**Veterinary Clinical Pharmacology Myths**  
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**Morphine cannot be used in cats due to CNS excitement, aka morphine mania.**  
This is false. Morphine is commonly used in cats without producing morphine mania. Morphine mania was termed when CNS excitement was noted after doses of 5-20 mg/kg SC, which are at least 20 times higher than the clinically recommended dose. High doses or rapid administration to any species can result in CNS excitement and even seizures.

**Morphine is slowly metabolized and eliminated in cats.**  
This is false. The pharmacokinetics of morphine in cats demonstrate it is rapidly metabolized and has a short elimination half-life (just over an hour). The myth came about because cats are deficient in some glucuronide conjugations enzymes which metabolize morphine in most species. However cats rapidly metabolize morphine through a different pathway, sulfate conjugation and as a result morphine has a short half-life in cats.

**Morphine cannot be administered IV to dogs due to histamine release and severe hypotension.**  
This myth is false. Morphine can be administered IV to dogs, resulting in some histamine release, but hypotension does not occur at clinically relevant doses. The high end of clinically relevant IV morphine doses are 0.5 mg/kg as a bolus. Higher doses can cause more profound histamine release, but marked hypotension does not occur until around 3 mg/kg IV bolus in dogs. However the effect of IV morphine on histamine release and cardiovascular status have not been investigated in dogs with mast cell tumors. Other IV opioids may be better choices for dogs with mast cell tumors until further data are available.

**Morphine and other opioids cause cardiovascular and respiratory depression in animals**

This is partially true, but rarely clinically relevant. Morphine and other opioids at clinically recommended doses have minimal detrimental effects on cardiovascular function in animals. Even massive overdoses have minor cardiovascular effects in healthy animals.

Although opioids do cause dose dependent respiratory depression, the magnitude of the depression is small and plateaus at relatively minimal respiratory depression. If substantial respiratory depression occurs it is often due to other factors contributing such as other drugs (e.g. inhalant or injectable anesthetics), concurrent disease (pulmonary disease) or head trauma in which the respiratory centers are affected. If the animal is at great risk for respiratory depression, which is clinically very rare, than constant rate infusions can be administered. The CRI minimizes peak drug concentrations while maintaining effective concentrations resulting in little to no effect on the respiratory function if appropriate doses are administered.

**Fluoroquinolones are broad spectrum antimicrobials**

This is false. Fluoroquinolones have little to no activity against anaerobes (except pradofloxacin) and very poor activity against Streptococcus species. Therefore most fluoroquinolones are not broad spectrum, but have a limited spectrum that are effective against many gram negative and some gram positive aerobic bacteria.

**Acepromazine lowers the seizure threshold**

This myth appears false because of the way acepromazine in used in veterinary medicine. Phenothioazines administered at high doses for long periods of time decrease the seizure threshold in human psychiatric patients. However acepromazine is used as single doses or for very short periods of time in veterinary medicine, which does not appear to increase the risk of seizures. A retrospective study demonstrated potential anticonvulsant effects of acepromazine or at least no worsening of seizures in 36 dogs with a history of seizure activity.

Disclaimer: The information is accurate to the best of the author’s knowledge. However recommendations change as new data become available and errors are possible. The author recommends double checking the accuracy of all information including dosages.