The following drugs are commonly needed for emergency resuscitation and are kept in the *crash cart.*

Which one of the following choices correctly combines the drug, its mechanism of action, and the effect seen in the patient?

Vasopressin, vasopressin receptor agonist, vasoconstriction	HIDE
Atropine, anti-cholinergic, decreased heart rate	HIDE
Epinephrine, adrenergic agonist, vasodilation	HIDE
Dobutamine, alpha agonist, substantially decreased cardiac output	HIDE
Lidocaine, calcium channel blocker, anti-arrhythmic	HIDE

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Vasopressin (V), also called anti-diuretic hormone (ADH), is released in large amounts from the pituitary gland in times of emergency or great stress, especially when hypotension is present.

It acts on V receptors found on blood vessels (V1) and causes profound vasoconstriction.

V is used during cardiopulmonary cerebral resuscitation to increase blood flow back to the heart via this peripheral vasoconstriction.

During normality, ADH acts at receptors on renal tubules and collecting ducts (V2) to prevent excretion of water when osmolality increases.

So, when dehydrated, ADH helps retain fluid to bring blood volume and osmolality back to normal.

Neurohypophysis:

The neurohypophysis (pars nervosa, posterior lobe) has three anatomic subdivisions. Secretion granules that contain the neurohypophyseal hormones, ie, antidiuretic hormone (ADH, vasopressin) and oxytocin, are synthesized in the hypothalamus but are released into the bloodstream in the pars nervosa. The infundibular stalk joins the pars nervosa to the overlying hypothalamus.

ADH, an octapeptide synthesized in the hypothalamus, is packaged into membrane-limited granules with a corresponding binding protein (neurophysin) and transported to the pars nervosa, where it is released into the circulation. ADH binds to specific receptors in the distal part of the nephron and collecting duct of the kidney; it increases the renal tubular reabsorption of water from the glomerular filtrate.

The output of ADH is directly related to the degree of hydration of the body. Hydration of the body inhibits release of ADH, whereas hydration or injection of hypertonic electrolyte solutions favors release of ADH, which in turn causes increased water resorption from the glomerular filtrate, resulting in dilution and decreased osmolarity of body fluids. Barbiturates, ether, chloroform, morphine, acetylcholine, nicotine, and pain increase ADH release, which leads to less urine formation. Ethanol inhibits ADH release, which leads to diuresis.

The pressor effect of ADH is less prominent than the antidiuretic effect. At a dosage several hundred times larger than the antidiuretic dosage, ADH has a pronounced pressor effect, which may also lead to coronary constriction. The contractile mechanism of the capillaries, as well as GI and uterine muscle, is stimulated, and a prolonged increase in blood pressure follows.

Oxytocin has specific effects on the smooth muscle of the uterus and the myoepithelial cells of the mammary gland. It has no established physiologic function in the male, although an effect on sperm transport has been suggested.

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SAVE & EXIT

PREV NEXT

1	2	3	4	5	6	7	8	9	10

Which drug family is associated with tooth enamel problems?

Aminoglycosides	HIDE
Macrolides	HIDE
Cephalosporins	HIDE
Tetracyclines	HIDE
Quinolones	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this | Lab | Definitions | Report

Correct:

Tetracyclines chelate calcium in teeth and bones; they inhibit calcification (including dental enamel), staining developing teeth and bones yellowish then brownish.

Tetracyclines are also potentially nephrotoxic and are contraindicated (EXCEPT for DOXYcycline) in renal insufficiency.

Quinolones like enrofloxacin (Baytril®) have been associated with damage to articular cartilage in young growing animals and with neurotoxicity (like convulsions) at higher doses.

Aminoglycosides like gentamicin and amikacin are most often associated with NEPHROTOXICITY, ototoxicity, and neuromuscular blockade.

Macrolides like erythromycin do not have many side effects. One particular macrolide, TILMICOSIN (Micotil®), is contraindicated in pigs, and should not be used in an automatically powered syringe because an accidental self-injection can kill humans.

Cephalosporins like cephalexin (Keflex®), ceftriaxone (Rocephin®), and ceftiofur (Naxcel®) are relatively nontoxic, but there may be pain at injection site.

Refs: The Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

1	2	3	4	5	6	7	8	9	10

Which of these side effects is correctly matched to its drug family?

Ototoxicity / chloramphenicol	HIDE
Hemolytic anemia / macrolides (e.g., erythromycin)	HIDE
Gastrointestinal disturbance / lincosamides (e.g., clindamycin)	HIDE
Neuromuscular blockade / sulfonamides (e.g., trimethoprim sulfa)	HIDE
Keratoconjunctivitis sicca / aminoglycosides (e.g., gentamicin)	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this | Lab | Definitions | Report

Correct:

Lincosamides like clindamycin (Antirobe®) may be associated with GI upset. DO NOT use clindamycin in rabbits, guinea pigs, chinchillas, hamsters, horses, and ruminants for this reason. It is CONTRAINDICATED IN HORSES because a severe, even fatal, colitis can occur.

Macrolides like erythromycin do not have many side effects. Remember that one particular macrolide, tilmicosin (Micotil®), is contraindicated in pigs and should not be used in an automatically powered syringe because an accidental injection can kill humans (that would be you, doctor).

Aminoglycosides like gentamicin and amikacin are most often associated with NEPHROTOXICITY, ototoxicity, and neuromuscular blockade.

Aminoglycosides like gentamicin and amikacin are most often associated with NEPHROTOXICITY, ototoxicity, and neuromuscular blockade.

Chloramphenicol has been associated with bone marrow suppression/aplastic anemia in exposed humans and is CONTRAINDICATED IN FOOD ANIMALS.

With sulfonamides like trimethoprim-sulfa (TMS), think keratoconjunctivitis sicca (also type 1 and 3 hypersensitivities, hepatitis, hemolytic anemia, urticaria, hematuria, hypothyroidism, bone marrow depression and thats just in DOGS).

Refs: Plumb's Veterinary Drug Handbook, 7th ed. pp. 309-13 and the Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

1	2	3	4	5	6	7	8	9	10

Which one of the following choices best explains how acepromazine produces sedation?

Stimulates GABA receptors resulting in the release of inhibitory neurotransmitters	HIDE
Agonist at alpha-2 receptors which decreases release of norepinephrine	HIDE
Antagonizes glutamate receptors in the dorsal horn of the spinal cord	HIDE
Inhibits release of acetylcholine at presynaptic receptors	HIDE
Dopamine receptor antagonist in the brain	HIDE

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Correct:

Acepromazine antagonizes dopamine receptors in the brain, most likely in the basal ganglia and the limbic system. Decreased dopamine in these areas produces sedation and muscle relaxation.

Ketamine antagonizes N-methyl-D-aspartate receptors in the spinal cord, decreasing release of glutamate, the major excitatory neurotransmitter in the CNS.

Click here to see an image of a cat sedated with acepromazine. Elevation of the third eyelid is a common.

Acetylcholine release is blocked by neuromuscular blockers such as atracurium and succinyl choline.

The GABA receptor is activated by many anesthetic and sedative drugs, including benzodiazepenes, barbiturates, propofol, and inhalants, but not acepromazine.

Medetomidine, xylazine, and detomidine are alpha-2 receptor agonists; norepinephrine secretion is decreased by this action, which produces profound sedation. Acepromazine also blocks alpha-1 receptors, causing vasodilation peripherally.

Refs: Grimm, Tranquilli, and Lamont's Essentials of SA Anesthesia and Analgesia, 2nd ed., p. 47, Hubbell and Muir's Equine Anesthesia, 2nded., p. 187, Greene's Vet Anesthesia and Pain Mgt Secrets, p. 87-8, Muir et al. Handbook of Vet Anesthesia, 4th ed., pp. 29-34.

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SAVE & EXIT

PREV NEXT

1	2	3	4	5	6	7	8	9	10

Which one of these drug pairings has BOTH drugs contraindicated as injectable antibiotics in pigs?

Tilmicosin , Chloramphenicol	HIDE
Chloramphenicol, Oxytetracycline	HIDE
Lincomycin, Procaine Penicillin G	HIDE
Tilmicosin, Lincomycin	HIDE
Lincomycin, Oxytetracycline	HIDE

BACK NEXT LEAVE BLANK

Correct: **Tilmicosin , Chloramphenicol**

Chloramphenicol has been associated with bone marrow suppression/aplastic anemia in exposed humans, and is CONTRAINDICATED IN FOOD ANIMALS.

Tilmicosin (Micotil®) is a macrolide contraindicated in pigs (injection may kill them. May kill YOU too).

Remember that Tilmicosin should not be used in an automatically powered syringe because an accidental self-injection can be FATAL to humans.

Refs: The Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

1	2	3	4	5	6	7	8	9	10
✓	✗	✓	✓	✓	✗	✗			

Which drugs are phosphodiesterase (PDE) inhibitors?

Enalapril, Benazapril	HIDE
Diltiazem, Verapamil	HIDE
Propranolol, Atenolol	HIDE
Pimobendan, Milrinone	HIDE
Dobutamine, Isoproteronol	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this | Lab | Definitions | Report

Pimobendan and milrinone are phosphodiesterase (PDE) inhibitors, one of three types of positive inotropes.

Positive inotropes increase the cardiac muscular contraction strength by two mechanisms:
1) Increasing intracellular calcium available for contractile proteins.
2) Increasing the sensitivity of contractile proteins to calcium.

Pimobendan is an orally administered "inodilator" (has both inotropic and vasodilator effects) approved for treatment of congestive heart failure due to dilated cardiomyopathy and degenerative valvular disease in dogs.
It is not approved in cats, but preliminary work suggests it may be safe and effective.

Milrinone is intravenously administered and generally reserved for patients in severe heart failure.

The three classes of positive inotropes are the phosphodiesterase inhibitors and:
Beta adrenergic agonists (dopamine, dobutamine, isoproterenol and epinephrine)
Cardiac glycosides (digoxin, digitoxin)

Verapamil and diltiazem are calcium channel blockers with antiarrhythmic and negative inotropic effects (decrease force of cardiac muscle contraction).

They are used to treat atrial fibrillation and supraventricular tachycardias, as well as hypertrophic cardiomyopathy (HCM) and hypertension.

ACE inhibitors like enalapril are vasodilators that help increase cardiac output.

Beta-blockers like propranolol and atenolol are used to treat arrhythmias, systemic hypertension, and HCM.

Refs: Plumb's Vet Drug Handbook, 8th ed. online, and the Merck Veterinary Manual

Heart Failure

By **Mark D. Kittleson, DVM, PhD, DACVIM (Cardiology), Professor Emeritus, School of Veterinary Medicine, University of California, Davis**

Heart failure is a clinical syndrome that occurs secondary to severe, overwhelming cardiac disease. It occurs because the heart is no longer able to maintain normal venous/capillary pressures, cardiac output, and/or systemic blood pressure. It is most commonly caused by a chronic disease that results in a severe decrease in myocardial contractility, severe regurgitation or shunting, or severe diastolic dysfunction. However, it is common to have all three abnormalities present simultaneously (but with one predominating). By far, the most common clinical manifestations seen with heart failure are directly due to edema and effusion (congestive or backward heart failure). Much less commonly, animals present because of signs referable to a decrease in cardiac output (forward heart failure). Very rarely, they present in cardiogenic shock (low blood pressure due to decreased cardiac output). This occurs because the cardiovascular system operates under a system of priorities. Its three primary functions are to maintain a normal blood pressure and normal cardiac output, both at a normal venous/capillary pressure. When the system is overwhelmed, it allows venous/capillary pressure to increase first (and so allows edema or effusion to form) and then allows cardiac output to fall. Only after cardiac output has fallen remarkably does cardiogenic shock occur. In acute heart failure, before any compensation has occurred, cardiogenic shock may predominate, but even in this situation, acute chordal rupture is the most common cause of acute heart failure in animals and results in an increased left atrial pressure and thus pulmonary edema.

Initial changes in cardiac chamber dimension (volume) or wall thickness that occur are best understood in relation to preload (the tension imposed by venous return on the ventricular walls at end-diastole) and afterload (the tension imposed on the ventricular walls at end-systole). Alterations in preload or afterload may be caused by structural cardiac abnormalities, systemic compensatory mechanisms, or both. Volume overload states, such as those that occur with chronic valvular disease/valvular insufficiencies, patent ductus arteriosus, atrial or ventricular septal defects, peripheral left-to-right shunts, anemia, or hyperthyroidism, cause an increase in preload that leads to ventricular growth and chamber enlargement (euphemistically called dilation) via eccentric myocyte hypertrophy. Pressure overload states, such as those that occur with pulmonary or systemic hypertension, and pulmonic or aortic stenosis, cause an increase in afterload (systolic intraventricular pressure) that leads to ventricular wall thickening via concentric hypertrophy. Neither volume nor pressure overload is synonymous with heart failure; either state may result in heart failure, depending on the severity of the overload and the degree of compensation.

PREV NEXT

1 2 3 4 5 6 7 8 9 10

A five-year old neutered male cat is presented with a chronic intermittent history of straining to defecate.

On physical exam, the cat has a hard tubular density in the caudal abdomen. Subsequent work-up diagnoses megacolon.

After acute care to clear the constipation, which one of the following choices is most appropriate for long-term treatment?

Lincomycin	HIDE
Lactulose	HIDE
Lomustine	HIDE
Loperamide	HIDE
Lomotil	HIDE

BACK NEXT LEAVE BLANK

Correct:

Lactulose is a disaccharide given daily with osmotic effects in the colon, causing colonic hydration and facilitating stool evacuation for a case of megacolon.

Lomotil and loperamide are opiate antidiarrheals which inhibit GI motility and are contraindicated in megacolon cases.

Lincomycin is an antibiotic.

Lomustine (CCNU) is a chemotherapeutic agent used to treat mast cell disease, CNS disease or as a rescue agent for lymphosarcoma.

Refs: Cote, Clinical Veterinary Advisor-Dogs and Cats, 3rd ed. pp. 221-2, Plumb's Vet Drug Handbook, 7th ed. pp. 781-2 and the Merck Veterinary Manual online.

Constipation and Obstipation in Small Animals

By **Alice Defarges, DVM, MSc, DACVIM, Assistant Professor in Internal Medicine, Ontario Veterinary College, University of Guelph**

Constipation is the infrequent or difficult evacuation of feces, which are typically dry and hard. Constipation is a common clinical problem in small animals. In most instances, the problem is easily rectified; however, in more debilitated animals, accompanying clinical signs can be severe. As feces remain in the colon longer, they become drier, harder, and more difficult to pass. Obstipation is intractable constipation characterized by an inability to evacuate the mass of dry, hard feces; impaction extending from the rectum to the ileocolic valve can result. Megacolon is a pathologic condition of hypomotility and dilation of the large intestine that results in constipation and obstipation.

Etiology and Pathophysiology:

Peristaltic waves are responsible for the aboral movement of fecal material in the colon. Giant migrating waves that occur intermittently throughout the day move this matter farther and more rapidly. These waves constitute the “gastrocolic reflex” and are common after ingestion of a meal. A reduction or loss of these waves may contribute to constipation. Similarly, an increase in segmentation wave activity may predispose to constipation. However, diet is the most important local factor affecting colonic function.

Chronic constipation may be due to intraluminal, extraluminal, or intrinsic (ie, neuromuscular) factors. Intraluminal obstruction is most common and is due to the inability to pass poorly digested, often firm matter (eg, hair, bones, litter) mixed with fecal material. The lack of water intake or the reluctance to defecate on a regular basis because of environmental (eg, stress) or behavioral (eg, dirty litter box) factors or painful anorectal disease predisposes to formation of hard, dry feces. Intraluminal tumors may also impede the passage of feces. Extraluminal obstruction may be caused by compression of the colon or rectum from a narrowed pelvic inlet after suboptimal healing of pelvic fractures or from enlarged sublumbar lymph nodes or prostate gland. Colonic stricture due to trauma or neoplasia should also be considered. Finally, some animals (usually cats) with chronic constipation or obstipation may have megacolon, likely caused by a lesion of the neuromuscular bed of the colon. The etiology of megacolon often remains undiagnosed. Other diseases that affect neuromuscular control of the colon and rectum include hypothyroidism, dysautonomia, and lesions of the spinal cord (eg, Manx sacral spinal cord deformity) or pelvic nerves. Hypokalemia and hypercalcemia also adversely affect muscular control. Some drugs (eg, opioids, diuretics, antihistamines, anticholinergic

agents, sucralfate, aluminum hydroxide, potassium bromide, and calcium channel-blocking agents) promote constipation via differing mechanisms.

Clinical Findings:

The classic clinical signs of constipation are tenesmus and the passage of firm, dry feces. If the passage of feces is hindered by an enlarged prostate or sublumbar lymph nodes, the feces may appear thin or "ribbon-like" in appearance. Abdominal palpation and rectal examination can confirm the presence of large volumes of retained fecal matter. Passed feces are often putrid. Some animals are quite ill and also have lethargy, depression, anorexia, vomiting (especially cats), and abdominal discomfort.

Diagnosis:

A history of dietary indiscretion and physical evidence of retained feces confirms the diagnosis. Detailed information regarding the duration of constipation and influencing factors may help determine the cause, as will a history of ingestion of indigestible material that may increase fecal bulk or cause pain that can terminate the defecation reflex. Other historical factors that may be relevant include recent surgery, previous pelvic trauma, and possibly radiation therapy. A complete neurologic examination with special emphasis on caudal spinal cord function should be performed to identify neurologic causes of constipation, eg, spinal cord injury, pelvic nerve trauma, and Manx sacral spinal cord deformity.

Abdominal palpation and rectal examination, including evaluation of the prostate and sublumbar lymph nodes, should be performed to determine the presence of perineal hernia, foreign material, pain, or masses. Plain abdominal radiographs may help establish the inciting factor(s) of fecal retention and give some indication of what the feces contain (eg, bones). A barium enema, ultrasonography, or colonoscopy may facilitate demonstration of obstructive lesions or predisposing causes of chronic constipation.

A CBC, biochemical profile including a serum T_4 level, urinalysis, and detailed neurologic examination should be completed in cases of chronic or recurring constipation.

Treatment and Control:

Affected animals should be adequately hydrated. Mild constipation can often be treated by dietary adjustment consisting of avoidance of dietary indiscretion, ready access to water and high-fiber diets, and use of suppository laxatives. Continued or longterm use of laxatives should be discouraged unless absolutely necessary to avoid constipation.

A number of pediatric rectal suppositories are available for management of mild constipation. They include dioctyl sodium sulfosuccinate (DSS; emollient laxative), glycerin (lubricant laxative), and bisacodyl (stimulant laxative). The use of suppositories requires a compliant pet and a willing owner. Suppositories can be used alone or in conjunction with oral laxative therapy.

Mild to moderate or recurrent episodes of constipation may require administration of enemas or manual extraction of impacted feces, or both. Types of enemas include warm tap water (5–10 mL/kg), warm isotonic saline (5–10 mL/kg) with or without a mild soap to act as an irritant, DSS (5–10 mL/cat), mineral oil (5–10 mL/cat), or lactulose (5–10 mL/cat). Enema solutions should be administered slowly with a 10–12 French rubber catheter or feeding tube. Phosphate-containing enemas must be avoided in cats.

If enemas are unsuccessful, manual extraction of impacted feces may be needed. After adequate rehydration, the animal should be anesthetized with an endotracheal tube in place to prevent aspiration in case the colonic manipulation induces vomiting. Complete removal of all feces may require 2–3 attempts over as many days. Concurrent fluid and electrolyte abnormalities should also be corrected.

Laxatives are classified as bulk-forming, lubricant, emollient, osmotic, or stimulant types. Most act on fluid transport mechanisms and colonic motor stimulation. They should be avoided in the presence of dehydration. Bulk-forming laxatives are added to the diet. These products are dietary fiber supplements of poorly digestible polysaccharides and celluloses derived principally from cereal grains, wheat bran, and psyllium. They absorb water, soften feces, add bulk, stretch the colonic smooth muscle, and improve contractility. Many constipated cats respond to dietary supplementation with one of these products. Dietary fiber is preferable because it is well tolerated, more effective, and more physiologic than other laxatives. Commercial fiber-supplemented diets are available, or the pet owner may add psyllium (1–4 tsp/meal), wheat bran (1–2 tbsp/meal), or pumpkin (1–4 tbsp/meal) to canned food. Animals should be well hydrated before starting fiber supplementation to minimize the potential for impaction of fiber in the constipated colon.

Emollient laxatives are anionic detergents that increase the miscibility of water and lipids in digesta, thereby enhancing lipid absorption and impairing water absorption. DSS and disoctyl calcium sulfosuccinate are emollient laxatives available in oral and enema form. Docusate sodium (cats: 50-mg capsule/day; dogs: 50-mg capsule, 1–4/day) and docusate calcium (cats: 50-mg capsule, 1–2/day; dogs: 50-mg capsule, 2–3/day) are other examples of emollient laxatives.

Mineral oil and white petroleum are lubricant laxatives that impede colonic water absorption and permit greater ease of fecal passage. These effects are moderate, and lubricant laxatives are beneficial only in mild cases of constipation. Mineral oil use should be limited to rectal administration because of the risk of aspiration pneumonia with oral administration.

Hyperosmotic laxatives consist of poorly absorbed polysaccharides (eg, lactulose, 0.5 mL/kg, PO, bid-tid), magnesium salts (eg, magnesium citrate, magnesium hydroxide, magnesium sulfate), and the polyethylene glycols. Lactulose is the most effective agent of this group. The organic acids produced from lactulose fermentation stimulate colonic fluid secretion and propulsive motility. Lactulose osmotically retains water in the bowel to soften fecal material. It is also useful in management of hepatic encephalopathy because it decreases luminal pH, reduces bacterial production of ammonia, and favors formation of ammonium ions that are poorly absorbed. Stimulant laxative products (eg, bisacodyl [cats and small dogs: 5 mg; medium-sized dogs: 10 mg; large dogs: 15–20 mg]) increase the propulsive activity of the bowel. They are contraindicated in the presence of bowel obstruction.

Colonic prokinetic agents (eg, cisapride) enhance colonic propulsive motility by activating colonic smooth muscle 5-hydroxytryptamine-2A receptors in a number of species. Anecdotal experience suggest that cisapride (0.1–0.5 mg/kg, PO, bid-tid) effectively stimulates colonic propulsive motility in cats with mild to moderate idiopathic constipation. Higher dosages (up to 1 mg/kg) may be necessary in cats with moderate to severe constipation. No significant adverse effects have been reported in cats treated with cisapride at dosages of 0.1–1 mg/kg, PO, bid-tid). Cats with longstanding obstipation and megacolon are not likely to improve with cisapride therapy.

Ranitidine and nizatidine, H_2-receptor antagonists, are reported to stimulate colonic motility by inhibiting acetylcholinesterase. They stimulate motility by increasing the amount of acetylcholine available to bind smooth muscle muscarinic cholinergic receptors.

To prevent recurrence, high-fiber diets are recommended, ready access to water should be maintained, and frequent opportunities to defecate allowed.

Cases of simple intraluminal obstruction due to dietary indiscretion respond well to bowel evacuation and prevention of this habit in the future. Chronic constipation unresponsive to medical management (eg, some cats with megacolon) may respond to subtotal or total colectomy. Colectomy with colocolonic, ileocolonic, or jejunocolonic anastomosis may be performed depending on the extent of the disease. Mild to moderate diarrhea may occasionally persist for weeks to months after surgery, and some cats may have recurrent constipation. Pelvic osteotomy without colectomy has been recommended for cats with pelvic fracture malunion and hypertrophic megacolon of <6 mo duration. In such cases, pathologic hypertrophy may be reversible with early pelvic osteotomy. Subtotal colectomy is recommended in cats with pelvic fractures if hypertrophy and clinical signs have persisted for >6 mo. In these cases, hypertrophy is followed by muscular degeneration and pathologic dilatation, and pelvic osteotomy alone will not provide relief from obstipation.

1 ✓	2 ✗	3 ✓	4 ✓	5 ✓	6 ✗	7 ✗	8 ✓	9 ✓	10

Which of the following lists of drugs are all normally found in the *crash cart* for use with emergency patients?

Pentoxyfilene, polymixin B, amikacin, meloxicam	HIDE
Penicillin, enrofloxacin, saline, heparin	HIDE
Epinephrine, atropine, dobutamine, vasopressin	HIDE
Isoflurane, sevoflurane, desflurane, methoxyflurane	HIDE
Glycopyrrolate, catheters, morphine, ketamine	HIDE

Correct

Epinephrine, atropine, dobutamine, and vasopressin are used in the treatment of emergency patients, especially those in cardiac arrest or in shock.

Endotracheal tubes, intravenous catheters and fluids, syringes and needles, clippers, tape, and other supplies needed for emergency therapy are kept in the crash cart.

Oxygen, an electrocardiogram machine, and a defibrillator should be close by. A chart with drug dosages and emergency protocols should be posted on the wall in full view.

Morphine and ketamine are controlled substances that must be kept in a double locked box. Antibiotics are needed for some emergency patients but are not usually kept in the crash cart.

Refs: McCurnin's Clin Textbk for Vet Techs, 8th ed. pp. 915, 925 and the Merck

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SAVE & EXIT

PREV NEXT

11	12 M	13	14	15	16	17	18	19	20

Which one of the following choices is a competitive antagonist for aldosterone that is sometimes used in treatment of congestive heart failure?

Spironolactone	HIDE
Furosemide	HIDE
Mannitol	HIDE
Acetazolamide	HIDE
Hydrochlorothiazide	HIDE

BACK NEXT

Correct:

Potassium-sparing diuretics (ie. spironolactone) are aldosterone antagonists sometimes used with furosemide as part of the treatment plan for congestive heart failure (CHF).

Aldosterone, the mineralocorticoid produced in the adrenal cortex, acts on the distal convoluted tubules (and collecting ducts) of the nephron to retain Na+ and water, secrete K+ and increase blood pressure.

With CHF, aldosterone levels are **increased** due to activation of the renin-angiotensin-aldosterone system.

In contrast, see **decreased** aldosterone secretion (and decreased blood pressure) with hypoadrenocorticism.

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SAVE & EXIT

PREV NEXT

11	12 M	13	14	15	16	17	18	19	20

What is the mechanism of action of the GI toxic effects of NSAIDs such as phenylbutazone in horses, and what other adverse effect is classically seen?

Suppresses leukotriene formation; urticaria	HIDE
COX-1 inhibition; renal necrosis	HIDE
Blocking PGE_2; thrombocytopenia	HIDE
Local irritation; esophageal stricture	HIDE
Stimulates cytochrome P450; hepatic necrosis	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: COX-1 inh ibition; renal necrosis

With NSAIDs such as phenylbutazone, think GI ulceration (gastric, right dorsal colon) and acute renal disease/renal papillary necrosis due to COX-1 inhibition. While typically these adverse effects are dose-related, or associated with administration in dehydrated horses, they can be idiosyncratic.

COX-1 is constitutively expressed in almost all tissues to catalyze production of prostaglandins that are involved in normal physiological functions (e.g., GI mucosal protection, renal protection, hemostasis). COX-2 is expressed in inflammation/tissue damage and mediates production of inducible pro-inflammatory prostaglandins (those that cause fever, pain, inflammation).

The COX-1 inhibition prevents TXA2 (a potent aggregating agent) by platelets, which can lead to platelet dysfunction.

The COX-1 inhibition prevents TXA2 (a potent aggregating agent) by platelets, which can lead to platelet dysfunction.

There are reports of idiosyncratic liver dysfunction or failure associated with phenylbutazone administration in horses; can see increased liver enzymes in bloodwork without evidence of liver disease/dysfunction.

Long-term phenylbutazone therapy has been reported to be associated with blood dyscrasias in horses.

Refs: Plumb's Vet Drug Handbook, 7th ed. pp. 486-92, 1089-92, Smith's Large An Med 3rd ed. p. 825, and the Merck Veterinary Manual online edition.

11	12 M	13	14	15	16	17	18	19	20

A 10-year-old, 3-kg, male neutered Chihuahua is presented for dyspnea. The patient has had 2 prior episodes of congestive heart failure (CHF) for which he has been hospitalized and treated.

Thoracic radiographs reveal recurrence of CHF. The owners elect to euthanize but do not wish to be present for the procedure.

Intravenous catheter placement for administration of the euthanasia solution is unsuccessful.

Which one of the following is an acceptable method of euthanasia when there is no intravenous (IV) access?

Subcutaneous administration of a secobarbital	HIDE
Intrathecal injection of ketamine combined with xylazine	HIDE
Intraperitoneal injection of pentobarbital with lidocaine	HIDE
Intramuscular injection of propofol	HIDE
Intrapulmonary injection of phenobarbital	HIDE

BACK NEXT LEAVE BLANK

Correct:

Intraperitoneal injection of pentobarbital combined with lidocaine is an appropriate method of euthanasia if there is no IV access.

IF THE ANIMAL IS ANESTHETIZED PRIOR TO INJECTION, intra-organ (e.g. intra-cardiac, intra-hepatic, intra-renal, intra-splenic, intraosseous) injection with pentobarbital is an acceptable method of euthanasia.

Useful resources on euthanasia:

-AVMA Guidelines for the Euthanasia of Animals: 2013 Edition

-The Merck Veterinary Manual chapter on euthanasia.

Refs: The Merck Veterinary Manual online edition and the AVMA.

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SAVE & EXIT

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11	12 M	13	14	15	16	17	18	19	20

Which one of the following drugs will decrease gastric acid secretion in a dog?

Omeprazole	HIDE
Etodolac	HIDE
Carprofen	HIDE
Meloxicam	HIDE
Deracoxib	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Omeprazole is a proton pump inhibitor that decreases gastric acid secretion. Omeprazole inhibits the sodium/potassium proton pump at the luminal surface of parietal cells. Parietal cells normally secrete hydrogen ions nto the stomach, a key component of acidic HCL.

Other drugs that decrease gastric acid secretion include the H2-receptor antagonists cimetidine, ranitidine, and famotidine and a synthetic prostaglandin E1 analog called Misoprostol.

Carprofen, Etodolac, Deracoxib, Meloxicam and Firocoxib are all nonsteroidal anti-inflammatory drugs (NSAIDS) that may CAUSE gastric acid secretion.

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SAVE & EXIT

PREV NEXT

11	12 M	13	14	15	16	17	18	19	20

Which drug family is most associated with GI disturbances (ie: vomiting, nausea, diarrhea, colitis, colic, etc.)?

None of these	HIDE
All of these	HIDE
Macrolides	HIDE
Sulfonamides	HIDE
Lincosamides	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: **All of these**

Sulfonamides. Lincosamides. Macrolides.

Because they disrupt gut microflora ALL THREE of these families are correct.

Lincosamides are contraindicated in horses because a severe, even fatal colitis can occur.

Macrolides like erythromycin should be used with caution in adult horses because of GI problems. Pigs can also have GI problems with tylosin, another macrolide antibiotic.

Sulfonamides, like trimethoprim sulfa (TMS) are also associated with GI distress at high doses or after oral administration.
Refs: The Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28	29	30

Which drug provides good superficial analgesia but poor visceral analgesia?

Acepromazine	HIDE
Butorphanol	HIDE
Ketamine	HIDE
Xylazine	HIDE
Pentobarbital	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Ketamine is a dissociative general anesthetic that provides good superficial analgesia but poor visceral analgesia.

Remember to put ophthalmic lubricant ointment on CAT eyes to prevent excessive drying of the cornea when using ketamine because CAT eyes stay open on ketamine.

Refs: Plumb's Veterinary Drug Handbook, 7th ed. pp. 762-8 and the Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28	29	30

Which one of the following drugs is contraindicated in the treatment of acute equine laminitis?

Triamcinolone	HIDE
Flunixin meglumine	HIDE
Acepromazine	HIDE
Pentoxifylline	HIDE
Phenylbutazone	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Acute equine laminitis should NOT be treated with corticosteroids like triamcinolone or with ACTH, because these drugs decrease protein synthesis and promote tissue insulin resistance. Hyperinsulinemia can be a primary or contributing factor to laminitis.
Remember, "AVOID the 'ROIDs" in laminitic horses.

Acepromazine is an alpha-adrenergic blocker thought to decrease peripheral vasoconstriction and PROMOTE digital circulation.

Pentoxifylline is a phosphodiesterase inhibitor that has anti-inflammatory effects in the lamina as well as improves flexibility of erythrocytes, thereby improving cirulation.

Phenylbutazone ("bute") and flunixin meglumine (Banamine ®) are classic anti-inflammatory and analgesic meds used to treat acute laminitis in horses.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28	29	30

Phenylpropanolamine is typically used to treat which one of the following conditions?

Urethral sphincter incompetence	HIDE
Canine Cushing's disease (hyperadrenocorticism)	HIDE
Urinary tract irritation	HIDE
Detrusor muscle atony	HIDE
Degenerative myelopathy	HIDE

BACK NEXT



Correct:

Phenylpropanolamine is used to treat urinary incontinence associated with urethral sphincter mechanism incompetence (USMI) in dogs and cats.

Phenylpropanolamine is a sympathomimetic drug. Potential side effects include anxiety, restlessness, hypertension, tachycardia, urinary retention and vomiting.

This drug has been removed from the U.S. market for human use due to an increase risk of strokes. It is still available for veterinary use.

Phenazopyridine is a urinary tract analgesic agent.

Refs: Plumb's Veterinary Drug Handbook, 8th edition, *Phenylpropanolamine* and the Merck Veterinary Manual Online.

Urinary Incontinence

By **Patricia M. Dowling, DVM, MSc, DACVIM, DACVCP, Professor, Veterinary Clinical Pharmacology, Western College of Veterinary Medicine, University of Saskatchewan**

Urinary incontinence is most commonly caused by urethral sphincter incompetence. It is most common in large breed, spayed female dogs (11%–20% incidence) but may be seen in intact females, male dogs, and cats. Estradiol-17β concentrations decrease after ovariohysterectomy in bitches, resulting in deterioration of urethral closure within 3–6 mo. Currently, there are no approved drugs to treat incontinence in animals, and most of the human products traditionally used have been removed from the market because of toxicity concerns. Some estrogen compounds and α-adrenergic drugs may still be available to veterinarians through compounding pharmacies (see Table: Drugs Used to Treat Urinary Incontinence).

Drugs Used to Treat Urinary Incontinence

Drug	Dosage
Diethylstilbestrol	Dogs: 0.1–0.3 mg/kg/day, PO, for 7–10 days, followed by 1 mg/dog/wk
Phenylpropanolamine	Dogs: 1.5–2 mg/kg, PO, once to three times daily
Ephedrine	Dogs: 1.2 mg/kg, PO, bid-tid
	Cats: 2–4 mg/kg, PO, bid-tid
Pseudoephedrine	Dogs >25 kg: 30 mg/dog, PO, tid
	Dogs <25 kg: 15 mg/dog, PO, tid
Testosterone propionate	Dogs: 2.2 mg/kg, IM, every 2–3 days
Testosterone cypionate	Dogs: 2.2 mg/kg, IM, every 30–60 days

Diethylstilbestrol (DES) is a nonsteroidal estrogen derivative that closely resembles the natural estrogen, estradiol. Because it is inexpensive and infrequently administered, it is the first choice to

treat urinary incontinence in female dogs. It is orally bioavailable and reaches peak plasma concentrations in 1 hr in dogs; it has an elimination half-life of 24 hr because of enterohepatic recirculation. Estrogens sensitize the urethral sphincter to α-adrenergic stimulation; therefore, DES therapy is synergistic with α-adrenergic drugs. DES is given as a daily loading dose for 7–10 days and then reduced to once weekly dosing, if possible, to avoid toxicity. Treated dogs are susceptible to bone marrow suppression from estrogen, typified by early thrombocytopenia and potentially fatal aplastic anemia. Hematopoietic toxicity is rarely seen in cats. Other adverse effects seen in dogs include alopecia, cystic ovaries, cystic endometrial hyperplasia, pyometra, prolonged estrus, and infertility. When used once weekly in spayed female dogs, adverse effects from DES are rare.

α-Adrenergic agonists such as phenylpropanolamine (PPA), ephedrine, pseudoephedrine, and phenylephrine act directly on smooth muscle receptors to increase urethral tone and maximal urethral closure pressure. Although often more clinically effective than DES, their action is short lived, usually requiring dosing bid-tid. Of this class of drugs, PPA is the most effective and results in fewer cardiovascular adverse effects. Previously available in over-the-counter cold medications and appetite suppressants, it was withdrawn from the human market because of toxicity associated with overuse as a diet aid. Ephedrine, pseudoephedrine, or phenylephrine may be tried but are less efficacious than PPA. Adverse effects of α-adrenergic drugs include excitability, restlessness, hypertension, and anorexia.

In male dogs, testosterone injections are used to treat urinary incontinence but are generally less effective than estrogen therapy in female dogs.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28	29	30

Ketamine is classified as which one of the following types of anesthetic agents?

Barbiturate	HIDE
Dissociative	HIDE
Synthetic codeine analogue	HIDE
Inhalant	HIDE
Opioid	HIDE

BACK NEXT

Overview | Mark this Question | Lab Values | Definitions

Correct:

Ketamine is a **dissociative anesthetic** - it interrupts communication between areas of the brain that control conscious and unconscious activity.

Analgesia and cataplexy are produced, but muscle relaxation is very poor. Twitching and tremors are significant - ketamine is never used alone.

Sedation and muscle relaxation is most often provided by the addition of acepromazine and an opioid, or an alpha-2 agonist and an opioid as premedication, and a benzodiazepine is usually combined with ketamine for induction.

Other effects of ketamine include sympathetic release that increases heart rate and blood pressure, salivation, increased intraocular pressure, increased intracranial pressure, and bronchodilation.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28	29	30

What adverse effect will azathioprine have on feline patients?

Anorexia	HIDE
Acute hepatic toxicity	HIDE
Bone marrow suppression	HIDE
Renal toxicity	HIDE
Hemorrhagic gastroenteritis	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | | Report a Problem

Correct:

Azathioprine is a chemotherapeutic agent, contraindicated in feline patients due to the likelihood for bone marrow suppression.

Refs: Cote, Clinical Veterinary Advisor-Dogs and Cats, 3rd ed. pp. 62-3, **1515**, Plumb's Vet Drug Handbook, 7th ed. pp. 97-9 and the Merck Veterinary Manual online.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28 M	29	30

Which of the following pharmaceutical agents has been shown to bind to endotoxin, which helps to minimize the effects of endotoxemia in horses?

Flunixin meglumine	HIDE
Lidocaine	HIDE
Pentoxifylline	HIDE
Polymyxin B	HIDE
Doxycycline	HIDE

BACK NEXT

Overview Lab Values

Correct:

Polymyxin B is a polypeptide antibiotic that, at low doses, has been shown to bind endotoxin in horses' circulation, thus ameliorating its effects.

At higher doses polymyxin B can be nephro- and neurotoxic.

Topical applications are common due to low systemic absorption and thus, toxicity.

The other listed medications are used in various situations to combat endotoxemia, but none have the same mechanism of action in binding to endotoxin.

Pentoxifylline is a xanthine-derivative phosphodiesterase inhibitor with anti-tumor necrosis factor (anti-TNF) activity (among other anti-inflammatory and rheologic properties) also used to manage endotoxemic horses.

Flunixin meglumine is a nonsteroidal anti-inflammatory drug (NSAID) used at low doses to combat endotoxemia.

Lidocaine is a local anesthetic that acts as an anti-inflammatory when used as a continuous rate infusion.

Doxycycline is an antimicrobial that also inhibits matrix metalloproteinases.

Refs: Plumb's Veterinary Drug Handbook, 8th ed. and the Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

21	22	23	24	25	26	27	28 M	29	30

Which of the following patients will benefit the most from the use of pre-emptive analgesia?

German shepherd with osteosarcoma of the humerus	HIDE
Dog presented for ovariohysterectomy	HIDE
Cat needing limb amputation after being hit by car	HIDE
Dachshund with intervertebral disk disease	HIDE
Horse with chronic laminitis	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

A dog presented for ovariohysterectomy will benefit the most from the use of pre-emptive analgesia because she is not painful prior to surgery. *Pre-emptive* means to intervene prior to an expected event or consequence.

Treatment prior to beginning of surgery minimizes sensitization of the pain and stress responses, and results in fewer complications and shorter hospital stays.

Analgesics are often combined with sedatives as premedication. This also decreases the stress response and helps make the entire episode of anesthesia and surgery smooth and less or un-eventful.

The pain response is already highly stimulated in all the other patients listed. Chronic pain is present in the german shepherd with osteosarcoma and the horse with laminitis.

These patients will have central sensitization and neuropathic pain as these are seen in chronic pain and with direct damage to the nervous system.

Neuropathic pain is also present in the dachshund since damage to spinal nerves is seen with disk disease.

Click here to see a good summary on pain management, courtesy of the American College of Veterinary Surgeons (ACVS).

Refs: Gaynor & Muir Handbook of Vet Pain Mgt 2nd ed. pp. 57, 351,Tranquilli, Grimm, & Lamont Pain Mgt for the SA Practitioner 2nd ed. p. 10, Greene's Vet Anes & Pain Management Secrets pp. 331-3 and the Merck Veterinary Manual online edition.

Pain Alleviation

By **Sandra Allweiler, DVM, DACVA**

Acute perioperative, traumatic, and disease-related (eg, cancer, pancreatitis, pleuritis, otitis externa) pain is generally treated pharmacologically with one or more analgesics. The optimal drug or drug combinations are determined principally by the anticipated severity of pain, health status, and available drugs for the given species. The more extensive the tissue trauma or disease-induced tissue damage is, the greater the need to use analgesics from more than one drug class (multimodal or balanced analgesia). Multimodal analgesia maximizes the beneficial analgesic effects of multiple drugs through additive or synergistic interactions while minimizing adverse drug effects by lowering the dose of any individual drug.

A perioperative approach to managing surgically induced pain should be used, beginning with the administration of an analgesic before surgery (preemptive analgesia) and continuing with appropriate analgesia throughout the intraoperative period. Three days is a useful guideline for the duration of analgesic therapy after acute surgical pain. Depending on multiple factors (eg, procedure performed, rehabilitation plan, species, breed), some animals require a shorter duration of therapy, whereas other animals require analgesia for longer periods. Aggressive analgesic therapy of several days' duration should be tapered rather than stopped abruptly. As-needed dosing schedules are less effective than scheduled analgesic dosing to treat pain. As-needed protocols require the animal to demonstrate overt pain behaviors to the extent they are recognized by the veterinarian and/or owner. Aggressive prevention and management of acute pain often prevents wind-up of the nociceptive pathways, hastens return to normal function, and decreases the risk of development of chronic pain syndromes.

Minimizing stress and ensuring that overall care and husbandry are in accordance with the needs of the animal improve pain management. Proper housing conditions, nutritional support, and interaction with other animals and/or people should be optimal for the given species and breed. For example, separating a sheep from the flock for pain management may be quite stressful, whereas separating a companion animal from other animals may not be stressful, provided there is sufficient interaction with human caregivers.

Appropriate analgesia after surgery or trauma allows animals to rest. For example, dogs and cats often sleep but should be arousable after surgery if their pain is controlled. The use of pain as a means of restraint (ie, to prevent the animal from injuring a surgical site) is inappropriate; many efficacious chemical and physical restraint modalities are available.

Managing painful and distressed animals requires a combination of good nursing care, nonpharmacologic methods (eg, bandaging, ice packs or heat, physical therapy), and pharmacologic methods. Pharmacologic methods available for the treatment of acute pain may include opioids, NSAIDs, corticosteroids, local anesthetics, α_2-agonists, and N-methyl-D-aspartate (NMDA) receptor

antagonists such as ketamine. Many animals benefit from the management of anxiety. Acepromazine is an effective anxiolytic in small animals but should be used only after appropriate analgesics have been administered. Acepromazine does not have analgesic properties and is not reversible.

ACVS | AMERICAN COLLEGE of VETERINARY SURGEONS

Pain Management

Associated Terms:
Analgesia

The term "ACVS Diplomate" refers to a veterinarian who has been board certified in veterinary surgery. Only veterinarians who have successfully completed the certification requirements of the ACVS are Diplomates of the American College of Veterinary Surgeons and have earned the right to be called specialists in veterinary surgery.

Your ACVS board-certified veterinary surgeon completed a three-year residency program, met specific training and caseload requirements, performed research and had research published. This process was supervised by ACVS Diplomates, ensuring consistency in training and adherence to high standards. After completing the residency program, the individual passed a rigorous examination. Only then did your veterinary surgeon earn the title of ACVS Diplomate.

Language: English / Español

Overview:

"Pain" is a perception that the brain creates from input called "nociception" (pronounced no-si-sep-tion). This is the physiology term to describe the chemical processes that are at work in the body that receive a stimulus, modify it, and transfer it to the brain for interpretation and reaction. The stimulus can be physical, temperature, chemical or inflammatory damage to tissues. The brain processes this nociceptive input, mixes it with other data, and creates the perception we call pain. Everyone's pain perception and reaction to it may be different.

Not all pain is bad—pain lets us know that something may be harming our bodies that we need to stop. But "pathologic pain" is a type of pain that is no longer serving this helpful purpose. Most of the pain issues we are likely to encounter with our pets in the medical situation are examples of pathologic pain.

Pain can be caused by many things:

- physical trauma, such as falling down or being hit by something
- internal organ problems, such as intestinal upset or kidney blockage
- surgical procedures, such as abdominal surgery or bone surgery
- brain or spine problems, such as a slipped disc, pinched nerve or headache
- degenerative changes, such as arthritis and joint damage

Signs and Symptoms:

Common pain behaviors are:

- growling and/or purring (cats)

- not grooming (cats)
- not moving from one spot (cats)
- squinting (cats)
- crying and/or whining (dogs)
- glassy-eyed, vacant look (dogs)
- hunched up body (cats and dogs)
- restlessness and changing positions a lot (dogs)
- shaking and trembling (dogs)
- hiding (cats and dogs)
- irritable or aggressive (cats and dogs)
- no appetite (cats and dogs)
- protecting the hurting body part (cats and dogs)

During and right after a painful injury or other illness, the body responds in several ways; the heart and breathing rate go up, muscles tense, endorphins (natural pain killers) are released internally. But after awhile, other stress hormones are released as the pain continues. This is when the down side of pain starts to outweigh the earlier benefits. The bad effects of pain include:

- no eating or drinking
- poor intestinal function, poor nutrient uptake
- increased risk of infection or delayed wound healing
- poor hygiene and ability to move around
- inability to sleep
- irritable or aggressive behavior, preventing nursing care or therapy

Any one or all of these complications may be present and may risk the recovery of your pet. Sometimes it means your pet does not recover; sometimes it means treatment is more prolonged, intensive, invasive, ineffective or costly. Painful illnesses in a pet's life may make future illness or injuries more difficult to treat. Your pet may have a bad memory of veterinary care he/she received, and be fearful or aggressive the next time they need treatment.

Not all pain is created equal. Some is short-lived (like an injection), some is manageable w/ accommodation (like limping to relieve a sore ankle), and some is incredibly severe (like a broken back that makes a pet bite their owner when being helped). If pain goes away quickly and is minor enough, the negative impact is slight. As the duration and/or severity of pain rises, all of the negative impacts start to add up, and pets need our help.

Treatment:

There are several stages in a pet's medical experience when we can address pain; each stage is an opportunity for providing treatment or "analgesia" (i.e. pain relief) and having a plan for each stage allows us to keep each treatment to a minimum (less risk, less cost).

- "Pre-emptive" analgesia means pain medications are provided before your pet is exposed to painful procedures, like surgery.
- *Physically restrain them;* essentially force them to experience the painful experience. This might be appropriate for a simple injection, for example, one that has a mild and very short duration of pain associated.
- *Make them unconscious at the brain level*, so they don't perceive the pain at that moment. This is usually called general anesthesia. This might be appropriate for diagnostic tests that may hurt while they are being performed, but don't hurt when they are over (an endoscope procedure, for example.)
- *Ongoing pain*; usually this pain starts high and tapers off over a variable timeframe (hours, days, weeks). During these early hours and days when a pet is in the hospital and under 24-hour supervision, we can

continue to strike the pain pathway in multiple locations. This “multimodal analgesia” is more effective and safer for your pet. In hospital treatments might include, for example, IV drips with continuous pain medications, frequent injections of pain medications that work in different ways, and supportive physiotherapy such as optimal bedding, ice pack therapy, and massage.

- *Pain at Home*; once out of the hospital or home from the day-clinic, our options for managing a pet’s pain are narrower, but a multimodal analgesia approach remains the most effective. Multiple oral medications that work in different ways and the same simple physiotherapy techniques can reduce the pain a pet is experiencing well into the at-home period.

We can treat one or more steps in the pain pathway so that your pet does not feel pain while it is being applied, but also so that pain is stopped or minimized after the test or therapy is over. This **combination approach, called balanced anesthesia,** allows us to not only use smaller, safer drug doses, but it appears to be the most effective approach for procedures, such as surgery, that will hurt even after the cutting has stopped.

Additionally, techniques such as acupuncture may be used by trained veterinarians to act as an adjunct to certain acute and chronic pain management protocols. Acupuncture may control pain and inflammation by stimulating the endocrine, nervous and immune systems to activate self-healing. This technique has been shown effective in the pain management of various musculoskeletal and soft tissue injuries.

Pain medications and techniques should be viewed like antibiotics—part of the treatment. Some clinics and hospitals will itemize these charges while others will simply include the therapy as part of advanced anesthesia or hospitalization charges. Different veterinarians will prescribe different medications or use different techniques based on their expertise, experience and/or knowledge. There are current “standard of care” guidelines in the veterinary medical field, but there are no “the best” protocols. A pain management plan must be tailored to your pet, their medical condition, and their pain; charges for these services will vary from patient to patient.

A pain management plan must also be supervised to allow for modifications as your pets response unfolds; the time-frame may be over minutes, hours or weeks depending on the medical condition and pain being treated. Pain is a common component of illness, injury, and medical therapy. It starts with some sort of insult to the body, becomes perceived by the body as the signals travel thru the nervous system, and ultimately alters the physiology and behavior of your pet. It is not a pleasant thing to experience, and if it is severe or prolonged can be quite difficult to tolerate. Exactly when the pain experience becomes true “suffering” is variable from patient to patient, but it can usually be agreed upon that avoiding, preventing or reducing pain is preferred.

The veterinary profession is sufficiently advanced to recognize and successfully manage pain in our patients. We have medications, techniques and experience that can be customized to the species and the medical condition; current standard of care allows for the vast majority of patients to be made comfortable the majority of the time. **Pet owners should feel empowered to be part of the medical decision-making** regarding this, and other, aspects of their pet’s medical care. From the common spay procedure to the complex trauma case, reserve the time for these pain management discussions with your primary care veterinarian or your veterinary surgeon.

This Animal Health Topic was written by and reviewed by Diplomates of the American College of Veterinary Surgeons. Any opinions stated in this article are not necessarily the official position of the American College of Veterinary Surgeons.

The American College of Veterinary Surgeons recommends contacting an ACVS board-certified veterinary surgeon or your general veterinarian for more information about this topic.

To find an ACVS Diplomate, visit www.acvs.org/find-a-surgeon.

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SAVE & EXIT

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Which one of the following medications is associated with extrapyramidal neurologic side effects (movement disorders, aggression)?

Diazepam	HIDE
Metoclopramide	HIDE
S-Adenosyl-Methionine (SAMe)	HIDE
Enrofloxacin	HIDE
Ketamine	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Metoclopramide crosses the blood-brain barrier, where dopamine antagonism at the medullary chemoreceptor trigger zone (CTZ) causes an antiemetic effect.

This dopamine antagonism can also cause adverse extrapyramidal signs, like involuntary muscle spasms, motor restlessness and inappropriate aggression.

The pyramidal and extrapyramidal systems are a complex series of upper motor neurons (UMN) that connect the cerebral cortex to distant body parts and influence muscular tone and control.

The pyramidal system controls skilled muscle movement. The extrapyramidal system helps support the body against gravity and recruits spinal reflexes to initiate voluntary movement.

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SAVE & EXIT

PREV NEXT

31	32	33	34	35	36	37	38	39	40

Which one of the following statements is true regarding the legal utilization of compounded medications?

Compounded medications must undergo significant testing to substantiate drug label claims	HIDE
Lower price is an acceptable reason to prescribe a compounded medication	HIDE
Compounded medications are numerically identified by either a six-digit ANADA (United States) or DIN (Canada)	HIDE
Use of a compounded medication constitutes extra-label drug use	HIDE
The term "compounded" is equivalent to the term "generic"	HIDE

BACK NEXT LEAVE BLANK

Correct:

The use of a compounded medication is an example of extra-label drug use (the use of a drug in a way not in accordance with the label, for example a different dosage, interval, route, or indication).

Compounding medications involves the manipulation of a federally-approved drug by a licensed veterinarian or pharmacist to meet the specific needs of a veterinary patient. This includes adding flavoring, using crushed tablets to prepare a paste or suspension, or mixing different drugs together.

Legal compounding does not typically involve the use of bulk or raw active ingredients. If a federally approved drug is available, it must be used for the compounding process.

In rare situations where no United States Food and Drug Administration (FDA) or Health Canada approved drug is available that would relieve an animal's suffering or prevent death, then compounding is acceptable but this is a "last resort." Legal compounding is not a way to avoid the drug approval process or market drugs for a cheaper price.

Generic drugs are not the same as compounded drugs. Generic drugs are approved by the FDA or Health Canada, meaning that their safety and efficacy has been proven by testing to be equivalent to that of the pioneer drug product. Compounded drugs are subject to no federal testing or approval.

Refs: The Merck Veterinary Manual Online. Click here for compounding information from the AVMA and click here for information from the CVMA.

Extra-Label Drug Use, Compounded Drugs, and Generic Drugs

By **Dawn Merton Boothe, DVM, PhD, Professor, Department of Anatomy, Physiology, and Pharmacology, College of Veterinary Medicine, Auburn University**

A **new animal drug (NAD)** is "any drug intended for use in animals other than man…not generally recognized as safe and effective for the use under the conditions prescribed, recommended, or suggested in the labeling of the drug." A drug's label includes the label on the product itself as well as any accompanying material in or on the package, including the package insert.

To use an NAD in a legal manner, veterinarians must adhere to the specifications noted on the label. Otherwise, the drug is being used in an **extra-label** manner. Extra-label drug use, whether actual or intended, occurs when the drug is used in a manner not in accordance with approved label directions. This includes but is not limited to a different dosage, interval, route, indication, or species.

In 1994, Congress passed the Animal Medicinal Drug Use Clarification Act (AMDUCA), which legalized extra-label drug use by veterinarians as long as specific criteria or restrictions are met. Most restrictions are largely applicable to extra-label drug use in food animals. For both food and nonfood animals, a valid **veterinary-client-patient relationship** must exist. For food animals, in the absence of a drug labeled for the intended use, extra-label drug use might involve drugs approved for use in other food animals, approved for use in nonfood animals, or approved for use in people. Restrictions regarding extra-label drug use of nonfood animal and human drugs are progressively restrictive. Extra-label drug use in food animals is permitted only by or under the supervision of a veterinarian, is allowed only for therapeutic purposes (ie, the animal's health is suffering or threatened), is not allowed when the drug is administered in feed, is not permitted if it results in violative food residues or any residues that may present a risk to public health, and is not allowed if specifically prohibited by the FDA.

Drugs specifically prohibited in food animals by the FDA as of May 2015 include chloramphenicol, clenbuterol, diethylstilbestrol, dimetridazole, ipronidazole, other nitroimadazoles, furazolidone, nitrofurazone, sulfonamide drugs in lactating dairy cattle (except for those specifically approved), fluoroquinolones, aglycopeptides (eg, vancomycin), phenylbutazone in female dairy cattle ≥20 mo old, cephalosporins (except cephapirin) in cattle, swine, chickens, or turkeys for disease prevention, and amantadine or neuraminidase inhibitor classes of drugs used to treat influenza A in poultry and ducks.

Use of a **compounded preparation** also constitutes extra-label drug use. However, a major distinction is that a compounded preparation undergoes no regulatory assessment or approval.

Conditions under which compounding is legal also are specified in the AMDUCA. Compounding includes any manipulation of the drug beyond that stipulated on the label. Guidelines regarding the compounding of pharmaceuticals under the direction of a veterinarian are delineated in Compliance Policy Guideline 7125.40. Among the greatest concerns of the FDA regarding compounded products is compounding intended to circumvent the drug approval process, resulting in mass marketing of products that have had little or no quality control to ensure purity, potency, and stability. The FDA considers a compounded product to be an adulterated, ie, unapproved, new animal drug and thus a violation. Conditions under which compounding is not subject to regulatory actions include a legitimate practice (pharmacy or veterinary; includes licensure), operation within the conformity of state law, for pharmacists in response to a prescription, and for veterinarians operating within a valid veterinary-client-patient relationship. Compounding of human drugs and, very occasionally, bulk drugs into appropriate dosage forms may be acceptable in certain circumstances. A legitimate medical need must be identified (eg, health or life of the animal is threatened or suffering may occur). Additionally, there must be no marketed, approved animal or human drug, regardless of whether it is used as labeled or in an extra-label fashion, that may be substituted for the compounded agent. Occasionally, other rare circumstances may be considered. In 2013, Congress passed the Drug Quality and Security Act that, among other things, increases regulation of compounding in human medicine. However, this Act does not cover compounding of animal products, and Congress is examining further actions to more effectively regulate such compounding.

Pharmacists often can dispense an equivalent, less expensive, nonproprietary (generic) drug without prescriber approval. An exception occurs if a state has a mandatory substitution law or if the brand name product is dispensed along with a “dispensed as written” order. **Generic products** must not only contain the same active ingredient as the proprietary drug but also meet bioequivalence standards. Generics may be pharmaceutically equivalent but may not be therapeutically equivalent. Substitutions of generic drugs for proprietary drugs are recommended only for those drugs shown to be therapeutically equivalent. Those human drugs tested by the FDA and found to be therapeutically equivalent are listed in *Approved Drug Products with Therapeutic Equivalence Evaluations*, otherwise known as the “Orange Book.” However, status of a generic drug is relevant only for the approved species and, as such, therapeutic equivalence of a human generic drug does not apply to extra-label use of that drug in an animal.

Although AMDUCA legalizes extra-label drug use in the USA, selected states or other countries may have additional or complementary regulatory or legal restrictions. In all instances, it is important to read carefully the label instructions for use of specific drugs.

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SAVE & EXIT

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Which one of the following choices is the correct mechanism of action of atropine?

Blocks muscarinic cholinergic receptors	HIDE
Activates alpha-2 receptors in the brain and spinal cord	HIDE
Potentiates chloride channels in the dorsal horn	HIDE
Stimulates nicotinic parasympathetic receptors	HIDE
Closes sodium channels on myocardial sympathetic nerves	HIDE

BACK NEXT LEAVE BLANK

Overview | Lab Values | Definitions | Report a Problem

Correct

Atropine is an anticholinergic, parasympatholytic drug. It **blocks the action of acetylcholine at muscarinic cholinergic receptors**.

These receptors are part of the parasymphathetic nervous system. Since the vagal input to the heart is inhibited, the effect of sympathetic tone is no longer opposed and the heart rate increases.

Salivation, tearing, and bronchial secretions are decreased. Intestinal motility is inhibited, the pupils and bronchioles dilate. Mild sedation may be seen, and some causes of vomiting may be reduced.

An anticholinergic is most often used in anesthetized patients to inhibit the vagal effects of some drugs (e.g., opioids (vagal nuclei), inhalants (indirect via decreased sympathetic tone)) and surgical procedures (e.g., manipulation of the globe).

Atropine is also helpful when drugs that encourage salivation are used, especially in animals prone to laryngospasm (eg, ketamine in SA and pigs).

Atropine is recommended for use in very young animals with immature cardiovascular systems - blood pressure is mostly dependent on heart rate.

Refs: Grimm, Tranquilli, and Lamont's Essentials of Anes and Analgesia in SA, 2nd ed. pp 41-3, Muir et al., Handbook of Veterinary Anesthesia, 4th ed., pp. 25-9.

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SAVE & EXIT

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31	32 M	33	34	35	36	37	38	39	40

Which of the following choices is an effect of xylazine in horses?

Anhidrosis (decreased sweating)	HIDE
Tachypnea	HIDE
Anuria	HIDE
Bradycardia	HIDE
Hypoglycemia	HIDE

BACK NEXT

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Xylazine, an alpha-2 agonist, causes bradycardia. Peripheral vasoconstriction is an immediate effect. Also seen with xylazine in horses: hypertension and decreased cardiac output. Respiratory rate and volume decrease somewhat but oxygenation is maintained.

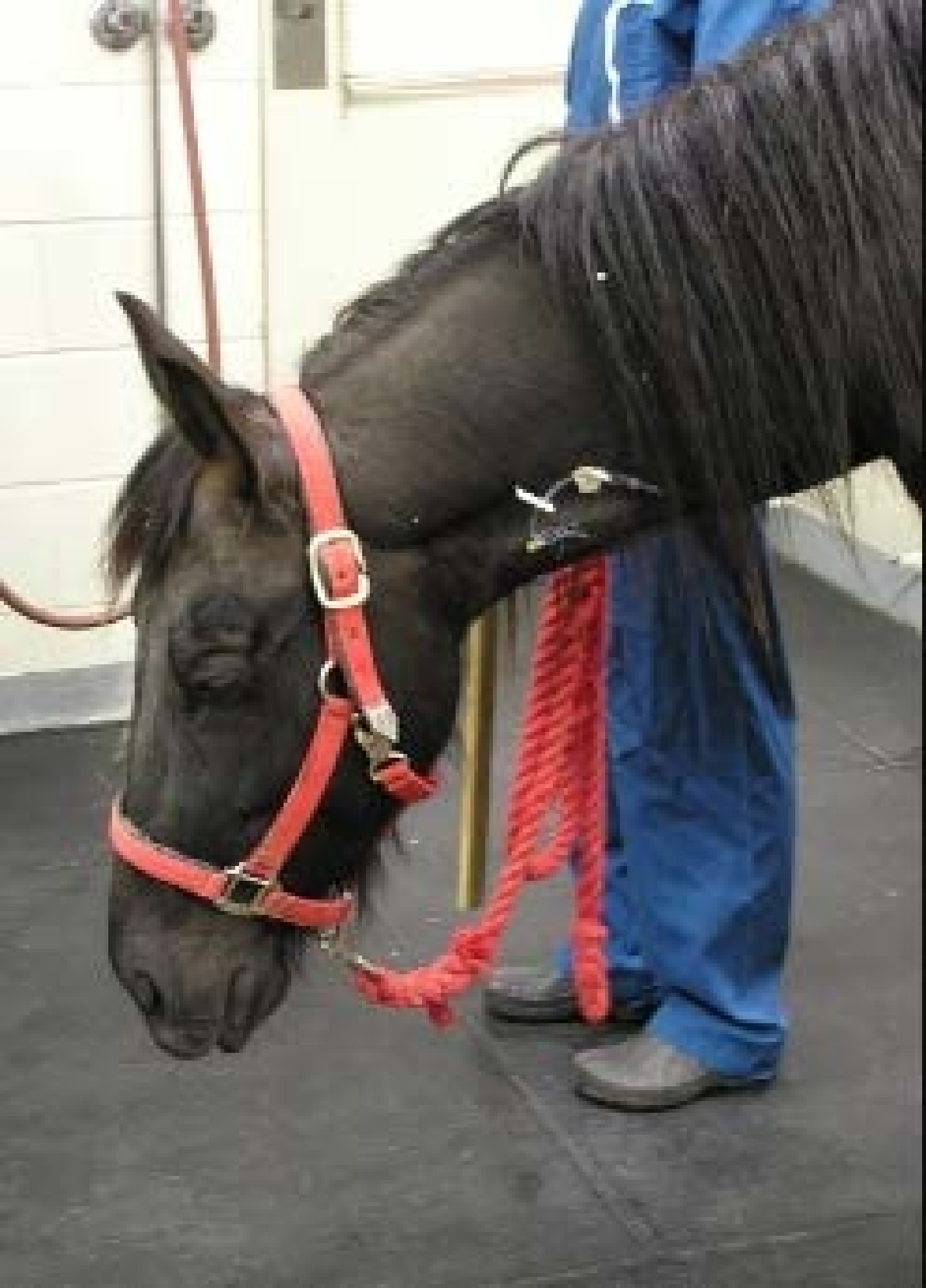
Profound dose-dependent sedation, analgesia, muscle relaxation, ataxia, and sweating are prominent clinical signs of alpha-2 agonist sedation. The head drops low, lips become flaccid, and stertor may develop from relaxation of the nasal passages and alar folds.

Click here to see an image of a horse sedated with xylazine.

Although they are heavily sedated, horses sedated with alpha-2 agonists may suddenly awaken and kick, especially when startled or if the hindquarters are touched. Sudden arousal is a characteristic of sedation with alpha-2 agonists in most companion animals.

Hyperglycemia results from inhibition of insulin release by the pancreas. Urine volume is increased and ileus develops that lasts between 30 minutes and a few hours.

Refs: Muir and Hubbell's Equine Anesthesia, 2nd ed. pp. 192-8, Doherty and Valverde's Manual of Equine Anesthesia and Analgesia, pp. 130-2, and Muir et al., Handbook of Veterinary Anesthesia, 4thed., 36-41 and the Merck Vet Manual online.



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SAVE & EXIT

PREV NEXT

31	32 M	33	34	35	36	37	38	39	40

Which one of the following drug families is most associated with nephrotoxicity as an adverse effect?

Lincosamides	HIDE
Aminoglycosides	HIDE
Sulfonamides	HIDE
Cephalosporins	HIDE
Penicillins	HIDE

BACK NEXT

Overview | Lab Values | Definitions | Report a Problem

Aminoglycosides, like gentamicin and amikacin, are most often associated with NEPHROTOXICITY, ototoxicity and neuromuscular blockade.

Cephalosporins like cephalexin (Keflex®), ceftriaxone (Rocephin®), and ceftiofur (Naxcel®) are relatively nontoxic, but there may be pain at injection site.

Sulfonamides, like trimethoprim sulfa, are associated with HYPERSENSITIVITY (i.e., allergic) reactions.

Lincosamides, like clindamycin, may be associated with GI upset and are CONTRAINDICATED IN HORSES because severe, even fatal, colitis can occur.

Penicillins are not associated much with organ toxicity, but hypersensitivity (allergic) reactions occur, particularly in cattle (e.g., skin reactions, angioedema, drug fever, serum sickness, vasculitis, and anaphylaxis).

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SAVE & EXIT

PREV NEXT

31	32 M	33	34	35	36	37 M	38	39	40

Which one of the following choices best describes the mechanism of action of ivermectin when used to treat ear mites in cats?

Gamma amino butyric acid downregulation	HIDE
Activation of glutamate gated chloride ion channels	HIDE
Nicotinic acetylcholine receptor antagonist	HIDE
Neuronal sodium channel blockade	HIDE
Nicotinic acetylcholine receptor agonist	HIDE

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Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Activation of glutamate gated chloride ion channels

Ivermectin works by inhibiting neuronal activity and muscular contractility in invertebrate parasites in two ways: binding to glutamate gated and GABA gated chloride ion channels.

Mammals are normally protected from toxicity because they do not have neuronal glutamate gated chloride channels, and only have GABA-gated chloride channels in the central nervous system, which is protected by the blood brain barrier and the presence of functional P-glycoprotein.

P-glycoprotein is encoded by MDR1, a gene that is mutated in individuals in many dog breeds such as Collies and Australian Shepherds.

Spinosad works at the level of the nicotinic receptor.

Pyrethrins disrupt sodium channels.

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SAVE & EXIT

PREV NEXT

31	32 M	33	34	35	36	37 M	38	39	40

Atropine is commonly used to treat anesthesia-induced bradycardia. Atropine is contraindicated with the concurrent use of which anesthetic drug in dogs and cats?

Dexmedetomidine	HIDE
Sevoflurane	HIDE
Isoflurane	HIDE
Propofol	HIDE
Butorphanol-diazepam combination	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Definitions | Report a Problem

Atropine and glycopyrrolate (anticholinergics) should NOT be used to treat bradycardia associated with dexmedetomidine in dogs and cats.

Dexmedetomidine, an alpha-2 agonist, causes vasoconstriction and high blood pressure. Bradycardia occurs in response to this elevation in blood pressure ("reflex bradycardia").

Atropine used in conjunction with dexmedetomidine may cause an excessive increase in blood pressure and cardiac arrhythmias.

Dexmedetomidine is a commonly used sedative and preanesthetic in dogs and cats. Avoid it's use in severely debilitated patients and in those with with cardiac, renal, or hepatic disease.

The effects of dexmedetomidine can be reversed with atipamezole.

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SAVE & EXIT

PREV NEXT

31	32 M	33	34	35	36	37 M	38	39	40

Which one of the following choices best describes the reason why flea control measures often fail?

Failure to treat for concurrent *Dipylidium caninum* (tapeworm) infections	HIDE
Treatment regimens do not target all steps in the flea life cycle	HIDE
Difficulty in distinguishing fleas from *Cheyletiella*	HIDE
Product ineffectiveness due to flea resistance	HIDE
Concurrent pyoderma is not addressed	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values

Failure to target the various steps in the flea life cycle is the main reason why flea control measures may be ineffective.

Treatment regimens do not target all steps in the flea life cycle

Understanding the flea life cycle is key to formulating an effective treatment strategy:

-Female fleas lay thousands of eggs over a lifetime

-Eggs are shed into environment and hatch into larvae within 1-10 days

-Larva move deep into carpet fibers or soil

-Larvae can live/pupate for months in ideal conditions (ie. carpets)

-When a host arrives, adults emerge and immediately start feeding on host

-Mating and egg production can begin within 24 hours

Flea control must address ALL of the following:

-Kill adults

-Remove eggs and larvae from environment (ie. vacuum, wash bedding)

-Eliminate access to sources of flea exposure outside; organic debris removal

-Treat ALL pets in the house

-Rigorous flea prevention with effective products for ALL pets in the house

Refs: Cote, Clinical Veterinary Advisor-Dogs and Cats 3rd ed. p. 362-363, Kirk's Current Veterinary Therapy XV, pp.424-427 and the Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

41	42	43	44	45	46	47	48	49	50

Which one of the following statements about the use of steroids in horses is false?

Can cause immunosuppression	HIDE
Associated with laminitis	HIDE
Potent anti-inflammatory effects	HIDE
Used to treat Cushing's disease	HIDE

BACK NEXT

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: Used to treat Cushing's disease

Steroids are not used to treat Cushing's disease.

Equine Cushing's disease (otherwise known as pituitary pars intermedia dysfunction) is caused by a benign hyperplasia of the pituitary gland secondary to loss of dopaminergic inhibition. This is a different cause from Cushing's disease in dogs. The excess ACTH made by the hyperplastic pituitary leads to excessive cortisol release from the adrenal gland, so steroids would not be used to treat this disease.

Steroids do have potent anti-inflammatory effects and can cause immune suppression. Steroids have been associated with the development or exacerbation of laminitis in horses with preexisting risk factors for laminitis (e.g., insulin dysregulation, Cushing's disease, endotoxemia).

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SAVE & EXIT

PREV NEXT

41	42	43	44	45	46	47	48	49	50

Which of the following opioids is more potent than morphine, given most often via continuous intravenous infusion and can be administered via transdermal patch?

Tramadol	HIDE
Buprenorphine	HIDE
Fentanyl	HIDE
Oxymorphone	HIDE

BACK NEXT

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Fentanyl is very lipophilic, so it is absorbed and eliminated quickly. It is therefore not useful for pain control via single doses given intramuscularly (IM) or intravenously (IV).

However, it produces good analgesia when given continuously via intravenous infusion and, in many patients, via a transdermal patch. Serum levels can be variable in some patients with patches.

Absorption is affected by temperature; the use of heating blankets can increase levels, hypothermia will decrease levels.

Fentanyl is less likely to cause nausea and vomiting compared to morphine and other mu-agonist opioids. It does cause respiratory depression and dysphoria in some patients.

Other analgesics that can be administered via transdermal patches are lidocaine and buprenorphine.

Use of patches in patients at home must be evaluated carefully – toxicity and death has been seen in children and small animals that have ingested fentanyl patches.

See very nice articles on pain management and opioids in small animals: Murrell J. Clinical use of opioids in dogs and cats: Part 1. *Companion Animal* 2011; 16(4): 35-8, and Part 2, 16(5): 44-9.

Refs: Gaynor & Muir Handbook of Vet Pain Mgt 2nd ed. pp. 118-9, 244-5, 169, 348, 422, Greene, Vet Anes & Pain Mgt Secrets pp. 78-9, 336-7, Riviere & Papich Vet Pharm & Therapeutics, 9th ed. pp. 320-2 and the Merck Veterinary Manual online edition

Opioids

Opioids continue to be the cornerstone of effective pain treatment in veterinary medicine. The opioids are a diverse group of naturally occurring and synthetic drugs used primarily for their analgesic activity. Despite some well-known adverse effects and disadvantages, opioids are the most effective analgesics available for the systemic treatment of acute pain in many species, particularly dogs and cats. Opioid receptors are part of a large superfamily of membrane-bound receptors that are coupled to G proteins. Each opioid receptor has a unique distribution in the brain, spinal card, and periphery. Opioids combine reversibly with these receptors and alter the transmission and perception of pain. In addition to analgesia, opioids can induce other CNS effects that include sedation, euphoria, dysphoria, and excitement. The clinical effects of opioids vary between the mu opioid receptor agonists (eg, morphine, hydromorphone), partial mu agonists (ie, buprenorphine), and agonist-antagonists (eg, butorphanol). Species and individual differences in the response to opioids are marked, necessitating the careful selection of opioid and adjustment of dose for different species. For example, a 30-kg dog may receive a preoperative dose of morphine (15–30 mg) that is similar to that for a 500-kg horse. Likewise, although butorphanol is widely used as an effective analgesic in horses, its use as an analgesic in small animals is falling out of favor because of its expense, relatively poor somatic analgesic effect, and short duration of action. The clinical effect of an opioid depends on additional patient factors, including the presence or absence of pain, health status of the animal, concurrent drugs administered (eg, tranquilizers), and individual sensitivity to opioids.

Recent information regarding the peripheral endogenous opioid system (PEOS) has presented a unique opportunity to use the powerful analgesic effect of opiates while minimizing untoward systemic effects. The PEOS includes peripheral opioid receptors (POR) and peripheral leukocyte-derived opioids (PLDO): endomorphins, endorphins, enkephalins, and dynorphins. To activate the PEOS, tissue must have sufficient numbers of leukocytes able to secrete PLDO as well as functional POR in sufficient numbers. Inflammation due to tissue damage results in accumulation of PLDO-secreting leukocytes at the site of injury. Inflammation also increases the number and efficiency of POR. These receptors, inactive under normal conditions and expressed on primary sensory neurons, are synthesized in the dorsal root ganglion and transported distally to peripheral sensory nerve endings due to tissue injury and inflammation. Experimental trials and clinical studies show that peripheral opiates are effective, particularly in the presence of inflammation. For example, preservative-free morphine has been instilled into canine and equine joints after arthroscopy or arthrotomy.

zukureview

SAVE & EXIT

PREV NEXT

41	42	43	44	45	46	47	48	49	50

Aminoglycosides are most associated with which adverse effects?

Nephrotoxicity, ototoxicity	HIDE
Potentially fatal colitis (horses), hypersensitivity	HIDE
Hepatitis, nephrotoxicity	HIDE
Ototoxicity, hypersensitivity	HIDE
Pain at injection site, damage to articular cartilage	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | | Report a Problem

Aminoglycosides like gentamicin and amikacin are most often associated with NEPHROTOXICITY, ototoxicity, and neuromuscular blockade.

Cephalosporins like cephalexin (Keflex®), ceftriaxone (Rocephin®), and ceftiofur (Naxcel®) are relatively nontoxic, but there may be pain at injection site.

Sulfonamides like trimethoprim sulfa are associated with HYPERSENSITIVITY (i.e.,nallergic) reactions.

Lincosamides like clindamycin may be associated with GI upset and are CONTRAINDICATED IN HORSES because severe, even fatal colitis can occur.

Penicillins are not associated much with organ toxicity, but hypersensitivity (allergic) reactions occur, particularly in cattle (e.g.: skin reactions, angioedema, drug fever, serum sickness, vasculitis, and anaphylaxis).

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SAVE & EXIT

PREV NEXT

41	42	43	44	45 M	46	47	48	49	50

Which drug is associated with penis protrusion in large animals, especially horses?

Lidocaine	HIDE
Ketamine	HIDE
Acepromazine	HIDE
Atropine	HIDE
Xylazine	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Acepromazine maleate may cause penis protrusion in large animals (especially horses). Acepromazine maleate is a phenothiazine tranquilizer used as a sedative in many animals, as an anti-emetic to control motion-sickness in dogs and as a pre-anesthetic (often with atropine).

Remember 4 things about ace:

1. NEGLIGIBLE ANALGESIC effects

2. Giant dog breeds and sight hounds may be overly sensitive

3. May cause significant HYPOtension

4. May need to DECREASE DOSE in debilitated, geriatric animals

zukureview

SAVE & EXIT

PREV NEXT

41	42	43	44	45 M	46	47	48	49	50

What is the effect of atipamezole administration on the patient with amitraz toxicosis?

Seizures	HIDE
Progression	HIDE
Bradycardia	HIDE
Sedation	HIDE
Reversal	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: Reversal

Atipamezole (Antisedan) can be administered to act as an antidote to amitraz toxic

Side effects of amitraz, a formamidine, include sedation, which may be due to stimulation of alpha 2-adrenergic receptors.

Atipamezole competitively inhibits alpha 2-adrenergic receptors, which tends to reverse sedation and speeds up heart and respiratory rates. Since atipamizole has relatively short half-life, it needs to be administered frequently.

Yohimbine is an alternative treatment option.

Refs: Blackwell's 5-Min. Vet Consult Canine-Feline, 4th ed. pp. 844-5, Plumb's Vet

zukureview

SAVE & EXIT

PREV NEXT

41	42	43	44	45 M	46	47	48	49	50

What is the mechanism of action of phenylbutazone?

H2 histamine antagonist; blocks specific receptors	HIDE
Cyclooxygenase inhibitor; decreases prostaglandin biosynthesis	HIDE
H1 histamine agonist; upregulates histamine receptors	HIDE
Upregulates arachidonic acid metabolism; blocks acetylation of serine residues	HIDE
Lipooxygenase inhibitor; decreases leukotriene metabolism	HIDE

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Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: **Cyd ooxygenase inhibitor; decreases prostagland in biosynthesis**

Phenylbutazone is a nonsteroidal anti-inflammatory drug that inhibits cyclooxygenase (COX) to reduce metabolism of arachidonic acid and biosynthesis of prostaglandins. Phenylbutazone is antipyretic, anti-inflammatory, and analgesic.

H1 antagonists are antihistamines used in the treatment of immediate hypersensitivity reactions, and H2 antagonists are antihistamines used to inhibit the gastric secretory effects of histamine and increase gastric pH.

Refs: The Merck Veterinary Manual online edition.

Antihistamines

By **Scott H. Edwards, BSc, BVMS, PhD, MANZCVSc, 'Senior Lecturer, Veterinary Pharmacology, Charles Sturt University**

Antagonists that selectively block specific histamine receptors have been developed. H_1 antagonists block the actions of histamine responsible for increased capillary permeability and wheal and edema formation. However, because histamine is only one component of an incredibly complex inflammatory cascade, antihistamines have very weak anti-inflammatory activity. H_1 antihistamines may be useful to treat immediate hypersensitivity reactions such as anaphylaxis by blocking bronchoconstriction and vasodilation. H_1 antagonists may be less effective to treat allergic inflammatory diseases, such as atopy, primarily because mediators other than histamine play important roles in such conditions. H_2 (now classified as inverse agonists of the H_2 receptor, such as cimetidine and ranitidine) antagonists are routinely used to block the gastric secretory effects of histamine and have limited anti-inflammatory effects.

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SAVE & EXIT

PREV NEXT

41	42	43	44	45 M	46	47	48	49	50

Which drug can be used to induce vomiting in cats?

Famotidine	HIDE
Meperidine	HIDE
Xylazine	HIDE
Diazepam	HIDE
Ketamine	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Xylazine is used as an EMETIC in CATS, causes vomiting.

Three things to remember about xylazine:

1. Cattle are EXTREMELY SENSITIVE to xylazine. For example, cattle need only 1/10th the dose of xylazine used in horses.
2. Reversed with yohimbine, atipamezole, or tolazoline.
3. Pretreatment with Atropine can decrease bradycardia, hypersalivation seen with xylazine in cattle.

Two ANTI-emetics in cats are diphenhydramine (Benadryl®), and metoclopramide.

Refs: Plumb's Veterinary Drug Handbook, 7th ed. pp. 1397-1402. Bassert and

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SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60

By what mechanism do opiates like morphine and hydromorphone cause post-operative constipation?

Increase intestinal inflammation	HIDE
Reduce intestinal secretion	HIDE
Promote colonic sphincter tone	HIDE
Decrease intestinal motility	HIDE
Stimulate segmental contractions	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Opiates decrease intestinal motility, which can lead to post-operative constipation.

Refs: Plumb's Vet Drug Handbook, 7th ed. pp. 818-20 and the Merck Veterinary Manual online.

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SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60

Which one of the following NSAIDs is the least likely to cause gastrointestinal ulcers and renal disease in horses?

Flunixin meglumine	HIDE
Ketoprofen	HIDE
Phenylbutazone	HIDE
Firocoxib	HIDE
None; these all have a similar risk	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Firocoxib is a COX-2 selective nonsteroidal anti-inflammatory (NSAID) and therefore has lower ulcerogenic and renal risk than the other nonselective NSAIDs listed.

NSAIDs inhibit at least one step in the metabolism of arachidonic acid, usually by inhibiting cyclooxygenase (COX) production of prostaglandins.

COX-1 is present in almost all tissues and is involved in normal homeostatic physiology. COX-2 is activated by inflammation or trauma in tissues and its inhibition is the main desired effect of NSAIDs which leads to their anti-inflammatory, antipyretic, and analgesic actions.
Concurrently blocking COX-1 leads to the possible adverse effects of NSAIDs - ulcerating gastroenteritis (oral cavity, stomach, and right dorsal colon) with hypoalbuminemia and diarrhea, and renal papillary necrosis.

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SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60

Doxycycline is distinguished from oxytetracycline and tetracycline in several important ways. Which one of these statements about doxycycline is true?

IV injection of doxycycline is safe in horses	HIDE
Doxycycline has poor penetration of cerebrospinal fluid compared to oxytetracycline and tetracycline	HIDE
Doxycycline is less likely to cause skeletal abnormalities than oxytetracycline and tetracycline	HIDE
Doxycycline is contraindicated in patients with renal insufficiency	HIDE
Doxycycline is used with niacinamide to treat certain inflammatory dermatoses	HIDE

BACK NEXT LEAVE BLANK

Doxycycline is LESS likely to cause skeletal abnormalities than tetracycline and oxytetracycline.

UNLIKE other tetracyclines, DOXYcycline CAN be used in patients with renal insufficiency.

TETRAcycline is used with niacinamide to treat certain inflammatory dermatoses in dogs.

Small doses of doxycycline IV have been associated with cardiac arrhythmias, collapse, and death in horses (Refs: Plumb's veterinary Drug Handbook, 7th ed. pp. 486-92).

Remember these 2 things:

Remember these 2 things:

1. Pregnancy is the MAIN contraindication for use of tetracyclines in general, which cross the placenta and retard fetal skeletal development.

2. IF you must use tetracyclines in a pregnant animal, Plumb's Vet Drug Handbook states that they should only be used in the LAST HALF of pregnancy when benefits outweigh fetal risks.

US vet students can find an online version of Plumb's free at the Veterinary Information Network (VIN).

Refs: Plumb's Veterinary Drug Handbook, 8th edition, *Doxycycline Calcium, Oxytetracycline, Tetracycline HCl*, and Merck Veterinary Manual online edition.

zukureview

SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60

Mitotane, (o,p, DDD), is used to treat a dog with a particular endocrine condition.

Which of the following correctly identifies this endocrine problem and the additional therapy that is needed during times of stress?

Hypoadrenocorticism, DOCP	HIDE
Pituitary-dependent hyperadrenocorticism, prednisolone	HIDE
Gastrinoma, omeprazole	HIDE
Insulinoma, diazoxide	HIDE
Diabetes insidpidus, chlorpropamide	HIDE

BACK NEXT LEAVE BLANK

Correct: Pituitary-dependent hyperadrenocorticism, prednisolone

Mitotane (o,p DDD) is used to treat pituitary-dependent hyperadrenocorticism (Cushing's disease). Mitotane selectively destroys the glucocorticoid-secreting cells of the adrenal cortex.

REMEMBER dogs receiving mitotane therapy should also be given glucocorticoids during times of stress (ie: surgery, trauma, acute illness). Owners should carry **prednisolone** for times of stress (2.2 mg/kg).

Alternative treatments include l-Deprenyl (decreases pituitary ACTH secretion) or Ketoconazole (inhibits enzymes of cortisol synthesis). These are used in dogs who do not tolerate Mitotane.

Trilostane is also used for treatment of hyperadrenocorticism. Survival times appear to be similar between Trilostane and Mitotane.

Trilostane is typically given SID to BID lifelong, while maintenance Mitotane is typically given 2-3 times per week.

Long-term, Trilostane is more expensive than Mitotane. In general, Trilostane appears to be safer but patients must be monitored for adrenal necrosis, which can lead to an Addisonian crisis and death if unrecognized. Experienced practitioners may disagree on whether to use Trilostane vs. Mitotane.

Refs: *Survival Times for Hyperadrenocorticism after Mitotane and after Trilostane Treatments*, Chastain and Panciera, Sm Anim Clin Endocrinol. January 2006;16(1):19, Plumb's Veterinary Drug Handbook,, Blackwell's 5-Minute Vet Consult Canine Feline, 4th ed. pp. 646-8 and the Merck Veterinary Manual online edition.

Hypoadrenocorticism

(Addison disease)

By **David Bruyette, DVM, DACVIM, Medical Director, VCA West Los Angeles Animal Hospital**

A deficiency in adrenocortical hormones is seen most commonly in young to middle-aged dogs and occasionally in horses. The disease may be familial in Standard Poodles, West Highland White Terriers, Great Danes, Bearded Collies, Portuguese Water Dogs, and a variety of other breeds. The cause of primary adrenocortical failure usually is unknown, although most cases probably result from an autoimmune process. Other causes include destruction of the adrenal gland by granulomatous disease, metastatic tumor, hemorrhage, infarction, adrenolytic agents (mitotane), or adrenal enzyme inhibitors (trilostane).

Clinical Findings:

Many of the functional disturbances of chronic adrenal insufficiency are not highly specific; they include recurrent episodes of gastroenteritis, a slowly progressive loss of body condition, and failure to respond appropriately to stress. Although hypoadrenocorticism is seen in dogs of any breed, sex, or age, idiopathic adrenocortical insufficiency is most common in young female adult dogs. This may be related to its suspected immune-mediated pathogenesis.

A reduction in secretion of aldosterone, the principal mineralocorticoid, results in marked alterations of serum levels of potassium, sodium, and chloride. Potassium excretion by the kidneys is reduced and results in a progressive increase in serum potassium levels. Hyponatremia and hypochloremia result from renal tubular loss. Severe hyperkalemia may result in bradycardia and an irregular heart rate with changes in the ECG. Some dogs develop a pronounced bradycardia (heart rate ≤50 bpm) that predisposes to weakness or circulatory collapse after minimal exertion.

Although the development of clinical signs is often unnoticed, acute circulatory collapse and evidence of renal failure frequently occur. A progressive decrease in blood volume contributes to hypotension, weakness, and microcardia. Increased excretion of water by the kidneys, because of decreased reabsorption of sodium and chloride, results in progressive dehydration and hemoconcentration. Emesis, diarrhea, and anorexia are common and contribute to the animal's deterioration. Weight loss is frequently severe. Similar clinical signs are seen in cats with hypoadrenocorticism.

Decreased production of glucocorticoids results in several characteristic functional disturbances. Decreased gluconeogenesis and increased sensitivity to insulin contribute to the development of moderate hypoglycemia. In some dogs, hyperpigmentation of the skin is seen because of the lack of

negative feedback on the pituitary gland and increased ACTH release. Atypical Addison disease has been reported in dogs and is associated with hypocortisolemia with normal electrolytes. Clinical signs are similar to those seen in dogs with both glucocorticoid and mineralocorticoid insufficiency.

Lesions:

The most common abnormality in dogs is bilateral idiopathic adrenocortical atrophy, in which all layers of the cortex are markedly reduced in thickness. The adrenal cortex is reduced to one-tenth or less of its normal thickness and consists primarily of the adrenal capsule. The adrenal medulla is relatively more prominent and, with the capsule, makes up the bulk of the remaining adrenal glands.

All three zones of the adrenal cortex are involved, including the zona glomerulosa, which is not under ACTH control; however, no obvious pituitary lesions have been seen in dogs with idiopathic adrenal cortical atrophy.

A destructive pituitary lesion that decreases ACTH secretion is characterized by severe atrophy of the inner two cortical zones of the adrenal gland; the zona glomerulosa remains intact.

Diagnosis:

A presumptive diagnosis is based on the history and supportive (although not specific) laboratory abnormalities, including hyponatremia, hyperkalemia, a sodium:potassium ratio of <25:1, azotemia, mild acidosis, and a normocytic, normochromic anemia. Severe GI blood loss has also been reported. Occasionally, mild hypoglycemia is present. The hyperkalemia results in ECG changes: an elevation (spiking) of the T wave, a flattening or absence of the P wave, a prolonged PR interval, and a widening of the QRS complex. Ventricular fibrillation or asystole may occur with potassium levels >11 mEq/L.

Differential diagnoses include primary GI disease (especially whipworm infection), renal failure, acute pancreatitis, and toxin ingestion. For definitive diagnosis, evaluation of adrenal function is required. After obtaining a baseline blood sample, ACTH (gel or synthetic) is administered. Gel preparations are administered IM, and a second blood sample is obtained 2 hr later. Synthetic preparations are administered IM or IV with a second blood sample 1 hr later. Baseline (resting) cortisol concentrations >2.5 mcg/dL effectively exclude the diagnosis of hypoadrenocorticism, whereas values <2.5 mcg/dL require the use of ACTH stimulation testing to confirm the diagnosis. Affected dogs have low baseline cortisol levels, and there is little response to ACTH administration in classic and atypical cases. This test can be completed in most animals before replacement hormone therapy is started.

Treatment:

An adrenal crisis is an acute medical emergency. An IV catheter should be inserted, and an infusion of 0.9% saline begun. If the dog is hypoglycemic, the saline should include 2.5%–5% dextrose. The hypovolemia is corrected rapidly by administering 0.9% saline (60–70 mL/kg over the first 1–2 hr). Urine output should be assessed to determine whether the dog is becoming anuric. Fluids should be continued, at a rate appropriate to match ongoing losses, until the clinical signs and laboratory abnormalities have resolved.

Prednisolone sodium succinate (22–30 mg/kg) or dexamethasone sodium phosphate (0.2–1 mg/kg) may be used in the initial management of shock. Dexamethasone will not interfere with cortisol measurements during the ACTH stimulation test. Prednisolone or prednisone should be given at 1 mg/kg, bid, for the first few days of therapy and then at 0.25–0.5 mg/kg/day. Mineralocorticoid

replacement therapy (*see* below) is also begun to help with electrolyte imbalances and hypovolemia. Electrolytes, renal function, and glucose should be monitored regularly to assess response to therapy.

In cases of severe, nonresponsive hyperkalemia, 10% glucose in 0.9% saline can be given for 30–60 min to increase potassium movement into the cells. Regular insulin (0.25–1 U/kg) administered IM will enhance glucose and potassium uptake, but 10% glucose (20 mL per unit of insulin) should be administered IV concurrently to avoid hypoglycemia.

For longterm maintenance therapy, the mineralocorticoid desoxycorticosterone pivalate (DOCP) is administered at 2.2 mg/kg, IM or SC, every 25–28 days. Electrolytes should be measured at 3 and 4 wk after the first few injections to determine the duration of action. Alternatively, fludrocortisone acetate is administered PO at 10–30 mcg/kg/day. Serum electrolytes should be monitored weekly until the proper dose is determined. Some dogs (especially dogs on DOCP) also require daily oral glucocorticoid therapy to adequately control clinical signs. Replacement doses of prednisone (0.2–0.4 mg/kg/day) are required in ~50% of dogs. Additional glucocorticoid supplementation may be required (2–5 times maintenance) during times of illness or stress. Dogs with atypical Addison disease require only replacement doses of prednisone, although it is recommended that electrolytes be monitored every 3 mo for the first year after diagnosis. Dogs with chronic hypoadrenocorticism should be reexamined every 3–6 mo.

Treatment of horses with hypoadrenocorticism is similar—aggressive replacement of fluids, steroids, and glucose if needed in an adrenal crisis. Supportive therapy and rest are indicated in cases of chronic Addison disease.

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zukureview

SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60

Which drugs can cause "euthyroid sick" syndrome in dogs?

Halothane, Timolol maleate, Ranitidine	HIDE
Furosemide, Phenobarbital, Mitotane (o,p DDD)	HIDE
Phenylbutazone, Phenoxybenzamine, Fluoroquinolones	HIDE
Allopurinol, Benzodiazepines, Prednisolone	HIDE
Tetracyclines, Iodine-containing supplements, Ipodate	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: **Furosemide, Phenobarbital, Mitotane (o,p DDD)**

Euthyroid sick syndrome in dogs occurs when drug treatment or a non-thyroidal illness causes a decrease in T3/T4 hormones.
Not to be confused with true hypothyroidism.

Many drugs can depress T3/T4 hormones, including:
Glucocorticoids, anabolic steroids
Anticonvulsants (phenytoin, phenobarbital)
Phenylbutazone
Ipodate (radiographic contrast agent)
Furosemide
Anesthetics/induction agents (methoxyflurane, halothane)
Mitotane (o,p DDD)

Think of these diseases with euthyroid sick syndrome:

Hyperadrenocorticism (Cushings)
Diabetes mellitus
Hypoadrenocorticism (Addisons)
Chronic renal disease (hard to miss-these dogs are very sick)
Hepatic disease
Calorie or protein deficiency
Surgery/anesthesia

Finally, (because endocrine problems like to be complicated), sulfonamide antibiotics can induce overt primary hypothyroidism.

Refs: Pasquini's, Tschauner's Guide to Sm An Clin, vol 1, 2nd ed. p. 675, Blackwell's 5-Minute Vet Consult Canine Feline, 4th ed. pp. 732-3 and the Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60

Which of the following choices is true about detemir insulin in dogs?

Most effective when administered every 48 hours	HIDE
Administered intramuscularly in dogs vs. subcutaneously in cats	HIDE
Lower starting dose than other long-acting insulins	HIDE
Risk of hypoglycemia is lower compared to other long-acting insulins	HIDE
Anti-insulin antibodies less likely because derived from pork	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct: Lower starting dose than other long-acting insulins

The starting dose for detemir is LOWER in dogs. Canine insulin receptors are 4x more sensitive to detemir than are human receptors. Detemir is a *human* insulin analog.

There is a **higher** risk of hypoglycemia with detemir.

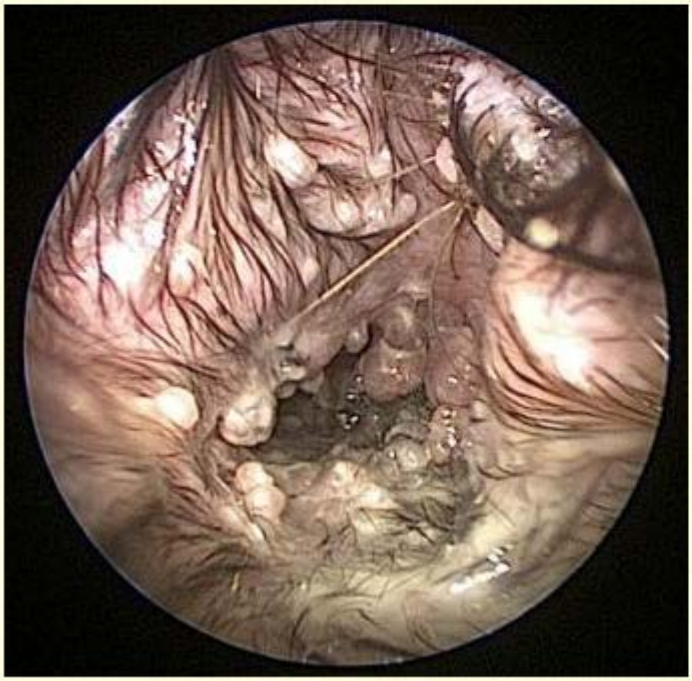
Determir is administered **subcutaneously every 12 hours**.

Refs: Plumb's Veterinary Drug Handbook, 8th edition, *Insulin*.

A 6-year-old male neutered cocker spaniel is presented with a history of chronic recurrent ear infections. Otoscopic examination shows marked erythema and severe glandular hyperplasia.

The tympanic membrane cannot be visualized. Cytology of the affected ear also shows lots of bacteria - primarily rods (too numerous to count) and many neutrophils. Chronic bacterial otitis is the primary diagnosis.

Since it is unclear so far if the tympanic membrane is intact, which one of the following antibiotics has the lowest risk of ototoxicity?



Amikacin	HIDE
Gentamycin	HIDE
Enrofloxacin	HIDE
Tobramycin	HIDE
Neomycin	HIDE

BACK NEXT LEAVE BLANK

Correct:

Fluoroquinolones such as enrofloxacin are safe.

If you cannot see the tympanic membrane due to severe inflammation and hyperplasia or if the tympanic membrane is ruptured you should avoid aminoglycoside antibiotics (ie: Amikacin, Neomycin, Tobramycin, Gentamycin).

Some authors also recommend that macrolide antibiotics (ie: erythromycin) should be avoided when it is unclear if the tympanic membrane is intact.

Refs: Cote, Clinical Veterinary Advisor-Dogs and Cats, 3rd ed. pp. 742-4, 1316, Plumb's Vet Drug Handbook, 7th ed. pp. 504-9. Image courtesy of Dr. Patrick Hensel.

zukureview

SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60
✓	✗	✓	✓	✗	✗	✗			

Which insulin type acts the fastest but has the shortest duration of action in the dog?

Lente	HIDE
Ultralente	HIDE
Regular	HIDE
Insulin Glargine	HIDE
NPH	HIDE

BACK NEXT LEAVE BLANK

Overview | Mark this Question | Lab Values | Definitions | Report a Problem

Correct:

Regular insulin acts fastest and has the shortest duration. The easiest way to remember these:

1. **Regular is FASTEST**-acting insulin (so often used to Rx emergencies), but SHORTEST duration (4-10 hrs).

2. **Ns are Ntermediate** duration-NPH (6-18 hr, dogs; 4-12 hr, cats) and LeNte (8-20 hr, dogs; 6-18 hr, cats).

3. **U and Z are at the end** of the alphabet, and they are longest duration too: Ultralente means "ultra-slow" (6-24 hr duration, dog and cat) and PZI (6-28 hr, dogs; 6-24 hr, cats).

In general, duration of insulin action is SHORTER IN CATS than in dogs, which may explain why 75% of cats need BID insulin.

Increasingly, Glargine (a long-acting insulin) is preferred in cats, in combination with a high protein, low carbohydrate diet. Lente insulin or NPH insulin are commonly used in dogs.

The types of insulin available change as products go on or off the market. For example, Ultralente insulin and PZI are no longer available.

For more on diabetes mellitus, see the Merck Veterinary Manual online.

Refs: Pasquini's, Tschauner's Guide to Sm An Clin, vol 1, 2nd ed. pp. 662-4, Plumb's Vet Drug Handbook, 7th ed. pp. 710-8 and the Merck Veterinary Manual online edition.

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SAVE & EXIT

PREV NEXT

51	52	53	54	55	56	57	58	59	60
✓	✗	✓	✓	✗	✗	✗	✗	✓	

Alprazolam is a benzodiazepine tranquilizer used for the treatment of underlying behavioral issues causing feline elimination disorders.

Which one of the following choices is the mode of action of this drug?

Gamma amino butyric acid potentiation	HIDE
Gamma amino butyric acid inhibition	HIDE
Dopamine (1) receptor agonist	HIDE
Dopamine (1) receptor antagonist	HIDE
Dopamine (2) receptor antagonist	HIDE

BACK

This is the last question. Click Save and Exit after you finish it.

FINISH LEAVE BLANK AND FINISH

Correct.

GABA potentiation

Alprazolam is a benzodiazepine tranquilizer used to moderate anxiety in the urine spraying cat.

Benzodiazepines **potentiate** the effects of the neurotransmitter gamma amino butryic acid (GABA) by increasing its affinity to the GABA receptor.

Activation of the GABA receptor actually produces hyperpolarization of neurons and therefore inhibition of neuronal transmission. A calming effect is seen from inhibition in the limbic and reticular formations in the brain.

GABA receptors are also known as benzodiazepine receptors.

Refs: Cote, Clinical Veterinary Advisor-Dogs and Cats, 3rd ed. pp. 541-3, Grimm,