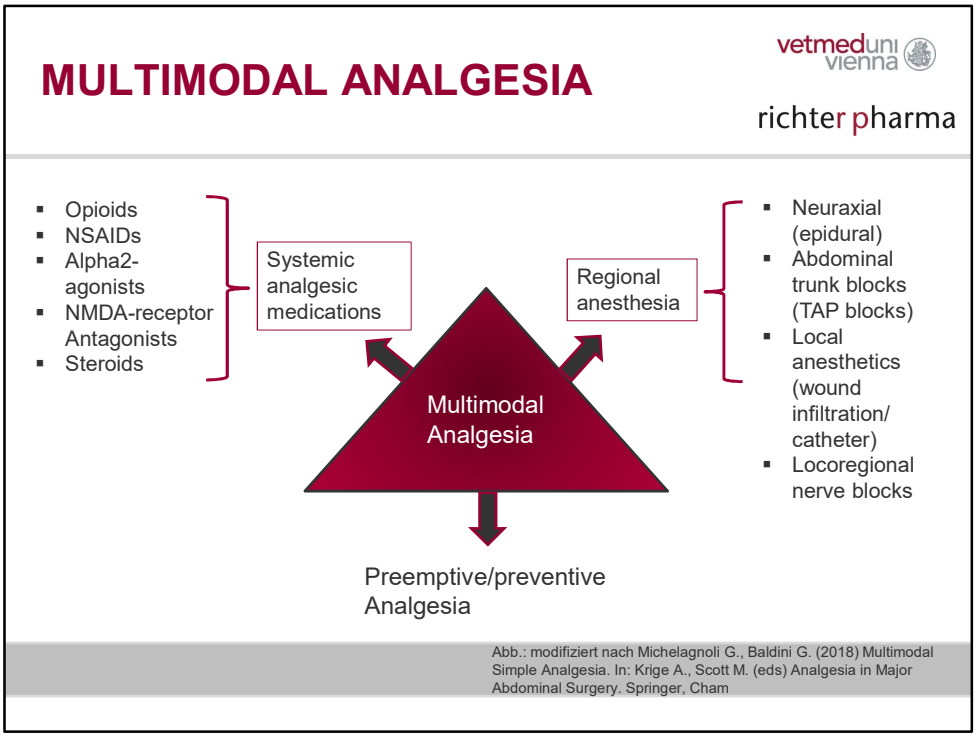

Multimodal analgesia relies on the principle of administering different types of analgesic medications, which through an additive or synergistic effect can improve post-operative pain control
A multimodal analgesic approach allows a reduction in systemic opioid requirements and their associated adverse effects.

MULTIMODAL ANALGESIA

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- A multimodal analgesia protocol should be:
  - ✓ Procedure-specific
  - ✓ Include non-opioid analgesics administered regularly
  - ✓ Systemic opioids to treat moderate-severe breakthrough pain
  - ✓ Regional anaesthesia techniques for certain surgical procedures

PRE-EMPTIVE ANALGESIA

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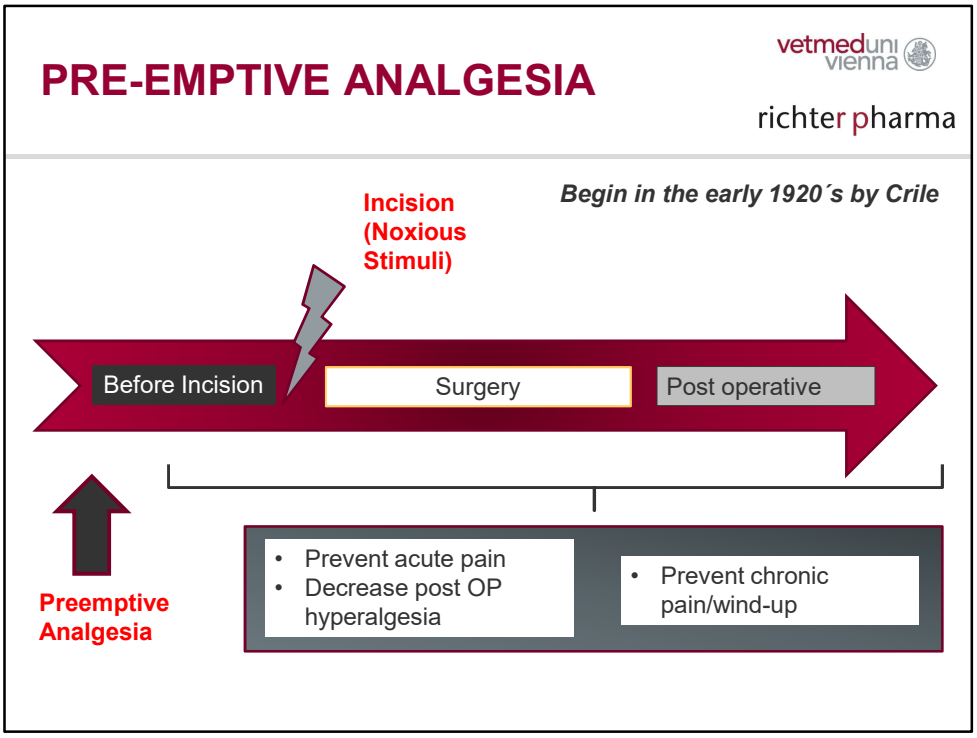
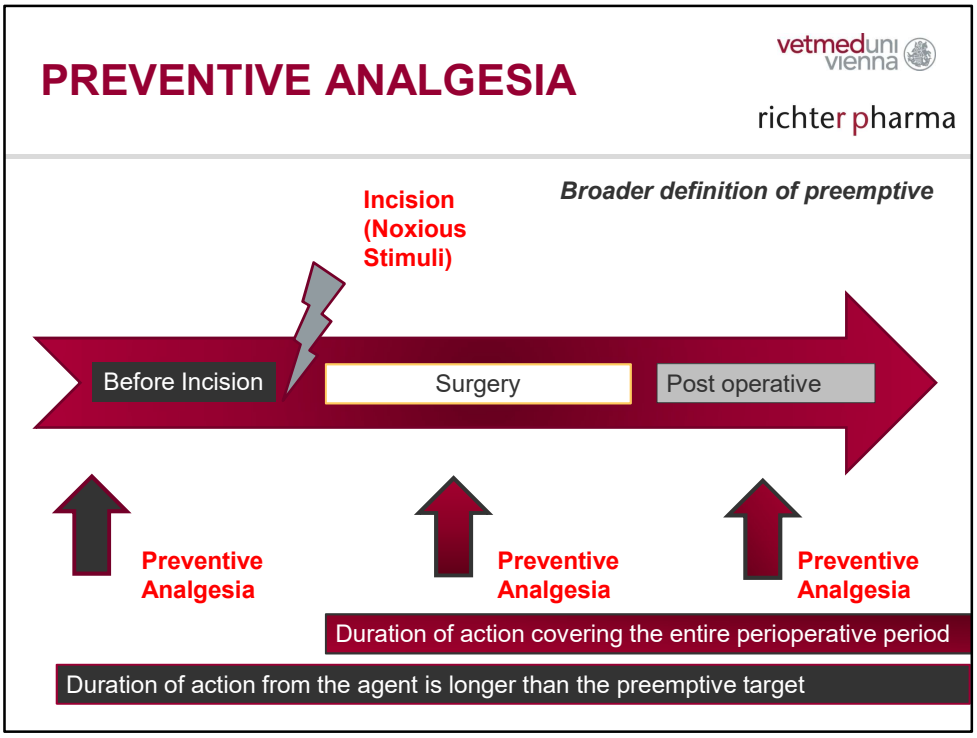
- Pre-emptive analgesia has been defined as antinociceptive treatment that:
  - ✓ Starts **before** surgery
  - ✓ Prevents establishment of central sensitization caused by incisional injury (**covers only the period of surgery**)
  - ✓ Prevents establishment of altered processing of afferent input, which amplifies postoperative pain



CNS Protection until post operative period
Note: pain modulation by the CNS takes place before perception
Pre-emptive analgesia would block the induction of **central neural sensitization** brought about by the incision and reduce the intensity of acute postoperative pain (proposed first by Crile and later by Wall)
General anesthesia may attenuate the transmission of afferent injury barrage from the periphery to the spinal cord and brain, but it **doesn't block** the transmission


PREVENTIVE ANALGESIA
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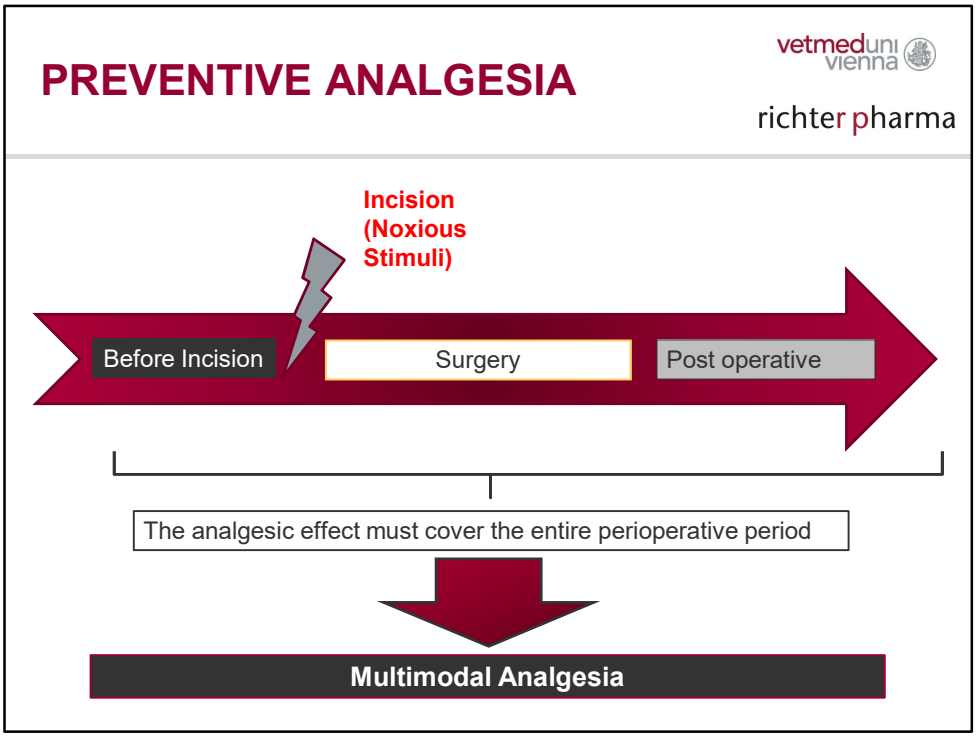
Broader definition of preemptive
Incision (Noxious Stimuli)
Before Incision
Surgery
Post operative
Preventive Analgesia
Preventive Analgesia
Preventive Analgesia
Duration of action covering the entire perioperative period
Duration of action from the agent is longer than the preemptive target


PREVENTIVE ANALGESIA
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Incision
(Noxious
Stimuli)
Before Incision
Surgery
Post operative
The analgesic effect must cover the entire perioperative period
Multimodal Analgesia

BALANCED GENERAL ANESTHESIA

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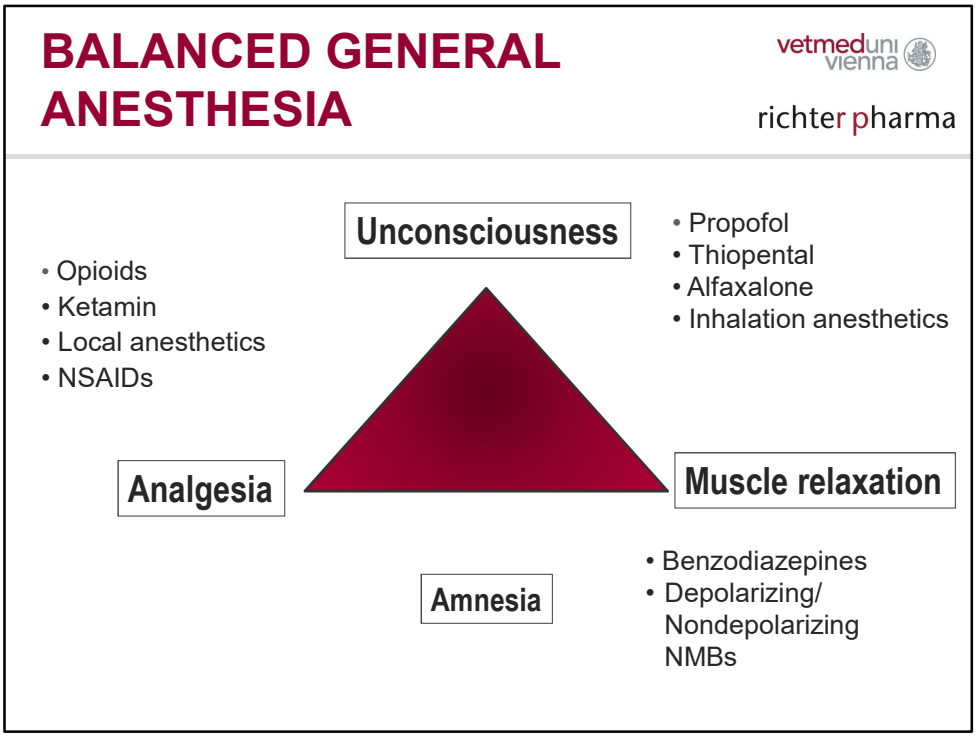
- Balanced general anesthesia - most common management strategy used in anesthesia care
- Entails administering a combination of different agents to create the anesthetic state
- There is evidence that balanced general anesthesia uses less of each drug than if the drug were administered alone
- This approach is believed to increase the likelihood of a drug's desired effects and reduce the likelihood of its side effects


BALANCED GENERAL ANESTHESIA
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Unconsciousness
• Propofol
• Thiopental
• Alfaxalone
• Inhalation anesthetics
• Opioids
• Ketamin
• Local anesthetics
• NSAIDs
Analgesia
Muscle relaxation
Amnesia
• Benzodiazepines
• Depolarizing/ Nondepolarizing NMBs

BALANCED GENERAL ANESTHESIA

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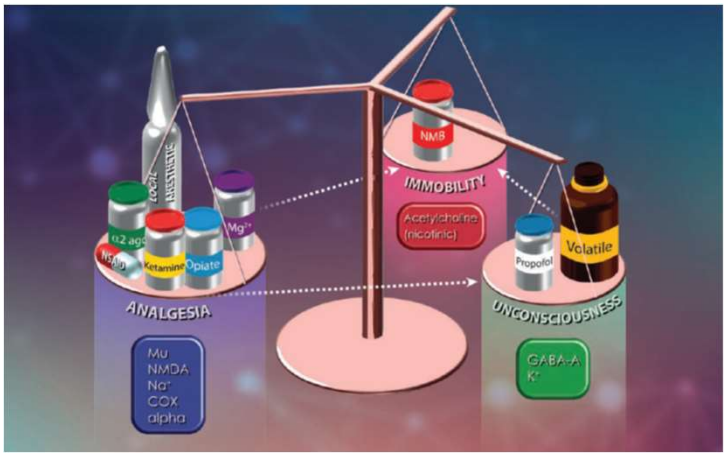

Picture: Balancing Act: Multimodal General Anesthesia; Nathan, Naveen MD
Anesthesia & Analgesia: November 2018 - Volume 127 - Issue 5 - p 1097

Bild selber zeichnen, weniger Medikamente in den Wagschalen!!!

BALANCED GENERAL ANESTHESIA

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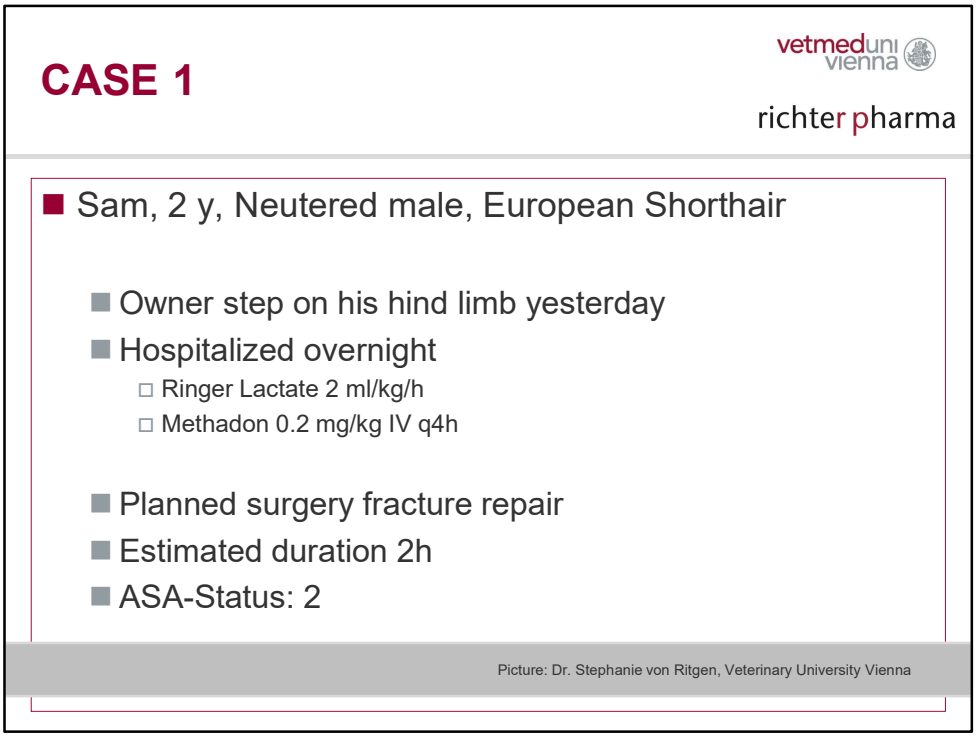
- A rational strategy for balanced general anesthesia should include:
  - ✓ Administration of a combination of antinociceptive and sedative drugs - chosen that each drugs targets a different part in the nociceptive and arousal system
  - ✓ Use sedative effects of the antinociceptive drugs to reduce the doses of hypnotic agents and inhaled anesthetics administered to maintain unconsciousness
  - ✓ Continuously monitor levels of nociception and unconsciousness
  - ✓ Continue multimodal pain control during the in-hospital postoperative period



You are preparing to provide general anesthesia for a 2-year-old neutered male, European Shorthair undergoing fracture repair of her right hindlimb, after the owner step on his cat yesterday. Sam was hospitalized over night receiving an infusion of ringer´s lactate and methadone every 4 hours as analgesia. Sam is otherwise healthy.

CASE STUDY 1

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- Question:
  - When you look to the analgesia the patient received over night, is there something missing?
  - Is/ or should an NSAID be part of the premedication or better not?

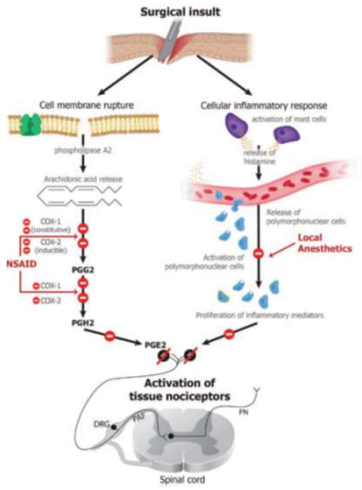
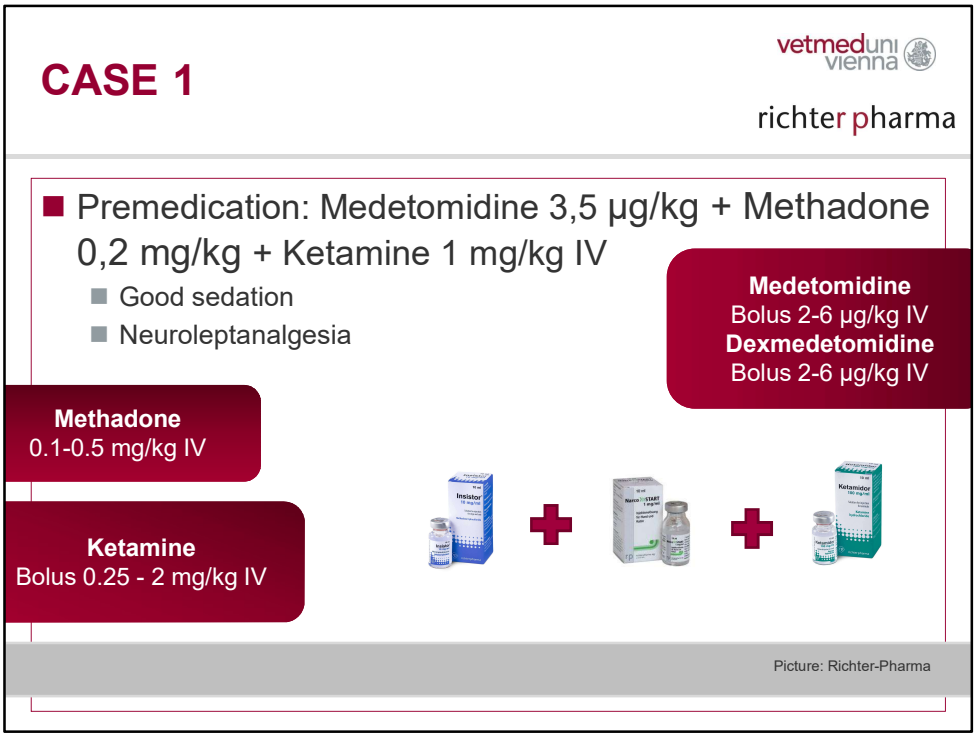


Figure from Brown, E.N.,Kara J. Pavone, K.J., Naranjo, M. Multimodal General Anesthesia: Theory and Practice. Anesth Analg 2018;127:1246–58

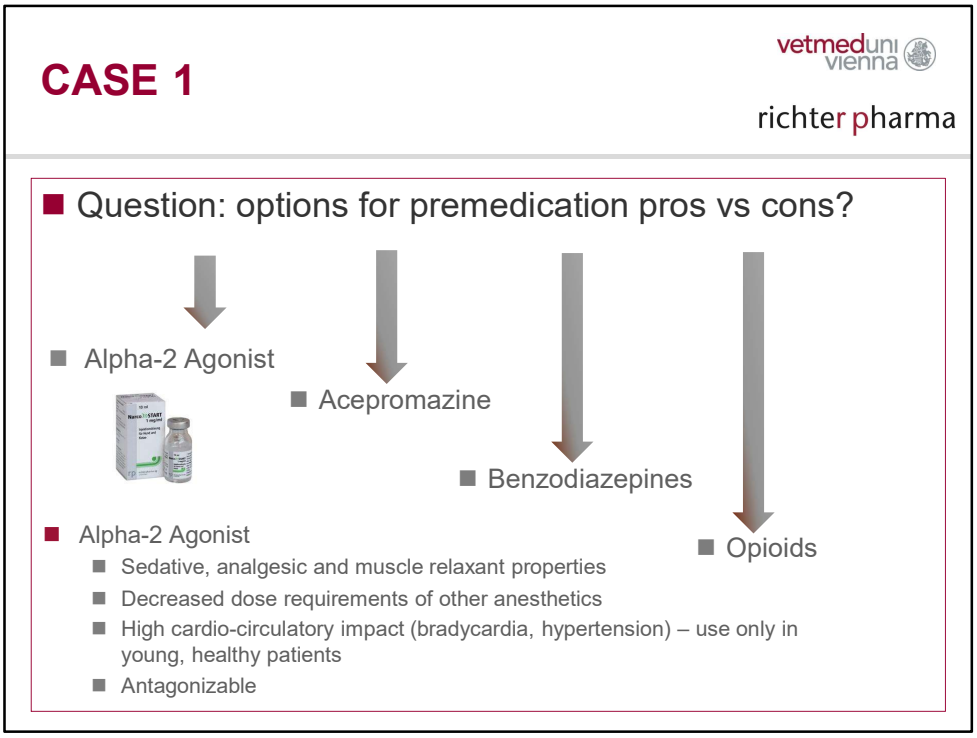
NSAIDs and lidocaine. Surgical insults induce rupture of cell membranes, leading to release of arachidonic acid, which, through the action of COX-1 and COX-2, is converted into
prostaglandins, which are potent inflammatory and nociceptive mediators. NSAIDs modulate the nociceptive response by blocking the actions of COX-1 and COX-2, and lidocaine exerts their nociceptive effects by inactivating sodium channels, thus inhibiting excitation of nerve endings and blocking conduction of action potentials in peripheral nerves. Lidocaine also impedes neutrophil degranulation, thereby impeding the amplification of the inflammatory response. COX indicates cyclooxygenase; DRG, dorsal root ganglion; NSAID, nonsteroidal anti-inflammatory drug; PAF, peripheral afferent fiber; PGE2, prostaglandin E2; PGH2, prostaglandin H2; PGG2, prostaglandin G2; PN, projection neuron.
In the preoperative phase or first phase of the anesthetic event, the patient's health status must be determined. A thorough patient history should be obtained, a full physical examination should be performed, and all appropriate preoperative blood work (e.g., complete blood count, serum chemistry panel) should be obtained. In addition, urinalysis, radiography, electrocardiography, and other diagnostic tests may be required in order for the veterinarian to fully evaluate the patient. After all of the test results have been reviewed, an anesthetic protocol can be developed. The drugs and monitoring equipment that are used should be based on patient assessment, the proposed

procedure, anticipated complications and pain, and the estimated length of the anesthetic period.1

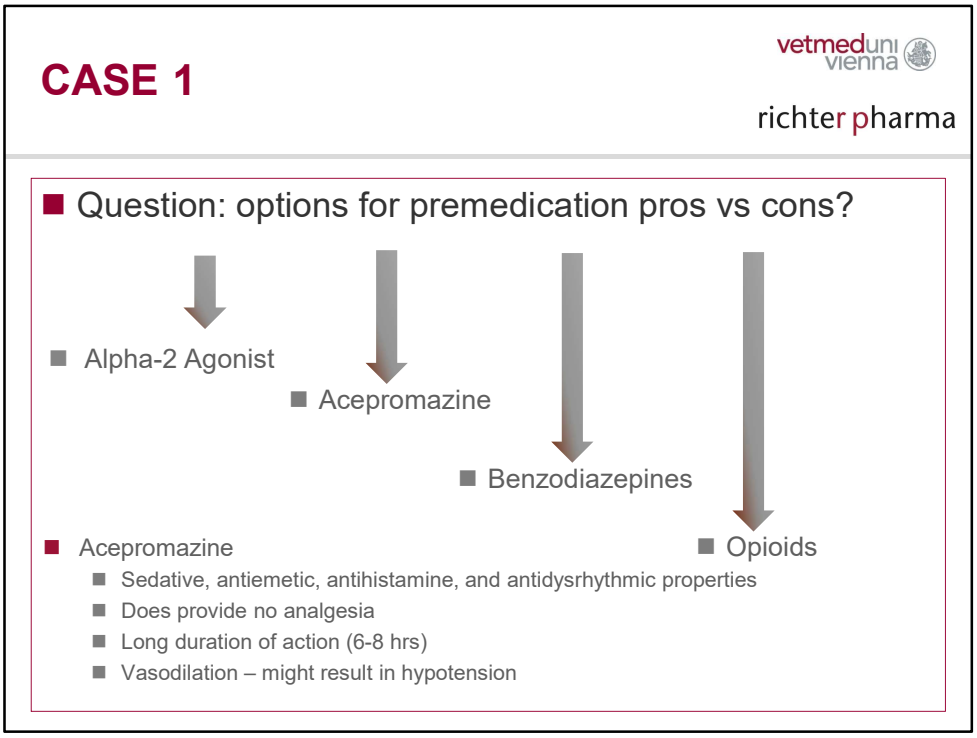


The second phase of the anesthetic event is premedication. The goals of this phase are to relieve apprehension, administer analgesia so that it is in full effect during the surgical phase, facilitate restraint, provide neuroleptanalgesia, and use sedation to decrease the dose rate and minimize adverse effects of the induction drugs. A combination of two or three agents from four or five drug classes — sedatives and tranquilizers, NSAIDs, and opioids — is commonly used during premedication.
Analgesics should be used to provide pain relief throughout a procedure. A multimodal approach that includes many combinations
Goal of premedication!
In order to reach the maximum effect of anesthesia in the perioperative phase, neuroleptanalgesia should be achieved in the premedication phase. The combination of an opioid and a major tranquilizer provides more sedation and analgesia than either drug would if used alone and provides mild to marked effects. For example, the combination of midazolam and butorphanol would be far less potent than the combination of acepromazine and morphine. When selecting a major tranquilizer, the patient's age, temperament, and preexisting health problems as well as the degree of tranquilization needed, should be considered. To choose an appropriate opioid, the patient's analgesic

requirements, as well as the drug's convenience, availability, speed of onset, length and adequacy of analgesia, side effects, and reversibility, should be considered.[1]



AMedetomidine, which is approved for use in dogs, is an a_2 agonist that has sedative, analgesic, and muscle-relaxant properties.[4] This drug can be used for short procedures in young, healthy patients. The advantages of medetomidine are that it decreases the dose requirements of other anesthetic agents, has excellent analgesic properties, and is reliably reversible by atipamezole. Because medetomidine has adverse effects on the cardiovascular system, including profound bradycardia and hypertension, decreased cardiac output, decreased respiration rate, and interference with temperature regulation, it is contraindicated in sick animals.[1] Medetomidine can be titrated from light to heavy sedation because it is dose dependent. Medetomidine should be used for premedication only after its advantages and disadvantages are carefully weighed. Dogs that are extremely excited or agitated may have a decreased response to medetomidine.



Acepromacine antiemetic, antihistamine, and antidysrhythmic properties. Its use is controversial because the drug is believed to decrease the patient's seizure threshold, cause decreased hematocrit levels due to splenic red blood cell sequestration, inhibit temperature regulation, and cause profound vasodilation and resulting hypotension.4 In addition, geriatric patients and large-breed dogs tend to be more sensitive to acepromazine and may require a lower dose.2

CASE 1

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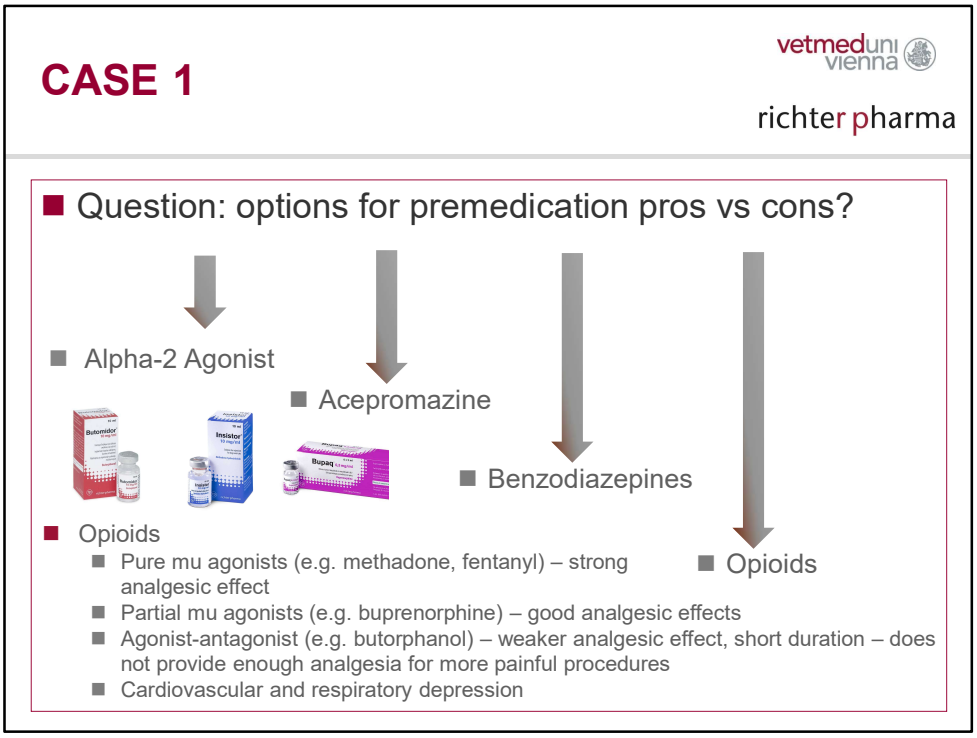
- Question: options for premedication pros vs cons?
  - Alpha-2 Agonist
  - Acepromazine
  - Benzodiazepines
  - Opioids

- Benzodiazepines
  - Sedative and calming effect, muscle relaxation and hypnosis – provide no analgesia
  - Use cautiously in animals with renal or hepatic insufficiencies
  - Excitatory effect in young, healthy animals, effective in geriatric or debilitated patients
  - Antagonizable

Over the past few years, the use of benzodiazepines (e.g., diazepam) for sedation has increased in veterinary medicine. Both diazepam and midazolam are DEA (Drug Enforcement Administration) Schedule IV controlled substances. The sedative or calming effect of benzodiazepines is achieved through depression of the limbic system, thalamus, and hypothalamus, and the muscle-relaxing properties are caused by inhibition of the

internuncial neurons at the spinal level.[2] Benzodiazepines, which are anticonvulsant and anxiolytic drugs, cause muscle relaxation and hypnosis.[1] They should be used cautiously in animals with renal or hepatic insufficiencies — because the liver is the primary organ of metabolism and elimination occurs in the urine. A prolonged effect may be seen in animals with dysfunctions of these organs — and in patients in shock in which underlying bradycardia or hypotension could be compounded.[3,4] Benzodiazepines can have an excitatory effect in young, healthy animals but are effective in geriatric or debilitated patients. Because they do not provide analgesia, benzodiazepines should not be used alone. Because diazepam binds to the plastic on the syringe, precipitates when mixed with drugs other than ketamine, and is not absorbed well when given intramuscularly, midazolam can be substituted.[3] The effects of benzodiazepines can be reversed with flumazenil.



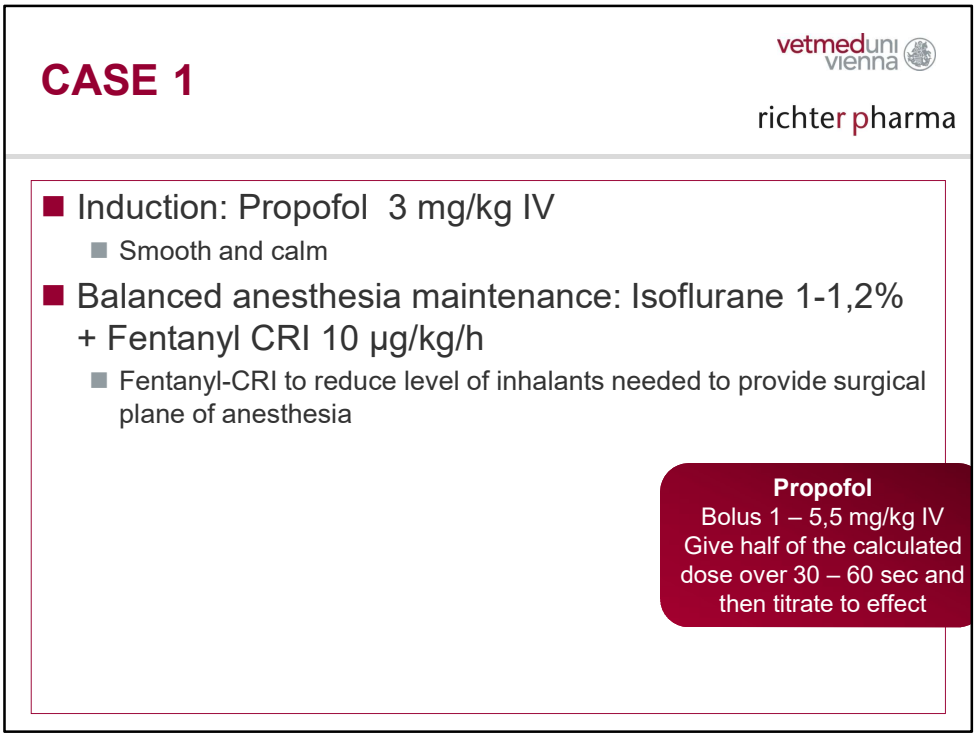
wo classes of opioids, pure mu agonists (e.g., morphine, oxymorphone, hydromorphone, fentanyl, meperidine) and partial mu agonists (e.g., buprenorphine), are generally used in veterinary medicine. When selecting an opioid, the drug's onset and duration of action should always be considered.

Morphine, oxymorphone, and hydromorphone can cause vomiting. This side effect should be considered when administering anesthesia to patients with esophageal or gastrointestinal obstructions, cervical vertebrae instability, or eye problems that are adversely affected by increased intraocular pressure. Vomiting can also cause increased cranial pressure.

Buprenorphine has a long duration of analgesic action similar to, but with far less respiratory and cardiovascular depression than, the pure mu agonists. The analgesic effect of buprenorphine is somewhat less than that of the pure mu agonists; however, the drug provides adequate analgesia for all but the most painful orthopedic procedures.

Butorphanol, which is neither a pure mu agonist nor a partial mu agonist, is an agonist-antagonist and displays agonist activity at the k receptors and antagonizes the pure mu agonists. Butorphanol is a mu antagonist and reverses the effects of the pure mu opioids, leaving the patient with k receptor analgesia, which is weaker than mu analgesia. During recovery, butorphanol may be used to reverse the effects of the mu opioids and to continue to provide analgesia without cardiovascular or respiratory depression.[1] The patient is also likely to be more alert during recovery. However, butorphanol has a short duration of action and does not provide sufficient analgesia for

more painful procedures, such as orthopedic surgery.



When premedication has reached its full effect, the third phase of the anesthetic event, the induction phase, provides a smooth transition to general anesthesia. The goal of this phase is to quickly anesthetize the patient, secure a patent airway by endotracheal or nasotracheal intubation, and prevent vomiting or regurgitation that may result in aspiration. Ketamine, propofol, and thiopental are the drugs most readily available in private practices. Etomidate can also be considered for selected patients. Any of these drugs may be used in most healthy patients. The choice of drugs may be based on cost or the veterinarian's familiarity with the drug.

he fourth phase of the anesthetic event, maintenance of a safe and consistent level of anesthesia, can be achieved with an inhalant anesthetic agent (e.g., isoflurane, sevoflurane, halothane).[1] In the past, halothane was used widely; however, adverse cardiovascular effects and hepatic disease have been attributed to the agent.

In a comparison of isoflurane and sevoflurane, cardiopulmonary effects are indistinguishable. Isoflurane has a higher potency and a lower minimum alveolar concentration value compared with sevoflurane. Less than 0.2% of isoflurane is metabolized; most is exhaled unchanged. Sevoflurane has a minimum alveolar concentration that is twice that of isoflurane and a 3% to 5% metabolism rate. Compared with isoflurane, sevoflurane has faster induction, recovery, and depth change rates.[3] The muscle-relaxing effects of neuromuscular blockers are

enhanced to a greater degree with isoflurane than with the other inhalants.[1] Neither agent should be used when the patient has increased intracranial pressure unless the patient is able to be hyperventilated through manual or mechanical positive-pressure ventilation. The assisted hyperventilation decreases the patient's $PaCO_2$, which, in turn, reduces intracranial pressure. Cerebral blood flow is not increased if adequate ventilation is maintained. If available, sevoflurane is the agent of choice in patients with significant heart disease. Compared with halothane, sevoflurane has a very low incidence of hepatic toxicity.[1]

Although isoflurane and sevoflurane are used during mask inductions, sevoflurane does not irritate the airways and has little or no noxious odor. These attributes of sevoflurane help induce anesthesia in certain species, such as rabbits, that tend to hold their breath.

A concern with the intermittent use of sevoflurane is the formation of compound A, a nephrotoxic by-product of sevoflurane reacting with soda lime. Compound A is formed primarily when low-flow or closed-circuit anesthesia and exhausted/denatured soda lime are used.[1] The concentrations of compound A measured in circle systems using nonexhausted soda lime are five to 10 times lower than those reported to produce toxic effects.[3]

Because isoflurane and sevoflurane are comparable, the expense of sevoflurane should be considered before discontinuing its use. Some clinics may opt to make both drugs available.

In balanced anesthesia, CRIs, intraoperative opioids, local anesthetics, and NSAIDs can be used to reduce the level of inhalants needed to provide a surgical plane of anesthesia.

CASE 1

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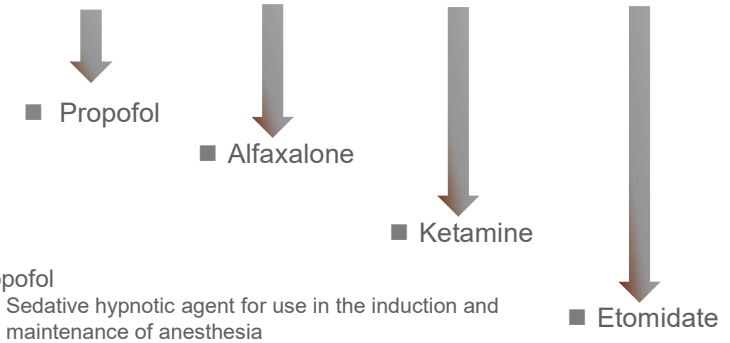
- Question: options for induction agents pros vs cons?



- Propofol
  - Sedative hypnotic agent for use in the induction and maintenance of anesthesia
  - No analgesic effect
  - Main side effect: respiratory depression and apnea – mainly with higher doses or when the drug is administered too quickly
  - May also cause hypotension and vasodilation

CASE 1

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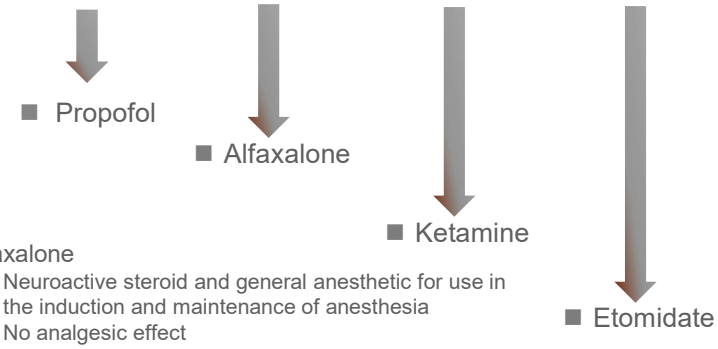
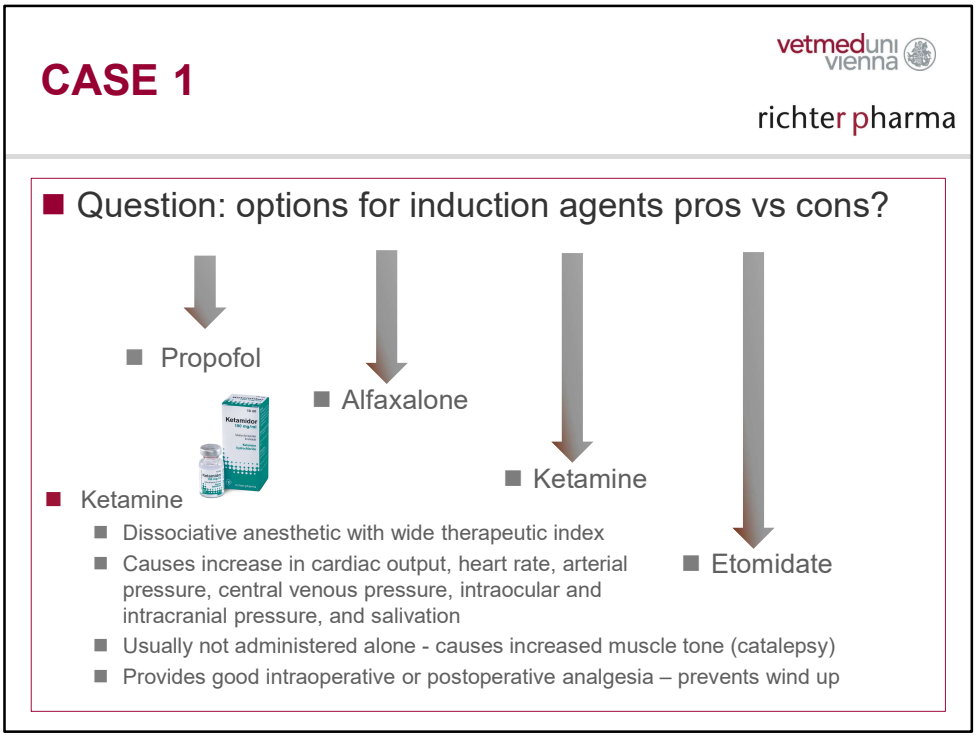
- Question: options for induction agents pros vs cons?

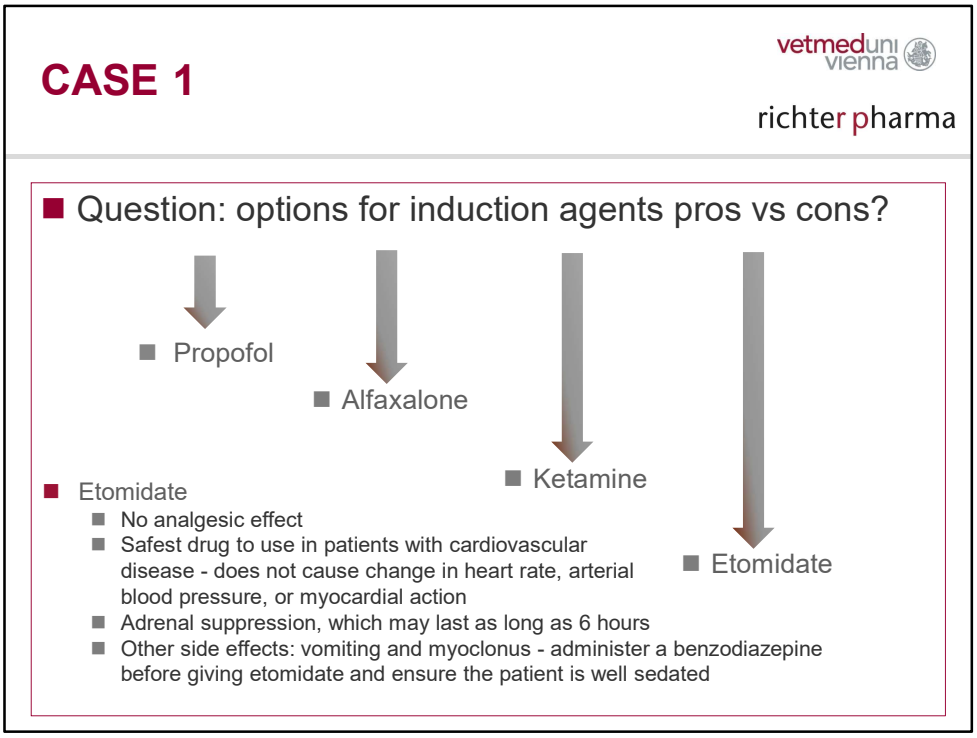


- Alfaxalone
  - Neuroactive steroid and general anesthetic for use in the induction and maintenance of anesthesia
  - No analgesic effect
  - Dose dependent respiratory depression, apnea likely to occur following rapid IV injection
  - Dose dependent cardiovascular depression - significant decreases in both cardiac output and blood pressure



etamine is a long-lasting dissociative anesthetic with rapid onset and a wide therapeutic index.[5] Increasing the dose of ketamine prolongs the duration of anesthesia but does not increase the depth of anesthesia. When ketamine is administered in excessive doses or too rapidly, significant respiratory depression may occur. Ketamine possesses good somatic analgesic properties, but visceral analgesia is poor; therefore, it is far more effective in soft tissue surgeries than in orthopedic surgeries. Ketamine does not cause depression of corneal, laryngeal, pharyngeal, pinnal, or pedal reflexes; however, it does increase cardiac output, heart rate, arterial pressure, central venous pressure, intraocular and intracranial pressure, and salivation, if there is an intact sympathetic system and adequate release.[2] Therefore, ketamine would not be a good choice in patients with tachyarrhythmia, hypertension, heart disease, head trauma, open globe injuries, significant blood loss, or a history of seizures. In patients with decreased sympathetic systems, significant cardiac depression can occur. Ketamine is usually not administered alone because it causes increased sympathetic and muscle tone; it is often combined with diazepam and given intravenously.

Ketamine is widely used for restraint in patients undergoing minor procedures or for sedation of feral or aggressive cats. It is most commonly used in combination with other drugs to produce neuroleptanalgesia and anesthesia. The use of ketamine in a CRI to provide intraoperative or postoperative analgesia is becoming more widespread because of the drug's *N*-methyl-d-aspartic acid antagonism, which prevents spinal wind-up.



Etomidate is not the drug of choice for young, healthy patients because it may not anesthetize them to a level deep enough for intubation.[1] Although etomidate is more costly than propofol or thiopental, it is the safest drug to use in patients with cardiovascular disease because it does not cause a change in heart rate, arterial blood pressure, or myocardial action. It does, however, lower intracranial and intraocular pressures.[4] There is no analgesic effect with thiopental, propofol, or etomidate. The main side effect of etomidate is adrenal suppression, which may last as long as 6 hours. Other side effects of etomidate include vomiting and myoclonus; therefore, it is important to administer a benzodiazepine before giving etomidate and to ensure that the patient is well sedated.

CASE 1

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- At the first manipulation, strong sympathetic response (HR jumps from 101 to 186, RR from 12 to 36)
- Fentanyl bolus 2 µg/kg IV (repeated 4 times at intervals of 1 min) and CRI to 20 µg/kg/h
- HR increase further, very superficial breathing, patient waking up (treated with Alfaxalone 0.5 mg/kg IV)

- Question: how can we treat that?

CASE 1

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- Why doesn't the patient remain unconscious?
  - Is it just to less inhalants?
  - Is there to less analgesia?
  - Is there some Hyperalgesia yet?
- Isoflurane/Sevoflurane
  - No analgesic effect
  - Profound respiratory depressant - as anesthetic dose is increases - both tidal volume and respiratory rate decrease
  - Increases in depth of anesthesia produce corresponding decreases in blood pressure – vasodilation
  - MAC Isoflurane dogs: 1.28 +/- 0.06%, cats: 1.63 +/- 0.02%

CASE 1

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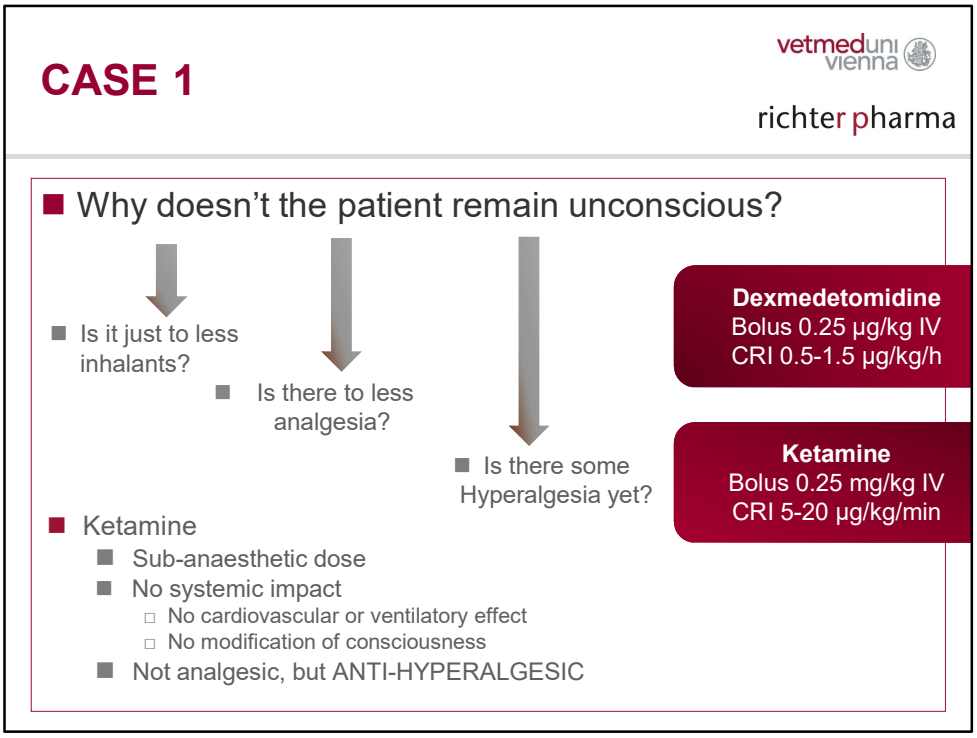
- Why doesn't the patient remain unconscious?
  - Is it just to less inhalants?
  - Is there to less analgesia?
  - Is there some Hyperalgesia yet?

Dexmedetomidine
Bolus 0.25 µg/kg IV
CRI 0.5-1.5 µg/kg/h

- Alpha-2 Agonist
  - Very strong analgesia
  - Short duration
  - Decreased dose requirements of other anesthetics
  - High cardio-circulatory impact (bradycardia, hypertension)


CASE 1
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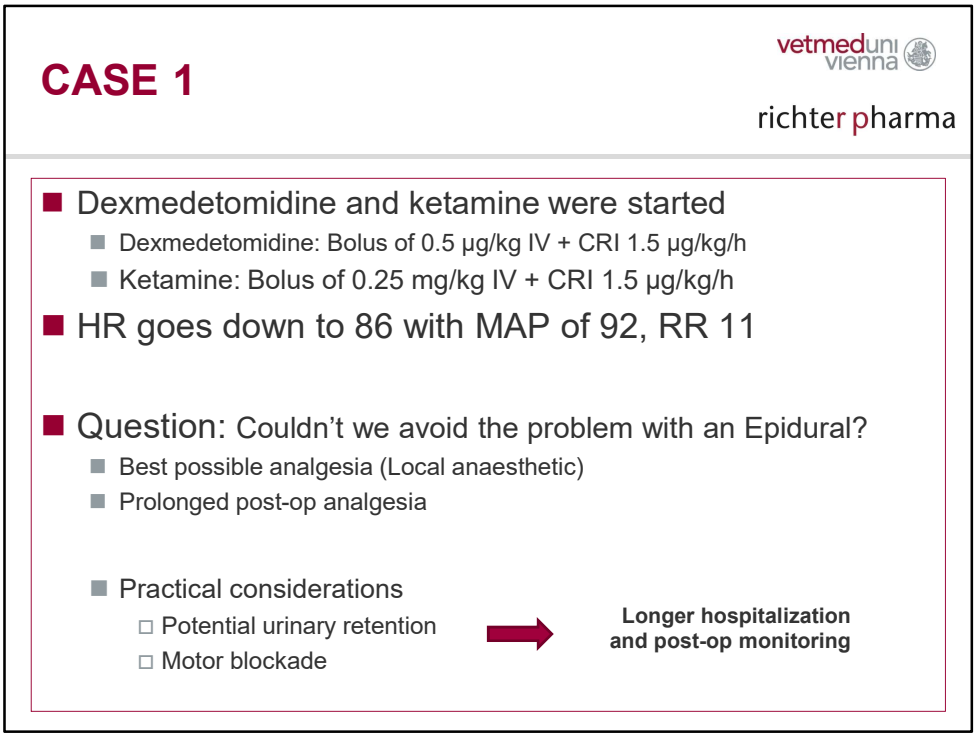
Why doesn't the patient remain unconscious?
Is it just to less inhalants?
Is there to less analgesia?
Is there some Hyperalgesia yet?
Dexmedetomidine
Bolus 0.25 µg/kg IV
CRI 0.5-1.5 µg/kg/h
Ketamine
Bolus 0.25 mg/kg IV
CRI 5-20 µg/kg/min
Ketamine
Sub-anaesthetic dose
No systemic impact
No cardiovascular or ventilatory effect
No modification of consciousness
Not analgesic, but ANTI-HYPERALGESIC



Couldn't we avoid the problem with an Epidural?

- Best possible analgesia (Local anaesthetic)
- Prolonged post-op analgesia

- General contraindication
  - Coagulation problems
  - Loss of anatomical references
  - Skin infection
  - Shock, if vasodilatative drugs are to be used

- Practical considerations

Potential urinary retention
Motor blockade

**Longer hospitalization
and post-op monitoring**

CASE 1

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- Multimodal post-op analgesia:
  - Buprenorphine 15 µg/kg IV q6h
    - Will take over on Fentanyl – decreases possibility of post-op dysphoria
    - Works differently from a pure mu agonist – less cardiovascular and respiratory depression
    - As effective as Methadon in cats
  - Ketamine CRI 2,5 - 5 µg/kg/min
  - Meloxicam 0.1 mg/kg IV once daily

Buprenorphine
10-30 µg/kg IV, IM, PO(cat)
q6h

Picture: Richter-Pharma

CASE 1

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- But Sam does not look comfortable 4 hours after end of surgery

Video: Dr. Stephanie von Ritgen, Veterinary University Vienna

CASE 1

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- Question: what´s the problem? Why is our multimodal analgesia insufficient?

HYPERALGESIA

- Gabapentin
  - Regulates presynaptic Ca+ release
  - Blocks postsynaptic Ca+ Channels
  - It is ANTI-HYPER-ALGESIC

Gabapentin
5-10 mg/kg q12-24h (cat)
5-10 mg/kg q6-12h (dog)

CASE 1

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CASE 1

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- 12 hrs later …

Video: Dr. Stephanie von Ritgen, Veterinary University Vienna

CASE 2

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- Humphrey, 2,5 y, neutered male, Husky
  - Stop eating and started vomiting 36 hours ago
  - Abdominal ultrasound suggests linear foreign body
  - Received infusion of ringers lactate and maropitant
  - Scheduled for exploratory laparotomy
  - Planned duration 2 hrs

Picture: Vetmediathek, Veterinary University Vienna

CASE 2

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- ASA-Status: 3
- **Premedication:** Methadone 0.25 mg/kg IV
- **Induction:** Midazolam-Propofol (0.2 mg/kg + 4.5 mg/kg IV)
- **Maintenance:** Isoflurane 1-1,2% + 10 min before skin incision - Fentanyl bolus 2 µg/kg + CRI 10 µg/kg/h

Methadone
0.1-0.5 mg/kg IV

Picture: Richter-Pharma

CASE 2

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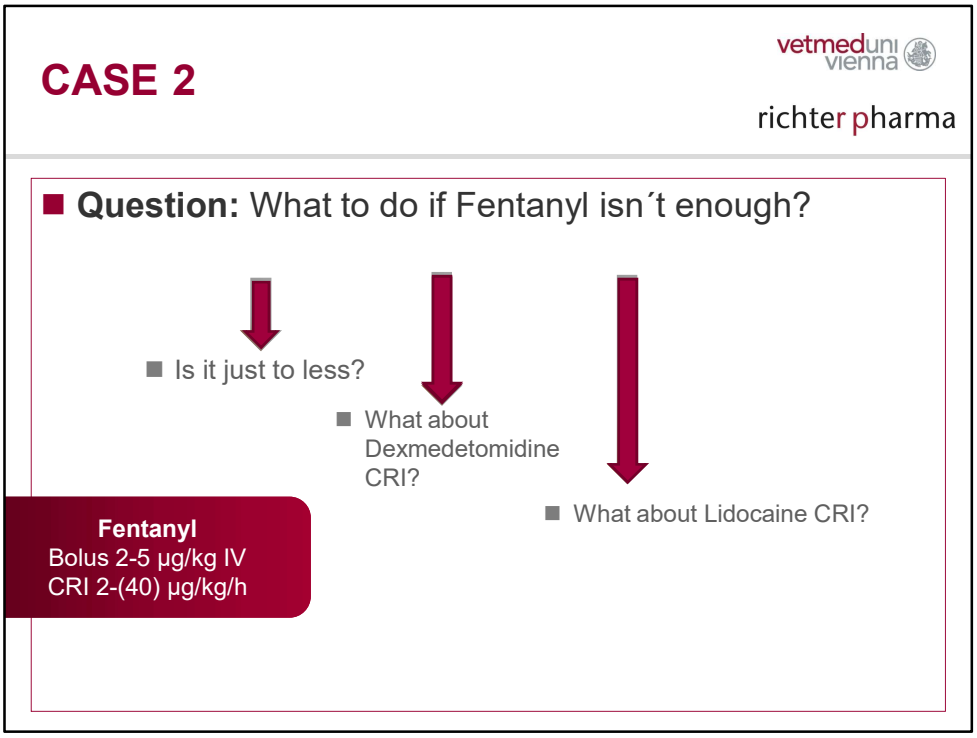
- There is peritonitis and some parts of intestinal tract are congested
  - Palpation around congested areas evokes strong sympathetic response

Picture: Vetmediathek, Veterinary University Vienna

As you could see in the picture – heart rates pretty high, breathing pattern increased, lid reflexes more pronounced, jaw tone increased

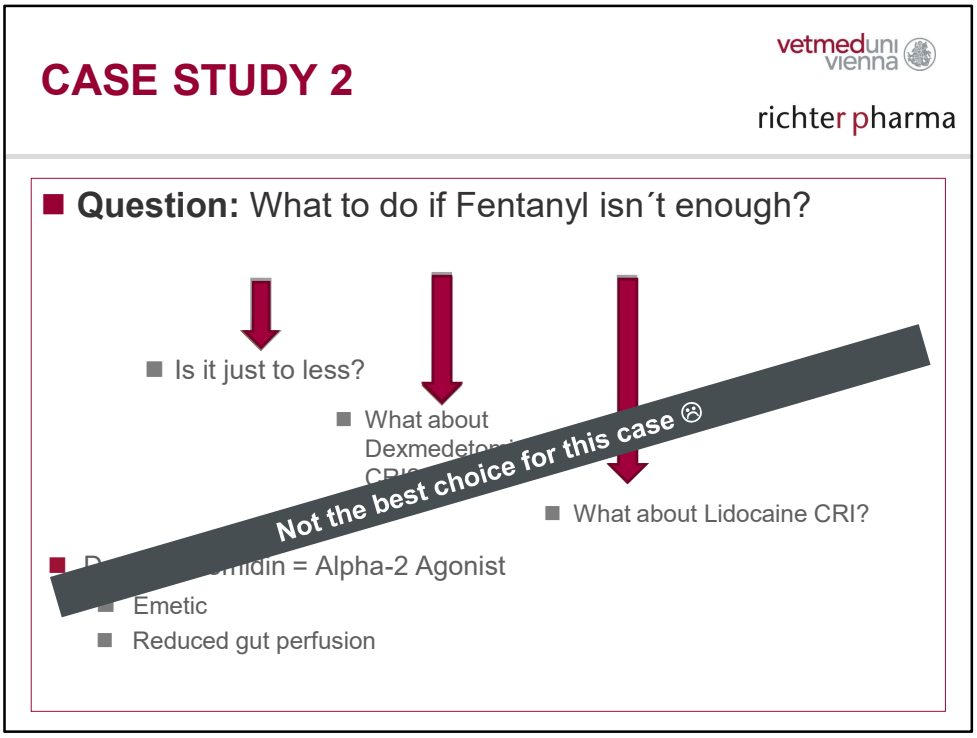

CASE 2
vetmeduni vienna
richter pharma
Question: What to do if Fentanyl isn´t enough?
Is it just to less?
What about Dexmedetomidine CRI?
What about Lidocaine CRI?
Fentanyl
Bolus 2-5 µg/kg IV
CRI 2-(40) µg/kg/h

CASE 2

vetmeduni vienna

richter pharma

- **Question:** What to do if Fentanyl isn´t enough?
  - Is it just to less?
  - What about Dexmedetomidine CRI?
  - What about Lidocaine CRI?
- Dexmedetomidine = Alpha-2 Agonist
  - Emetic
  - Reduced gut perfusion


CASE STUDY 2
vetmeduni vienna
richter pharma
Question: What to do if Fentanyl isn´t enough?
Is it just to less?
What about Dexmedetomidin CRI?
What about Lidocaine CRI?
Not the best choice for this case ☹
midin = Alpha-2 Agonist
Emetic
Reduced gut perfusion

CASE 2

vetmeduni vienna

richter pharma

- **Question:** What to do if Fentanyl isn´t enough?
  - Is it just to less?
  - What about Dexmedetomidine CRI?
  - What about Lidocaine CRI?

Lidocaine
Bolus 1-1.5 mg/kg IV slow
CRI 30-50 µg/kg/min

- Lidocaine
  - Analgesia
  - Anti-inflammatory
  - Anti-toxic (membrane stabilizer)
  - Prokinetic
    - **Not** before the foreign body is removed

CASE 2

vetmeduni vienna

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- Fentanyl bolus 2 µg/kg IV (repeated 2 times at intervals of 2 min) and CRI to 20 µg/kg/h
  - Still some responses at manipulation but minimal (<10% over base line)
- As the foreign body is removed, Lidocaine bolus 1 mg/kg IV and then CRI 50 µg/kg/min is started

Picture: Richter-Pharma

CASE 2

vetmeduni vienna

richter pharma

- Multi-modal post-operative pain management:
  - Methadone 0.2 mg/kg IV at end of surgery
  - Lidocaine CRI continued with 30 µg/kg/min
- In the next 2 hrs, Humphrey shows increasing signs of break through pain (hypersalivation, refuses to lay on the abdomen, vocalization)
  - Methadone is topped up with 0.1 mg/kg IV
  - Lidocaine CRI increased to 50 µg/kg/min

Picture: Vetmediathek, Veterinary University Vienna

Differentiation dysphoria vs. pain

CASE 2

vetmeduni vienna

richter pharma

- Minimal improvement
  - Passed to MLK 2 ml/kg/h
    - Morphine 0.25 mg/kg/h + Lidocaine 25 µg/kg/min + Ketamine 10 µg/kg/min

MLK
475 ml NaCl 0.9% + 6 ml Morphin (10 mg/ml) + 18.5 ml Lidocaine (2%) + 1.5 ml Ketamine (100 mg/ml)
CRI 1-2 ml/kg/h

Picture: Dr. Stephanie von Ritgen, Veterinary University Vienna

CASE 2

vetmeduni vienna

richter pharma

- Humphrey is more calm, feeling more comfortable, rest on his side

Picture: Dr. Stephanie von Ritgen, Veterinary University Vienna

TAKE HOME MASSAGE

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- Surgically induced nociception is the primary reason for administering general anesthesia and the primary source of the patient's hemodynamic and stress responses
- If nociceptive control is adequate, the stress responses will be minimized, and sympathetic stability will be achieved
- Suppression of nociceptive transmission has the benefit of decreasing arousal, which in turn reduces doses of anesthetics required to maintain unconsciousness

CONCLUSION

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- Reduction in anesthetic agent doses may facilitate intraoperative patient stability and faster recovery
- To achieve adequate postoperative pain control, multimodal pain management should be continued in the postoperative period and throughout the hospital stay


Questions ?
hank you to Dr. Attilio Rocchi, DECVAA for
ding some of the graphs and pictures!