ICVA NAVLE - Quiz Questions with Answers

Clinical Practice

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1.

A previously well-regulated diabetic, a 10-year-old FS Siamese, presents to the dental service for a dental. The cat has severe gingivitis and presumed resorptive lesions. Her diabetes has been difficult to manage for the past few weeks. A urinary tract infection was ruled out, no evidence of Somogyi was present after a 24-hour blood glucose curve, and the only sign of disease that could be negatively affecting regulation is her teeth. You need to advise the client about feeding, fasting, and insulin dosing. Which of the following is correct?

Fast the animal six to eight hours before surgery

Maintain blood glucose levels < 150 intraoperatively

Check blood sugar one to two hours pre-op, and if between 150–300 mg/dL, give regular insulin prior to surgery

IV fluid therapy should be at the standard surgical fluid rate

Correct answer: Fast the animal six to eight hours prior to surgery

Diabetics can be challenging when it comes to anesthesia. Maintaining adequate perfusion is paramount to preventing damage to the pancreas (including pancreatitis). Most people underestimate the degree of dehydration in these patients, especially when obese.

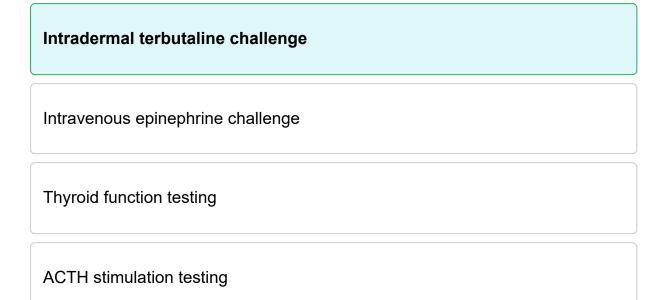
We want to fast them no more than six to eight hours pre-op. Ideally, have them fed normally and given their normal insulin the day before. One to two hours prior to surgery, check their blood sugar. If it is 150–300 mg/dL, the animal should get 1/2 of its normal insulin dose subcutaneously (long-acting, not regular insulin). It is ideal to do diabetics first thing in the morning to permit them then to get back to their normal feeding/insulin schedule. However, if you have to do surgery later in the day, they should have their blood sugars checked and a small meal and dose of insulin early in the morning and still, ideally, be fasted for six to eight hours before cutting time.

Sugars should then be checked at induction and then hourly. If the blood sugar is low, the patient should be started on a 2.5 % dextrose solution in 0.45% saline at 10–15 ml/kg for the first hour, then 5 ml/kg/hr afterward. If blood sugars are normal, lactated ringers can be used at the same fluid rates. (Again, remember that these patients are dehydrated, and usually, we underestimate this dehydration status). Frequent blood

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A Warmblood presents with progressive exercise intolerance, and the trainer feels that the horse is not sweating appropriately. However, there is still evidence of sweating under the horse's mane, groin, and axillary areas. With exercise, the trainer has noted that the horse's respiratory rate is elevated, and there is increased nostril dilation. The trainer took his temperature this morning, which was 103°F (39.44°C) after just being stabled for the night. The ambient temperature last night was > 75°F (23.89°C). You suspect anhidrosis.

What diagnostic test can confirm the presence of anhidrosis in our equine patients?



Correct answer: Intradermal terbutaline challenge

Testing for possible endocrine diseases or concurrent diseases that could affect an animal's ability to sweat and expel heat or lead to exercise intolerance is important. Still, it will not confirm the diagnosis of anhidrosis.

The ACTH stimulation test is used to diagnose PPID (Pituitary Pars Intermedia Dysfunction), where 75% of horses with PPID will have an exaggerated response.

Thyroid function testing (Serums T3 and T4 assays) is beneficial to rule out hypothyroidism as it may contribute to or accompany anhidrosis. However, the true etiology and clinical course of hypothyroidism and anhidrosis are not fully elucidated, and this test, while beneficial and may provide valuable information, doesn't definitively diagnose the condition.

We can diagnose anhidrosis by performing an intradermal sweat test. We can use terbutaline or epinephrine IM to do so.

1. An intradermal terbutaline challenge: 0.5 mg of terbutaline is injected IM with six serial dilutions (ten fold). Thus, instilling 0.1 ml in the neck or pectoral areas intradermally. Normal horses will have a local sweat response in that area at all concentrations except maybe the most dilute. The amount of sweat will be proportional to the dose. Normal horses usually start to sweat about five minutes post higher-concentration injections. If complete anhidrosis has yet to

 develop, we may see sweating at higher concentrations. Those with full disease will not sweat even at the highest concentration. 2. Intradermal epinephrine injection (not intravenous, as this will exacerbate the issue) may be used. Administer an injection of 1:1000 and one of 1:10,000 epinephrine intradermally. Those not affected should have local sweating within an hour, while those affected will not.

An 11-month-old male Thoroughbred is presented with a history of clumsiness. He did run into the fence two weeks prior and had seemed a bit ataxic, but the owner chalked it up to the trauma. On physical, the animal has lameness in the hind that you grade higher than in the forelimbs. He is BAR with no change in mentation. There are no cranial nerve deficits. He resents neck flexion. When you do a sway test (pulling the tail to one side firmly as the horse walks away), there is decreased resistance to pressure applied laterally. You walk the horse in tight circles, and he circumducts the outside pelvic limb.

What is the treatment of choice for this patient for the best chance of neurological improvement?

Ventral interbody vertebral fusion (arthrodesis) Corticosteroids or NSAIDs Exercise restriction and long-term dietary restrictions Stall confinement for six months

Correct answer; Ventral interbody vertebral fusion (arthrodesis)

This colt shows classic signs of cervical stenotic myelopathy, AKA wobblers, cervical vertebral stenotic myelopathy, CVSM, CVM, cervical compressive myelopathy, and CCM. Clinical signs may start as young as six months to five years, depending on the type of lesion. Animals are ataxic secondary to developmental abnormalities in the spinal cord. Type 1 has a focal/multifocal vertebral canal stenosis, worse on flexion, between C1-6. This refers to dynamic stenosis and usually occurs in younger animals. Type II consists of C5-T1 lesions most commonly and involves soft tissue and bony spinal cord impingement with bone remodeling and stenosis.

Though they can occur acutely, signs are often slowly progressive, with weakness, ataxia, and a dysmetric gait. Animals may have changes to the cutaneous truncii reflex, paresis, and a decreased slap reflex. Usually, the hind legs are more affected than the fore. They are usually mentally appropriate with normal cranial nerve exams.

Differentials include equine protozoal myeloencephalitis, equine degenerative myelopathy, trauma, fractures, neoplasia, equine herpesvirus-1, or other malformations/congenital abnormalities.

Treatment varies with the horse's age and if it has reached its full growth potential. Treatment may consist of pain management with NSAIDs/steroids, often only providing short-term relief. Exercise and dietary restrictions, stall rest, or intra-articular

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A one-year-old FS mixed-breed dog presents to your office with focal alopecic areas, varying scaling, crusting, hyperpigmentation, papules, and a few affected nails. The owner noticed changes about three weeks ago, and the dog is increasingly pruritic. The owner brought her in, however, because her mother-in-law, who is being treated for lymphoma, lives in the home and developed skin lesions as well. She has a few draining tracts starting, erythematous areas, and the lesions are asymmetrical. They are concerned that she got something from the dog. The dog is otherwise healthy, on Nexgard and Hartgard Plus, and up-to-date on vaccines. Given the concern for zoonotic disease, you are most suspicious of either scabies or ringworm.

Which of the following is the medical-legal test used to identify fungal species?

Dermatophyte culture
Dermoscopy
Wood's lamp examination
Real-time PCR

Correct answer: Dermatophyte culture

Dermatophytosis or ringworm is caused by various species, is zoonotic, and is more common in cats than dogs. It is not life-threatening and usually spontaneously resolves without therapy, though it can take weeks to months. Further, in this patient, if the mother-in-law does have a ringworm species known to infect dogs, such as M. canis, and because she is immunosuppressed due to chemotherapy, treatment of this dog is warranted if the disease is confirmed. While zoonotic, most ringworm infections are more commonly associated with immunosuppressed individuals. However, always be sure to discuss the risks with owners when pets are diagnosed with ringworm, regardless of the species of ringworm and pet.

Diagnostics include:

- 1. Dermoscopy is a Point-of-Care (POC) tool where you use a handheld device to identify hairs for direct examination or culture. It can confirm a diagnosis when used in conjunction with a direct exam. It provides magnification of the hairs, and you can look for broken "comma" shaped ones. Select those to pluck for evaluation/culture.
- 2. Wood's lamp, a POC tool, ID's hairs for direct examination and/or culture by producing an apple green fluorescence on the hairshafts. A direct exam can confirm the diagnosis. However, Not all species fluoresce. Animals who have had some treatment will be less likely to fluoresce than those who haven't.

- 3. Direct examination (cytology) POC permits one to evaluate the hair via skin scrapings, spatula, mineral oil, and/or forceps and identify disruption of the corticomedullary junction, pale hair, and arthroconidia in the cuticles may be evident.
- 4. Fungal culture the gold standard and the medical-legal test to ID fungal species. This can be either in-clinic or in the lab. Plates are preferred to vials for in-house tests, permitting inoculation with a toothbrush.

5. Skin biopsy – punch biopsy or wedge/excisional biopsy from a nodule; requires special stains, so inform the pathologist what you are looking for if ringworm is suspected 6. Real-time PCR – hairs, crusts, and scales but does require large amounts of sample. A negative test suggests no ringworm, but false positives from fomites are common. Further, they aren't yet currently used to monitor for a cure.

You have a green iguana that has presented with egg retention. However, the patient has had no exposure to intact males and, while anorectic, has remained BAR. Which of the following cannot cause non-obstructive egg retention?

Anatomical defects Lack of suitable egg-laying location Malnutrition Inappropriate environmental temperatures

Correct answer: Anatomical defects

Note that a BAR iguana with anorexia and egg retention is likely not having dystocia. Animals with dystocia are usually depressed and become rapidly unresponsive when gravid.

Causes of non-obstructive egg retention in iguanas include lack of a proper egglaying chamber, excessively low temperatures, dehydration, poor nutrition, and poor physical condition (which may be a factor of nutrition, husbandry, or other underlying causes). An infection could contribute to this type of egg retention, but it can be hard to determine whether it caused retention or resulted in infection.

Obstructive causes of egg retention and/or dystocia may include anatomical abnormalities preventing eggs/young from passing. These may include deformed, malpositioned, too-large, or fractured eggs, and stricture or torsion of the shell gland may also trigger it. Finally, cancer, uroliths, or abnormally sized kidneys could constrict the oviducts, preventing passage. A narrow pelvic canal or spinal deformity secondary to metabolic bone disease (secondary nutritional hyperparathyroidism) can also contribute. Finally, trauma from a previously difficult egg-laying period could result in obstructive changes.

Note: Dystocia is defined by Divers and Stahl (in Mader's book) as "the inability to successfully expel term eggs or fetuses from the lower reproductive tract." Causes may include abnormal egg sizes, poor contractility, improper or lack of nesting areas, and lack of viable embryos despite mating.

You note an umbilical hernia on a cow you are evaluating at slaughter. Upon incising it, you note that there is only fat and omentum within the hernia. You hadn't thought much about what breed of cattle it was, but upon seeing it in this cow, you recall what breed is predisposed?

Holstein Friesian
Herford Cattle
Jersey Cattle
Shorthorn cattle

Correct answer: Holstein Friesian

Umbilical hernias can happen in any species and can be of varying size and severity. They may contain just fat or omentum or, worse, GI loops. Holstein Friesian cattle are overrepresented relative to other breeds. We commonly see them in dogs and foals. Clinical signs may be associated with hernias if intestinal loops are within the body wall defect.

You are asked to evaluate a goat with clinical signs that include chewing, licking, depression, weight loss, hypertonicity, recumbency, hyperreflexia, proprioceptive deficits, wool break, coma, and death. You suspect a reportable disease and need to collect samples.

What samples can confirm your diagnosis?

Microscopic evaluation of the brain/spinal cord

Prp-Sc immunohistochemistry from tonsillar lymphoid tissue

Antibody blood levels

Prp-Sc immunohistochemistry from retropharyngeal lymph nodes

Correct answer: Microscopic evaluation of the brain/spinal cord

The clinical signs described above demonstrate key signs in sheep and goats with Transmissible Spongiform Encephalopathy (TSE), known as scrapie. Sheep are the natural hosts, though goats can laterally and vertically transmit the disease as well. Susceptibility and resistance occur in various breeds. Scrapie, unlike Bovine Spongiform Encephalopathy (BSE), another prion disease, affects not only the gut-associated lymphoid or nervous system tissue but also various lymphatic tissues, the kidneys, and the placenta. Because of this, biopsy techniques for lymphoid tissue have been developed. Immunohistochemistry can be used to evaluate biopsy sections of lymphoid tissue, including tonsils, retropharyngeal lymph nodes, or nictitans. However, sensitivity is variable; classically, diagnosis is made by microscopic examination of the brain and spinal cord. The disease is not overtly zoonotic but is fatal. Eradication measures vary globally.

A complete necropsy should be performed on any sheep (+/- goat) dying without an overt cause; this includes submitting the brain +/- spinal cord for diagnosis.

A bird presents with a known ingestion of a penny made within the last ten years. That was a day or two ago. The owner reports that the bird puts everything in her mouth. Today she became weak and was regurgitating. She also doesn't want to eat and seems to drink a lot. You are concerned with zinc toxicosis, and in addition to radiographs, which show what looks like multiple coins, you want to run zinc blood levels.

What type of blood tube is recommended to ensure results aren't artifactually elevated?

All-glass or all-plastic syringes and blood tubes

Blue-top tube with sodium citrate

Yellow-top tube with acid citrate dextrose solution

Purple-top tube

Correct answer: All-glass or all-plastic syringes and blood tubes

Red serum separator tubes are either plain or have a clot activator gel in them. These tubes can be used for serology and some infectious organism testing, but this varies with the laboratory used. Because of the rubber stopper or grommets within the tubes, these tubes are unsuitable for zinc levels.

Blue-top tubes (pastel blue) are used for coagulation-related testing, including d-dimer, PTT, and PT. The additive is sodium citrate.

Yellow-top tubes contain acid citrate dextrose and a separator gel. These tubes can evaluate chemistry panels, thyroid levels, and some immunology and serology testing. Again, it depends on the lab used.

Purple-top tubes contain EDTA (ethylenediaminetetraacetic acid), an anticoagulant to prevent clotting. This tube is generally used for CBCs and peripheral blood smears.

Finally, when testing for zinc levels, one must use all-plastic or all-glass tubing. The tube cannot have rubber of any kind, as this can jeopardize test results. Rubber stoppers or grommets on some tubes can falsely increase zinc levels. Sent in the appropriate tubes, zinc levels of > 2 ppm are positive for zinc toxicity.

Treatment includes supportive care and using a chelator such as calcium EDTA or d-penicillamine. If the pennies don't pass, surgical removal will be needed. Thankfully,

A client comes to you with a severely pruritic cat with miliary dermatitis on the dorsum, base of the neck, head, and tail. The cat has alopecia, ulcerations, and crusts and seems very uncomfortable. He flinches when you touch his skin. This has been going on for over a month. The cat is indoor-only, but there is a dog in the home, not on monthly flea/tick prevention. However, you find no fleas on the cat or any flea dirt. The cat is negative on skin scrape and has a significant secondary bacterial infection. The trichogram shows self-damage, but no obvious mites, and a DTM is pending. Tape prep shows no malasezzia. The owner declines to culture any of the papules. The owner also thinks something is mentally wrong with the cat because he will randomly start scratching and then run zoomies all around the house.

All of these are good first steps to help resolve what you suspect is the main cause of the cat's clinical signs before doing additional costly tests and treating secondary infections, except:

Topical glucocorticoids

A regularly applied prescription flea/tick preventative for cats

A prescription-strength, new-generation flea/tick preventative for the dog and any other pets in the home

Environmental cleanup and education regarding the life cycle of fleas

Correct answer: Topical glucocorticoids

This cat is classic for Flea-Allergy Dermatitis (FAD). The cat is severely pruritic, has freak-out periods and severe itchiness, and, while no fleas are found, has alopecia and lesions in the expected locations. > 90% of cats with a miliary dermatitis usually have a FAD. Owners need to learn and understand a flea life cycle and realize that just because they don't see fleas or flea dirt doesn't mean they aren't there. This cat is likely overgrooming and cleaning off every live flea immediately because he is so miserable.

If this client can safely bathe the cat, topical therapy may be sufficient (bathing) to treat the infection component of the condition. The use of medicated shampoos effective against bacteria and with soothing agents is often sufficient for superficial infections. However, most cats aren't amenable to regular bathing, and thus, oral antimicrobials are likely necessary for this pet. Glucocorticoids are not usually given topically to cats as they will probably groom them off. So a short oral tapering prednisolone dose may be beneficial until the reaction calms down and he is more comfortable.

However, it isn't just important that the owner treats and prevents fleas on the cat. The owner needs to ensure any pet in the house, whether it goes outside or not, has appropriate flea/tick prevention for three plus months and that they are diligent about daily environmental cleanup, which can also be required for three months or more, due to the life-cycle of the flea. The duration of in-home cleaning will also depend on the season, the temperature in the home, and additional factors. Failing to treat other pets and clean up the environment will permit the FAD to continue. Further, it will make diagnosis more difficult because FAD won't be able to be ruled out unless the owner is fully compliant.						

A chelonian presents with wheezing, tachypnea, open-mouth breathing, and stretching out its neck to breathe. The patient is depressed and lethargic, and the limbs move more than they should upon breathing. The turtle seemed to be swimming and/or in the water cockeyed for a few days before the onset of respiratory signs. The owner reports that his appetite has been decreased for a week or two. After discussing husbandry, diet, and other parameters, you discuss treatment plans. The owner would like to try to manage medically and consents to bloodwork and intraosseous catheter placement to permit fluid therapy and medication delivery, as you suspect venous access will be difficult due to the patient's small size, degree of respiratory distress, and species anatomical norms.

All of the following locations would be appropriate intraosseous or intravenous catheter sites in chelonians, except:

Ventral coccygeal vein
Humerus/femur
Jugular vein
Plastrocarapacial bridge

Correct answer: Ventral coccygeal vein

In chelonians, the right jugular is notably larger than the left and can be used for catheter placement. Given this patient's degree of respiratory distress, the ventral coccygeal vein would be an unfavorable location due to the required positioning and stress for the patient.

The cephalic vein can be used in patients greater than 15 kg. For those smaller than that, an ultrasound can be used to guide placement.

The plastrocarapacial bridge can be used for IO placement, but requires a drilled pilot hole and is considered less efficacious than one of the long bones.

The humerus and femur can both be used for IO placement with similar techniques to mammals.

You are discussing a patient's history with a client. The client tells you that her 12-year-old FS DSH has had decreased appetite and lethargy on and off for a few weeks. You inquire about any vomiting or diarrhea, but there isn't any. Your physical exam shows no abdominal pain, and the owner reports no overt signs of pain either. The owner is highly worried about cancer. You inquire about drinking and urination habits, and no changes have occurred. Given this owner's history, which of the following has to be a top differential for this patient?

Pancreatitis
Metabolic disease
Cholangitis
Neoplasia

Correct answer: Pancreatitis

Cats are not small dogs. Dogs may show a wide array of clinical signs with pancreatitis, from vomiting, and diarrhea, to anorexia, lethargy, abdominal pain, icterus, and weight loss. In some studies, less than 50% of cats have overt abdominal pain. The most common clinical signs appreciated are lethargy and decreased appetite. Some cats will have vomiting, varying degrees of dehydration, belly pain, icterus, and weight loss (depending on chronicity).

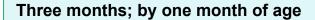
Metabolic disease is possible, but patients with endocrine disease usually have additional clinical signs such as changes in drinking/urination, coat quality changes, or other abnormalities that lead you in that direction.

Cholangitis patients (depending on the form, neutrophilic, lymphocytic, or chronic) tend to have signs ranging from vomiting to diarrhea and appetite changes. They, too, may be lethargic and may also have ptyalism. However, they generally have painful abdomens more consistently than those with pancreatitis. The vomiting and diarrhea may be acute and significant or chronic and intermittent. In acute cases, patients are generally febrile. The signs tend to be a bit less generalized than lethargy and a decreased appetite, and the prevalence of pancreatitis, while not fully elucidated in cats, is presumed to be common. These patients may present with icterus but do not have to have reached that state yet. However, it cannot be ruled out as we commonly see more than one inflammatory condition in cats at a time, associated with the pancreas/liver/or GI tract.

Given the patient's age, Neoplasia has to be on the list. Still, based on your history evaluation and discussion and the patient's clinical signs, this wouldn't be the first

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You are treating a foal for an angular limb deformity. When treating these types of deformities, you must know and understand the relative growth plate closure times of the various limbs/bones to ensure intervention occurs in a timely fashion. Active growth of the distal metacarpal physis occurs during the first how many months of life? Thus, treatment should begin by what age?



Nine months; by four months of age

Nine months; by six months of age

Six months; by five months of age

Correct answer: Three months; by one month of age

Angular limb deformities refer to a limb's medial or lateral deviation, named by the joint where the issue arises. The deviation's direction is based on the limb distal to the joint. Varus deformity is a medial deviation relative to the reference point, while valgus deformity is a lateral deviation. Angular limb deformities occur as a result of a:

- 1. Periarticular laxity
 - 2. Incomplete ossification of the cuboidal bones
 - 3. The disproportionate growth of both the epiphysis and metaphysis

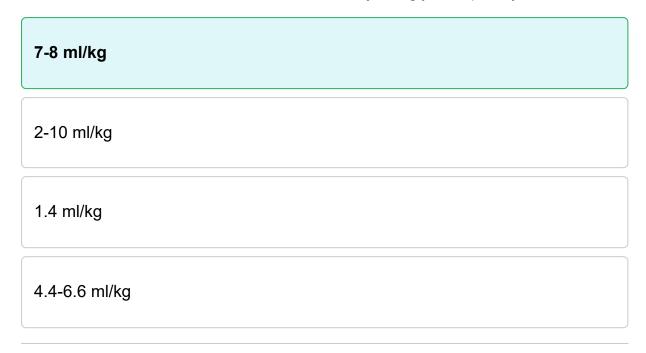
When we are referring to the disproportionate growth that commonly causes carpal or tarsal valgus. If the deviation is under ten degrees, treatment is conservative. Moderate cases require surgical correction. Understanding when the growth plates close is key to knowing when intervention must be initiated. The distal metacarpal physis grows during the first three months. Thus, any changes being made need to occur by the age of one month. Compare this to the distal radius and tibia, where those plates grow through nine months of age. Any manipulation must be achieved by four to six months of age. The more severe the deformity, the earlier the intervention may be needed.

If you have a patient with limb deformities, make sure you do a complete PE (Physical Examination). Always take radiographs. If any bandaging is required, ensure daily bandage changes are performed and the limb is assessed daily to prevent wounds.

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A friend of yours says their backyard chicken flock managed to escape the coop and was meandering around on the driveway, where they fear there was an antifreeze leak from one of the cars. They're concerned the flock could have been exposed. You go to investigate the flock and perform evaluations.

What is the minimum lethal dose of undiluted ethylene glycol in poultry?



Correct answer: 7-8 ml/kg

Ethylene glycol toxicity is common in domestic dogs and cats, but all species are susceptible. It is found in a variety of automotive products, most notably antifreeze, as well as in household products. Ultimately, toxicity leads to progressive and, if untreated, irreversible kidney failure. Signs may include GI upset, depression, cerebral edema, and metabolic acidosis. Treatment, if the problem is identified in time, is with fomepizole or ethanol.

Toxic doses in animals are as follows:

Cats: 1.5 ml/kg
Dogs: 4.4-6.6 ml/kg
Poultry: 7-8 ml/kg
Cattle: 2-10 ml/kg
Macaques: 1.6 ml/kg
Guinea pigs: 6.61 ml/kg

Poultry may exhibit watery feces, ataxia, lethargy, recumbency, torticollis, dyspnea, and ruffled feathers. They do not typically develop gross lesions suggestive of disease.

Toxicity is caused by the breakdown of ethylene glycol to oxalic acid. This, in turn, combines with calcium-forming calcium oxalate. The oxalate crystals then block renal tubules leading to necrosis and nephrosis.

A Great Pyrenees, two-year-old MN, presents because he keeps getting his double dewclaws caught on anything and everything. The owner has spent a lot of money on bandages and emergency room visits. Further, the dog has had to spend most of his second year of life exercise-restricted because he is always healing or in a bandage. The owner requests dewclaw removal to prevent further issues with her klutzy dog.

All of the following can provide appropriate pain relief for a minor procedure such as a hindlimb declaw removal, except:

Intravenous Regional Anesthesia (Bier block) (IVRA)

Metatarsal ring block

Methadone and dexmedetomidine in pre-op protocol

Post-removal incisional block, bupivacaine liposome

Correct answer: Intravenous regional anesthesia (Bier block)

All of the above, including a Bier block, will provide appropriate pain management as a general injection (methadone and dexmedetomidine) or by local anesthetic actions.

A line block in the incision upon closing will help either with a short-acting or semi-short-acting local anesthetic like lidocaine or bupivacaine or off-label use of the bupivacaine liposome (Nocita®), which can persist for up to 72 hours depending on the location. It is labeled for cranial cruciate repairs, but many surgeons use it for closing any incision.

A metatarsal ring block will desensitize the superficial and deep peroneal nerves and the tibial +/- plantar and lateral nerves. This should provide numbness in the area of concern. It helps block the paw and the metatarsal regions.

An IVRA can be more effective than a ring block for major procedures. However, it is more ideal for the forelimb below the elbow than the hindlimb. It can be used for the hindlimb, but is much easier and more commonly used for the forelimbs. Further, dewclaw removal, even with firmly attached ones, is usually a short and minor procedure when done by a trained professional, and a ring block should be sufficient. Further, it requires a tourniquet which limits surgical time and is likely overkill for this patient.

A 12-year-old MN Corgi presents with a several-month history of ataxia and hindlimb weakness without pain. They first noticed that he was dragging his toes in the back feet but was becoming more and more ataxic. He hasn't been able to jump onto the couch this week, leading to the visit as they miss cuddling with him while watching tv. His neurological exam shows he is ataxic and dragging his toes on both hind limbs. He is weak. He has normal to hyperreflexive reflexes in the hind and his patellar reflexes are not present. He has mild muscle atrophy over the pelvic limb. The muscle loss is symmetrical. What is your top differential for this patient?

Degenerative Myelopathy (DM)

Hansen Type II intervertebral disc herniation

Primary spinal cord neoplasia

Infectious meningomyelitis

Correct answer: Degenerative myelopathy

Degenerative Myelopathy (DM) affects dogs, usually over eight years of age. It is most commonly appreciated in the GSD but can be seen in other large breed dogs and the corgis. (GSDs, Boxers, Corgis, Rhodesian ridgebacks, poodles, Irish setters, and Bernese Mountain dogs are commonly noted with this condition). It is a symmetrical disease that presents with progressive weakness, an ataxic neurological disease, but one that is non-painful. MRI is usually normal with DM, ruling out spinal cord neoplasias or other causes.

Dogs with IVDD usually are painful, and while the disease can be progressive and symmetrical or asymmetrical, it doesn't usually progress in this fashion.

Dogs with spinal cord neoplasia often have asymmetrical progressive signs. They are usually painful with paraparesis. Some may have rapid onset, while others may have a slow onset. Advanced imaging would be necessary to confirm this versus DM.

Infectious meningomyelitis can be caused by various organisms, including bacteria, canine distemper virus, and fungal or protozoal diseases. These can be symmetrical or asymmetrical but usually have systemic clinical signs along with them. CNS tap and advanced imaging help differentiate.

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A five-year-old FS cat presents with abdominal pain, especially after eating, vomiting, a fever, and a slight hint of icterus presents after a three-day history of waxing and waning signs. Bloodwork shows mild leukocytosis and 2.5 bilirubin. You suspect the problem has been going on longer than the owner may have realized. Her GGT and ALP are four times normal. Radiographs are not helpful but show no obstruction. You send the patient for an abdominal ultrasound to a specialist and a 24-hour care facility to confirm your suspicion of cholecystitis and enable the patient to be hospitalized with 24-hour care. Which of the following would not be seen on ultrasound images of a cat with cholecystitis?

Gallbladder wall thickening > 2 mm

Thickening of the common bile duct

Gallbladder wall thickening > 1 mm

Hyperechoic gallbladder

Correct answer: Gallbladder wall thickening > 2 mm

Cats with cholecystitis will often have evidence on ultrasound to confirm the disease. Findings may include:

- 1. Gallbladder wall thickening (> 1 mm in cats and 2–3 mm in dogs)
- 2. Thickening of the common bile duct's wall
- 3. The thickened gallbladder wall may be hyperechoic or have a hypoechoic central area stuck between two hyperechoic areas (layered appearance).

Differentials for gallbladder wall thickening include:

- Edema (hypoalbuminemic patients or those with portal hypertension)
- Cystic mucinous hyperplasia
- Acute cholecystitis
- Rarely, neoplasia

In cats with cholecystitis, we can commonly see biliary sludge, choleliths, and even sludge balls (mobile, non-shadowing, rounded, luminal structures made of bile).

One can obtain an ultrasound-guided sample of the bile for culture (cholecystocentesis).

What is the life expectancy (prognosis) of a cat who develops an abortive FeLV infection?

The same as a cat without FeLV exposure

Protected for life from developing infection

Increased risk of developing FeLV-associated tumors

The same as a cat with regressive or progressive FeLV disease

Correct answer: The same as a cat without FeLV exposure

The feline leukemia virus has a complex pathogenesis. We can see several states associated with infection.

- 1. Abortive infection occurs mostly via the oronasal route. Some immunocompetent cats mount sufficient cell-mediated and humoral immunity to prevent the development of viremia. These cats will have high detectable levels of neutralizing antibodies. Still, they will fail to detect positive viral RNA or proviral DNA in the blood. For several years, though not likely a lifetime, they will have protective immunity against reinfection. Their life expectancy is no different than a cat who has never had/been exposed to the virus. Further, they have no increased risk of cancer development.
- 2. Regressive infection develops when animals mount a sufficient immune response, containing viremia before or just after it reaches the bone marrow. FeLV will spread via the lymphocytes and monocytes throughout the body. At this time, they will be shedding virus and they will be positive on an ELISA antigen test. Some cats may be asymptomatic, while others may show fever, enlarged lymph nodes, and lethargy. For the majority of patients, the viremia will last a few weeks as the virus travels to the thymus, spleen, salivary glands, and lymph nodes. Some of these cats will remain antigen positive. In contrast, others may never show positive on an antigen test, thus, complicating FeLV diagnosis. Some of these patients will develop high levels of viremia after replication in the bone marrow. Still, even some of these cats can clear the viremia in time. They will remain proviral DNA Positive because the DNA remains in the bone marrow. Still, they have a regressive (once called latent) form of the disease. Once this stage is reached, these cats will test negative on ELISA and IFA (antigen testing formats). Infection may reactivate with high-dose steroids or other immunosuppressive conditions, including during pregnancy, causing clinical disease in kittens. The closer to the viremic state a stressor occurs, the more likely the disease will reactivate. How often this type of infection can lead to active FeLV disease is unclear.

have low levels of neutralizing antibodies. They often develop fatal FeLV-associated clinical disease. Most commonly, this is seen in young and immunosuppressed patients.					

What is the primary causative agent and reason for developing neonatal septicemia in calves?

Escherichia coli; failure of passive transfer

Escherichia coli; Fetal hypoxia

Salmonella spp.; septic arthritis

Salmonella spp.; meconium impaction

Correct answer: Escherichia coli; failure of passive transfer

Neonatal septicemia in cattle is most commonly seen in the first two to seven days of life, though it can happen up to two to three weeks after birth. The intestinal mucosa becomes compromised by enteric bacterial colonization and subsequent infection or from another source where infection has arisen, such as a septic joint. However, these animals cannot fully protect themselves and become systemically sick because of a failure of passive transfer. Cattle require colostrum to obtain needed gammaglobulins, as they are born without them due to the complete separation of the maternal and fetal blood supplies. Thus, without this immune protection, they are highly susceptible to invasion by an array of organisms.

Neonatal septicemia, the third most common cause of calf deaths in the U.S., occurs most frequently in cattle with Failure of Passive Transfer (FPT). E. coli accounts for the majority of isolated bacteria. However, Salmonella spp., Campylobacter, Klebsiella, and others have also been identified in cases.

Signs of neonatal septicemia can include the loss or lack of a suckle reflex, fever (or low temp), scleral injection, depression, hypopyon, hyperesthesia, congested mucus membranes, increased capillary refill time, and signs of systemic illness. It is rapidly progressive and fatal without intervention. Animals will become tachycardic and eventually tachypneic. Hypotension and signs associated with decreased/poor cardiac output are likely. Some animals, when terminal, may develop diarrhea, but not all.

Septic arthritis could provide a source leading to systemic sepsis. It may trigger infection, but FPT is the primary underlying cause, with E. coli as the most common species.

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A two-year-old FS DMH presents with an acute onset of ataxia, bradycardia, hypothermia, and conjunctival injection. She was urinating outside of her litterbox, which isn't like her. She was both sleepy at times and then hyperactive at times. She is sleepy but rousable and almost hyperreactive. On physical exam, she is drooling and very vocal. She had some diarrhea in the carrier on the way over. Sadly, you suspect an intoxication based on the owner's presentation, odor, appearance, and behaviors. You ask if there is anything that the pet could have gotten into. The owner stated that she doesn't usually eat things that she shouldn't. She had been with the owner and his friends all day long. You ask to do a drug screen on the cat, and the owner initially hems and haws, then says, "Well, they were smoking weed all day long, and she was in the room." He did see her licking the bong water as well at some point. You explain that you aren't the cops but you need to know how to treat her and appreciate the owner's honesty. You explain that even secondhand smoke can be toxic to pets and that in the future, the cat needs to be in a separate part of the home or away from them when they are smoking. You explain that it is very uncommon in cats but that if she drank water and inhaled secondhand smoke all day as well as previously (as it can be cumulative). The marijuana is the most likely cause. You recommend bloodwork to evaluate parameters for other possible causes and problems and consider radiographs due to her age.

All of the following would be an appropriate therapy for this non-critical patient, except:

Intralipids
IV fluid therapy
Nutritional support
Heat support

Correct answer: Intralipids

Most patients with marijuana intoxication require no treatment — simply rest, minimal stimulation, and supportive care. They need to be removed from exposure and not reintroduced into it. Depending on the severity and degree of debilitation, they may require heat support, IV Fluid therapy, or even nutritional support (though most of these patients, if they are in control of the respiratory system, will eat). Seizure activity, if it develops, can be controlled with benzodiazepines. With marijuana products, intralipids usually aren't required. However, in severe life-threatening intoxications, we can use intralipids (a fat sponge, so to speak), which can help

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A dog presents with acute onset of deafness after initiating treatment for otitis media. All the following medications have been safely used to treat otitis media without causing deafness, except:

Tobramycin
Aqueous ciprofloxacin
Aqueous gentamicin
Nystatin

Correct answer: Tobramycin

No medications are actually labeled for use for the treatment of otitis media. Care must be used when the tympanum cannot be seen and/or is opaque, suggestive of a middle ear infection, or CT confirms this. Deafness may result from various medications.

The safest infusion medications for the ear would be aqueous solutions of ciprofloxacin, marbofloxacin, or enrofloxacin (fluoroquinolones). Additionally, aqueous gentamicin, an aminoglycoside, is considered safe. However, others, like tobramycin, have been associated with severe hearing loss. Further, the semisynthetic penicillin, ticarcillin, has caused deafness. The antifungals are generally safe and include miconazole, nystatin, clotrimazole, and tolnaftate. However, short-term deafness has been seen with clotrimazole or miconazole that resolves with cessation of the medication.

Often, when the eardrum is either absent or not visible (due to stenosis or severe debris), starting with an aqueous solution mixed with a fluoroquinolone +/- a steroid +/- an antifungal mixed in gets therapy going. A different formulation may be feasible once the eardrum is readily visible and the ear begins to heal.

You are seeing a new feline patient for behavior concerns at home. The patient has displayed what the owner terms as aggression towards her and one of the other family members. They are concerned and afraid of getting bitten. You observe the cat and watch for signs of aggression.

All of the following could be considered signs of aggression, except:

Defensive body position	
Hissing	
Growling	
Scratching/biting	

Correct answer: Defensive body position

Technically, all of the answers above can be considered normal cat behaviors. They may occur when stressed or frightened and do not always mean the cat is being aggressive. They may be behaviors that have been reinforced without owners even realizing it. However, a cat simply taking a defensive body posture doesn't mean that the cat is about to be or being aggressive. It is a natural self-defense behavior and response.

Aggressive behaviors can occur as a response to fear, misdirected or inappropriate play aggression, petting-induced or handling-associated aggression, redirected aggression, and aggression secondary to underlying medical conditions, including pain.

Animals that do show signs of aggression may hiss, bite, scratch, or attack.

A blue and gold macaw presents to the pathology service for necropsy after acute death. The bird was in good body condition, but its urates were bright yellow, and there was little feces prior to death. For a day or so before, the owner noticed weakness, regurgitation, and some diarrhea. She has other birds in the home and is concerned it is infectious and about exposure to the other birds. You find non-specific changes and hepatomegaly with a mottled appearance and abnormal color, renomegaly, and splenomegaly. The mesenteric fat and pericardium have both petechial and ecchymotic hemorrhages evident.

You suspect a viral disease and hope to identify intranuclear inclusions in any of the following tissues, except:

Heart
Liver
Pancreas
Intestinal epithelium

Correct answer: Heart

The clinical signs above, the presence of green urates, non-pathognomonic findings on necropsy, and the species should give you clinical suspicion for an infectious cause. You suspect Pacheco's disease, which is a psittacine alpha herpesvirus. In New World species such as this macaw, we can see viral hepatitis. In animals who do not succumb to disease, they will develop internal papillomatosis, most commonly seen in macaws, Amazon parrots, hawk-headed parrots, and conures. Birds can be asymptomatic carriers, shedding virus to those who are uninfected. For birds that are then stressed, as is commonly seen with other species and herpesviruses (cats, people), the disease can flare up.

Transmission occurs by the fecal-oral route, aerosol, or direct contact. Outcomes vary with the virus genotype, the bird's overall health status, and the species infected.

Once infected, even if clinical signs resolve, birds will be forever carriers and persistently yet intermittently shed for life.

Clinical signs include sudden death with normal body condition, bright yellow urates, and minimal feces. However, this can be preceded by green urates, lethargy, weakness, depression, regurgitation, or diarrhea.

For the birds the owner has at home, a DNA probe combining both oral and cloacal swabs can be used. Bloodwork may show leukopenia and elevated AST.

Intranuclear inclusions are histologically evident in the pancreas, spleen, liver, and intestinal epithelium, not the heart.

Treatment consists of supportive care. Acyclovir has been used as well, but each time a bird is handled and stressed, this increases the risk of transmission. An inactivated vaccine or autogenous vaccine can be used.

If birds develop papillomatosis, these lesions predominate in the oral and cloacal mucosa, but may be internal as well (GI tract, bursa, or conjunctiva). Sometimes owners fear a cloacal prolapse when part of the papilloma is peaking out of the vent. Treatment at this stage is supportive, and if lesions are bleeding/ulcerative, treating any secondary infections that arise may be needed.

Viral typing to determine outbreak serotypes for Infectious Bronchitis Virus (IBV) in poultry helps differentiate between wild infections and vaccination strains. What method to identify IBV serotypes is highly accurate but time-consuming and costly?

Molecular detection of the viral spike (S1) gene
Virus neutralization tests
Immunohistochemistry
PCR

Correct answer: Molecular detection of the viral spike (S1) gene

IBV is a worldwide avian coronaviral disease, with chickens as the primary host, though the disease has been seen in peafowl and pheasants. It is transmitted via inhalation and direct contact, and morbidity in birds is usually 100%. Various strains lead to different clinical pictures, including decreased egg yields, poor egg quality, upper respiratory tract disease, andacute nephritis. Respiratory lesions are not granulomatous but may contain serous, caseous, or catarrhal exudates when advanced and are foamy within the air sacs, then develop cloudiness as the disease progresses.

The preferred diagnostic test molecularly detects the viral spike (S1) gene. Once the S1 gene is identified, a lab can further perform sequence analysis to define the serotype. This is time-consuming and costly.

Less expensive methods of identification, without identifying the serovar-specific strain, include Reverse Transcriptase Polymerase Chain Reaction (RT-PCR), assessment of rising antibody titers against IBV preclinical to convalescent sera, or virus isolation in embryonated eggs.

If you are collecting samples from a large flock, be sure to collect samples from animals with and without clinical signs. Signs generally develop three to five days post-infection, when peak titer is no longer evident.

A 12-year-old FS DSH has a history of stable CKD (IRIS stage 2). Her most recent bloodwork shows mild hyperphosphatemia. What is the appropriate treatment recommendation for this patient pertaining to phosphorus regulation?

Phosphate-restricted diet

No therapy is needed in IRIS stage 2

Phosphate binders (oral)

Senior formulation of an over-the-counter diet

Correct answer: Phosphate-restricted diet

The best way at this stage to address phosphorus is by restricting intestinal phosphate absorption. This is most easily done via phosphate-restricted diets, which is accomplished by feeding a lower-protein, low-phosphorus diet. The IRIS staging helps provide target-specific phosphorus levels to shoot for and ways to control it. But be mindful that the body may take time to adjust total phosphorus levels because of whole-body phosphate retention. It may take two to four weeks for plasma phosphate concentrations to truly reflect the disease state. If cats at this stage of the disease won't take to the renal diets (low phosphate foods), a phosphate binder may need to be started sooner rather than later.

A five-year-old FS Pug presents to you on an emergency for respiratory distress, called as a stat. The triage nurse goes up front and finds a happy pug wagging his tail, snorting away as many brachycephalic dogs do, and panting. He is not in any overt respiratory distress. You ask the owner what the problem was, and she says he was making a god-awful noise that must have lasted minutes and was non-stop. He looked scared out of his mind and would die any second. It passed, but she still brought him in to see you. The nurse tells her to Google reverse sneezing pug online to get sample videos of what this is, and sure enough, it was exactly that. You offer to have the doctor still examine him in case there is something in the nose that triggered it or something else ongoing, such as an allergic reaction or other airway issues, given the breed. Still, the owner declines, and he has been triaged away. The technician properly puts notes in the computer of the encounter, and the client declines a doctor's evaluation.

All of the following could have contributed to this infrequent occurrence, except:

Laryngospasm

Elongated soft palate

Allergens such as pollen, chemicals, perfumes

Foreign body in the throat (grass, foxtail)

Correct answer: Laryngospasm

A protective reflex, laryngospasm, occurs, causing the laryngeal cartilages to rapidly close, spasm, and flutter. It can be triggered by irritants such as anesthetic gasses or positive continuous airway pressure. We commonly see it in veterinary medicine when trying to plate an endotracheal tube in cats more commonly than dogs. It can occur if the patient isn't fully anesthetized while trying to intubate and or you are forcing intubation. Brachycephalic dogs may be at increased risk.

Conversely, reverse sneezing is paroxysmal sneezing that occurs as a forceful, inspiratory airflow. It is most commonly in response to allergic or mechanical irritation to the nasopharynx or posterior nasal cavity. When occurring, animals hold their heads normally or have them thrust back some. It sounds like the dog is inhaling a sneeze, hence the name. It can last seconds to minutes. Basically, it is a spasm. The dog may extend the neck and expand the chest to inhale harder.

Causes include anything irritating the throat, though no obvious inciting cause is often readily evident. Things like excitement (especially in brachycephalic breeds), drinking (especially too fast), eating, leash pulling, pollen, and foreign bodies that get caught

in the throat, like grass awns, grass, or related items. Further, irritants like household chemicals (think bleach), perfumes, some viruses, and even simple post-nasal drip from an upper respiratory or lower respiratory tract infection. Brachycephalic breeds or others with elongated soft palates may inadvertently trap it in the throat and trigger an episode.

Usually, it is a minor issue, though scary for dogs and pet owners. If it becomes a chronic problem, there could be something stuck in the nasal passage or a mass or other problem, and scoping may be warranted.

Cats rarely develop this condition. However, a consideration to think of if a cat presents with this would be feline asthma. That "sneeze" may really be a cough that the owner misinterprets as a sneeze/reverse sneeze or truly is a reverse sneeze.

A four-month-old kitten FI presents with a history of vomiting, and radiographs show foreign material in the stomach with a non-obstructive intestinal pattern. Ultrasound confirms a foreign body in the stomach. You elect to attempt endoscopic retrieval, though caution the owner that it may not be feasible depending on the object type and size. (The owner has no clue what it could be.) The owner understands that a laparotomy/exploratory may still be needed. Still, you hope to be able to avoid abdominal surgery using the scope.

All of the following basic principles apply to the endoscopic retrieval of a foreign body, except:

Once the object has been snared, pull out against undue resistance

Use an overtube if the object has sharp edges or to dilate the lower esophageal sphincter

Reorient the object if needed before its removal

Always re-radiograph right before anesthetic induction

Correct answer: Once the object has been snared, pull out against undue resistance

For foreign body removal, each patient is different, and each procedure is different. However, a few basic principles should always be followed regardless of the patient:

- Ensure you re-radiograph before inducing to ensure the foreign material hasn't moved aborad. It is now out of reach of the scope or hasn't caused perforation or other abnormalities.
- 2. Don't just grab and pull. Often, the object needs to be repositioned, permitting the forceps to get the best grasp on it. Further, one may need to reposition it to ensure it is easily removable through the sphincters.
- 3. Select appropriate instruments. Use retrieval forceps that permit the firmest grasp.
- 4. Once an object has been snared, do NOT pull against undue resistance. We expect high resistance associated with the lower esophageal high-pressure zone, in the gastric cardia, at the base of the heart, the cricopharyngeal area, and the thoracic inlet. If too much resistance is appreciated, stop, reorient the object if needed, and begin again. You may even need to release it and re-grab or use hoods or overtubes to assist.
- 5. Overtubes can be used when sharp or irregular edges are present or to help you dilate the lower esophageal sphincter.

A new worker started one month ago. He is being trained on calf rearing, care, and other related needs. He has noticed behavioral changes in some of the bottle-fed calves, muscle tremors, depression, and one had a seizure today. He is concerned and tells his immediate supervisor, who contacts the veterinarian. When obtaining your history, you want to ask questions about findings and concerns. You may ask additional questions after your evaluation. Your evaluation shows several calves that are lethargic and drooling. Some are aggressive, while others seem ataxic and knuckling. They are PU/PD and appear to have abdominal pain. One is found head-pressing in the corner and vocalizing. You suspect something environmental may be contributing.

All of the following questions would be helpful to identify the cause, except:

Are soil magnesium levels low in the area?

Have any insecticides such as organophosphates been used around the animals recently?

When mixing the milk formula, is the water used softened?

Do the calves have access to any brackish water?

Correct answer: Are soil magnesium levels low in the area?

Remember, these are nursing calves, though they are being bottle-fed. These neurological signs and related issues suggest a toxin, like an insecticide or salt toxicity. Salt intoxication manifests when there is limited access to fresh water either because of overcrowding, weather issues, mechanical issues, or the freshwater available is brackish. Salt lick blocks can contribute. Softened water can be higher in salt and shouldn't be used to mix milk replacers until checked to ensure that concentrations fall below 100 ppm. Dehydration and hot weather can also increase the risk of intoxication.

Differentials for these animals include carbamate or organophosphate toxicity. Thus, knowing if these products were used near/around the calves is important. Other causes may include polioencephalomalacia, lead poisoning, rabies, or staggers (hypomagnesemic tetany). Animals not on pasture and with properly formulated milk shouldn't have issues with low magnesium levels. Whether the pasture has magnesium is irrelevant since they are currently bottle-fed, and their nutrition source is milk.

A client's lawn service inadvertently sprayed the grass and plants in the backyard without contacting the homeowner to alert her, despite a contract stating that only the front is to be sprayed because of your animals. You have several acres, all fenced in with dogs, horses, and goats. You weren't aware that they had sprayed until it was too late. You came home from work and let your dog outside only to find then that there was a bill, noting that they had been there, on your front door knob. Unbeknownst to you, they were using chemicals that are toxic to animals. Thankfully, the dog was only briefly exposed, and the other animals had not been removed from the barn. But your dog was showing signs of agitation, anxiousness, and ataxia. Further, he was drooling, had one episode of vomiting, and his pupils seemed smaller than normal. The owner brought the pet immediately to you at your office. After getting a brief history and seeing the clinical signs, you administer a test dose of what medication based on your suspicions on the type of chemical used?

Atropine
Pralidoxime chloride (2-PAM)
Glycopyrrolate
Activated charcoal

Correct answer: Atropine (+/- Pralidoxime chloride (2-PAM))

Based on clinical signs, you presume the toxin to be either an organophosphate or a carbamate product. Ideally, 2-PAM and atropine would be administered in the face of organophosphate intoxication. However, since we do not yet know if it is an organophosphate or carbamate product, we will start with atropine. Both of these are commonly used as pesticides and insecticides. They work by binding irreversibly (organophosphates) or reversibly (carbamates) to acetylcholinesterase within the muscles and tissues of the nervous system. This allows acetylcholine to build up, causing continuous stimulation (cholinergic). As a result, it causes overstimulation of various synapses, including the central, nicotinic, and muscarinic, accounting for the wide array of clinical signs.

The parasympathetic signs we see are based on the acronym SLUDGE. This refers to Salivation, Lacrimation, Urination, Defecation (diarrhea), GI distress, and Emesis that occur as a result. These are reversible with atropine sulfate (controlling the parasympathetic side effects). Titration and dosing should occur until the pupils are sufficiently dilated, salivation stops, and the patient is more with it. This medication doesn't address those side effects related to the cholinergic nicotinic impact, such as muscle tremors/fasciculations or paralysis.

Glycopyrrolate is also an anticholinergic/antimuscarinic-like atropine. However, it doesn't cross the blood-brain barrier and has a slower onset of action than atropine, though it does last longer. It is an alternative if atropine is not available, but generally, protocols against these toxins utilize atropine over glycopyrrolate.

2-PAM is an antidote for organophosphate toxicity, acting as a cholinesterase reactivator. It must be administered within 24–36 hours to be effective. It is not recommended for carbamate poisonings, so you may want to withhold it until the owner contacts the company to determine the product used. That being said, sometimes products can be a mixture of organophosphates and carbamates, and so, if the owner will not be able to get a hold of the company for 24+ hours, then it may be reasonable to presume that it includes an organophosphate and give the medication since delay can worsen prognosis.

Activated charcoal is being used less in less by ASPCA poison control. Depending on the degree of exposure and route (topical, dermal vs. oral ingestion), this may depend on additional therapies recommended for this patient. Removing them from the source is important. But activated charcoal will not address any of the potentially lifethreatening issues. It will not treat any of the clinical signs. It can be considered once the pet is stable, not vomiting, and alert enough. Contact poison control if in doubt. Since we know this patient's exposure was within a short time frame, it may be warranted once stable.

A five-year-old FS DSH presents for repetitive, compulsive behaviors, including tail-chasing and pacing. She had a full workup at the primary care veterinarian. No pain was identified, and bloodwork and radiographs were non-remarkable. There was no evidence of arthritis. She has no vomiting or diarrhea. She is not declawed, and she has no obvious underlying medical conditions. You are moving on to behavioral causes of the tail chasing and pacing. When reviewing incident information with owners, you want to discuss the ABCs of the behavior.

All of the following are appropriate questions pertaining to the A of behavior, except:

What happens immediately after the behavior ceases?

Was there a sudden, loud noise before the behavior?

What time of day does the behavior occur? Is it always at the same time?

Did the animal just come out of the litterbox and get into a fight with the other cat?

Correct answer: What happens immediately after the behavior ceases?

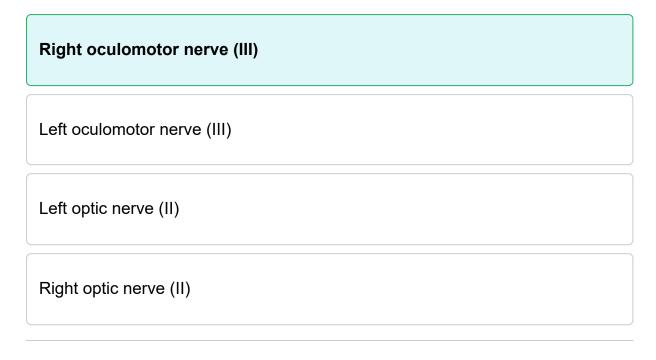
The ABCs of behavior refer to the antecedents, behaviors, and consequences.

Antecedents may be environmental triggers or owner-initiated behaviors that trigger the event.

- 1. Was there suddenly a loud crashing sound?
- 2. Does it only happen at night when the owner is getting ready for bed?
- 3. Was the animal sleeping right before the behavior started or pacing? Was it vocalizing?
- 4. Was the animal just fighting with another pet in the house?

"B" refers to what the behavior itself is, and "C" refers to what happens directly after the behavior ceases, and what consequences if any occur as a result of the behavior (i.e., does the owner give the cat love and affection or scream and yell at it?).

A five-year-old FS Ragdoll presents to the ER with a 24-hour history of anisocoria. Your ophthalmic exam shows that OD is non-responsive to light and mydriatic. Further, the right eye does not constrict when you shine light into OS. When you shine a light into the right eye, the left eye responds appropriately, constricting in response to an indirect Pupillary Light Reflex (PLR) test. The rest of the patient's physical exam is normal. You localize the problem to which cranial nerve?



Correct answer: Right oculomotor nerve (III)

Pupillary constriction occurs via the oculomotor nerve (III). Since the PLR is missing on the right eye but present when light is shone into the right eye on the left (indirect PLR), and directly, with light directly into the left eye, the lesion appears to be affecting the third cranial nerve on the right, the oculomotor nerve.

Cranial nerve II (optic nerve) is the afferent arm for the PLR and vision, which results in sensory transmission to the brain. Alternatively, cranial nerve III, or the oculomotor nerve, makes up the efferent arm of the PLR while also innervating many of the periocular muscles. Recall that afferent neurons carry information toward the CNS, while efferents carry info to the periphery (away from the CNS).

A ten-year-old MN DMH presents for non-respiratory illness, but the owner reports a history of chronic intermittent coughing. They are not concerned with the coughing as it hasn't affected the pet's quality of life. You take radiographs (thorax and abdomen) because the cat is dehydrated from vomiting on and off for the past 24 hours, needs IV fluids, and has a II/VI holosystolic murmur. You want to ensure no evidence of cardiomegaly before instituting rehydration therapy. Your chest radiograph looks like this.

What is your top differential for the pulmonary findings?

Atelectasis
Neoplasia
Pulmonary thromboembolism
Pneumonia

Correct answer: Atelectasis from chronic asthma

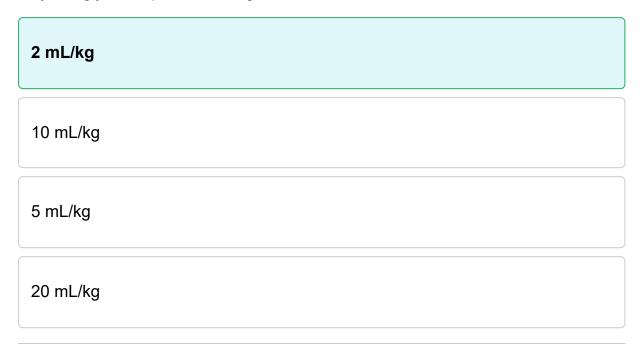
This patient's radiographs show chronic evidence of asthma. Atelectasis or lung collapse can occur due to asthma. The pulmonary pattern shown here is a diffuse, moderate broncho-interstitial pattern with suspected mucous plugs. The cat's chronic cough has been causing more problems for the pet than the owners have appreciated.

There is no evidence of metastatic or nodular disease.

Common reasons for atelectasis in the cat include asthma or Pulmonary Thromboembolism (PTE). Atelectasis most commonly occurs in the right middle lung lobe. It happens due to the accumulation of mucus within the bronchus. Patients with pulmonary thromboembolism often have significant clinical signs, including dyspnea, tachypnea, and depression. It is unlikely to be an incidental finding, as atelectasis can be in asthmatic cats.

The pulmonary pattern here most closely resembles asthma or bronchitis (chronic in nature) though atypical pneumonia cannot fully be ruled out. Often, animals with PTE have normal radiographs or non-diagnostic changes. However, given the history of coughing for years, the lack of respiratory signs, and the presumptive mucus plugs, chronic asthma is the most likely cause.

You are asked to evaluate a group of cattle. Several are showing depression, ataxia, and weakness. Some have been drinking excessively, and others are not urinating at all, or are doing so minimally. You review the environment and find that the area between the milking barn and the pastures recently had a vehicle breakdown, it is winter, and you suspect ethylene glycol intoxication. Several animals have evidence of acute kidney disease on bloodwork, and many have evidence of calcium oxalate crystals in the urine. You suspect ethylene glycol toxicity. You note that nursing calves are much worse than those who have been weaned. What is the toxic dose of ethylene glycol for pre-ruminating calves?



Correct answer: 2 mL/kg

Ethylene glycol intoxications are uncommon in livestock. However, it can be found in antifreeze, transmission/brake fluids, and de-icer products. The producer admits that a vehicle broke down and had to replace the brake fluid. They couldn't get the vehicle moved for 48 hours because of a storm that came through, and they then had to use a de-icer to gain entry because everything had frozen solid. Though they hadn't appreciated any chemical spills, you suspect that is what happened. The producer didn't realize the product was toxic, providing a great opportunity for education and discussing farm security and management improvements.

Prognosis is better for animals who ruminate than those who do not; thus, a smaller dose is required to cause clinical disease for young calves not yet ruminating. They have a worse prognosis, even with treatment. For non-ruminating calves, the toxic dose is 2 mL/kg, while for adult cattle/ruminating animals, it is 5–10 mL/kg.

A seven-year-old male neutered hunting lab has a healthy history except for a recent lameness. he is on Carprofen at a higher dose than standard. He is on 3 mg/kg BID. He presents to the ER today with lethargy, vomiting, minimal appetite, pu/pd, and belly pain. Further questioning of the owner revealed that the dog was drinking a ton. Still, his urination has dramatically decreased in the past 36–48 hours. He is painful on palpation in the mid-cranial abdomen. A fast scan shows no free fluid but an enlarged right kidney. His PCV/TS are elevated. His bloodwork showed moderate azotemia, metabolic acidosis, normal to high potassium, and elevated phosphorus. He had glucosuria despite a normal serum glucose level. He also had a historically elevated alkp in the 300 range, which is now in the 700 range; His ALT was two times normal. His total bilirubin was normal. He had mild muscle stiffness, didn't want to move on to the physical exam, and was about 5% dehydrated, you estimate.

What is one of your top differentials for this patient, and which of these causes is a reason for acute kidney injury?

Leptospirosis; Intrinisic renal disease

Leptospirosis; Decreased renal reserve

Chronic liver disease; Decreased renal perfusion from prerenal causes

Pyelonephritis; Post-renal etiologies

Correct answer: Leptospirosis; Intrinisic renal disease

The dog in this example is classic for leptospirosis. He has an acute kidney injury (AKI) due to intrinsic renal disease. Intrinsic disease can manifest as either glomerulonephritis or interstitial nephritis.

AKI can occur for three main reasons:

- 1. Decreased renal perfusion secondary to prerenal etiologies, such as dehydration, hypotension, or low circulating blood volume, as can be seen with chronic liver disease or heart disease. This patient has no notes suggesting any primary heart disease, and elevated liver values in chronic liver disease often include additional values, such as AST or GGT. They may have changes in glucose levels or other parameters. Further, the change in urination and metabolic acidosis suggests that, while there may be a degree of dehydration and prerenal component, the primary concern is kidney damage, an intrinsic renal disease.
- 2. Intrinsic renal disease would be secondary to inflammation (nephritis) or tubular necrosis (nephrosis). We see nephrosis as a consequence of toxic injury,

ischemic damage, or trauma. For example, toxins such as ethylene glycol or aminoglycosides can lead to nephrosis. On the other hand, nephritis is either glomerular or interstitial in nature. Commonly recognized causes include leptospirosis, Rocky Mountain spotted fever, ehrlichiosis, bacteremia/septicemia, and Lyme disease.

3. For post-renal causes think of the blocked cat or dog with a urethral stone that cannot urinate. Animals hit-by-cars with bladder ruptures would also fall into this category.

Chronic renal disease factors include a decreasing renal reserve with insufficiency leading to complete loss of kidney function (IRIS stage 4 chronic kidney disease [CKD]).

Leptospirosis is a zoonotic, global spirochete infection of mammals. General clinical signs in animals include fever, not wanting to move, anorexia, pu/pd, d/v, lethargy, abdominal pain, +/- icterus, or respiratory challenges. Key differential diagnoses include nephrotoxicosis (NSAID toxicity), Lyme, acute glomerular disease, cholangiohepatitis, pyelonephritis, canine monocytic ehrlichiosis, sepsis, leishmaniasis, or Rocky Mountain spotted fever.

Humans with leptospirosis develop mild flu-like illnesses all the way to life-threatening multisystemic diseases. Dogs are accidental hosts to Leptospira species. Organisms are shed in the urine, and transmission occurs through contact with the urine of infected animals. Several serovars have been associated with the disease, and vaccinations vary depending on what serovars are covered. Though increased disease occurrences may manifest in the rainy seasons or after floods, we are increasingly seeing this not just in rural areas, but also in cities.

Initially, dogs may demonstrate a high fever, which is often transient, with shivering and moderate muscle aches, +/- uveitis. Additionally, dogs may develop pulmonary hemorrhage and lung involvement. At the same time, others may have significant liver disease to the point of liver failure. Dogs with lepto may have increased bleeding tendencies resulting in hematuria, hematemesis, epistaxis, petechiae, or melena, though uncommon in the U.S. Any dog with elevated kidney values +/- liver values should be evaluated for Lepto.

Diagnostics for this pet should include CBC, chemistry, coagulation tests, and urinalysis (can have varied urine concentrations and variable proteinuria, bilirubinuria, and glucosuria). Radiographs may show pulmonary changes if lepto is invading the respiratory tract. Otherwise, rads may be non-remarkable. Ultrasound can show renomegaly, cortical thickening, pyelectasia (mild), increased renal cortical echogenicity (bright kidneys), a medullary band within the kidneys, and may have some perirenal fluid accumulation.

The testing culture is not generally effective because the organism is very slow-growing and insensitive. Serological diagnostics have significant limitations. Currently, the MAT (Microagglutination Test) is the primary means of detection. Several bedside urine tests using ELISA are now available. The Witness Lepto test can differentiate between wild strain and vaccine strain, while the SNAP lepto test cannot.

Treatment is with antimicrobials, fluid therapy, and supportive care. A urinary catheter should be placed to permit proper measurement of urine ins and outs and minimize the risk of contamination and exposure to staff). Proper protective equipment, including gloves, eye protection, and disposable gowns, should be worn when handling the patient. Handwashing pre- and post-handling is paramount. Those who are pregnant or immunosuppressed should not care for the patient. Regularly disinfect all areas that come in contact with urine. Proper isolation and/or signs noting the zoonotic contagious risk of the patient to others.

A cat presents with a skin tumor on the top of her dorsum. The mass is small, about 1 mm in diameter, raised, and densely pigmented. You remove it and biopsy it, which comes back as the most common skin tumor in cats. The biopsy reported clean margins. What is the prognosis for this patient?

Curative; non-malignant trichbolastoma

Follow-up recommended with oncology; malignant basal cell carcinoma

Curative; Sebaceous adenoma

Locally invasive, recommend follow-up with oncology; apocrine gland carcinoma

Correct answer: Curative; non-malignant trichbolastoma

The most common skin tumor in cats (15–26% of feline skin tumors) are histologically noted to be trichoblastomas. These are considered basal cell tumors, and less than 10% are malignant in cats, so surgery should be curative for this patient.

Malignant basal cell tumors in cats are uncommon but have a tendency towards lymphatic metastasis if present. Thus, follow-up with an oncologist may be warranted. Lymph node aspiration or biopsy may also be considered.

Dogs commonly get sebaceous adenomas, which, while benign, do not usually occur in cats.

Apocrine gland carcinomas (arising from sweat glands) are rarely malignant but can become aggressive if transformed into a carcinoma. Malignant lesions are usually found on cats' heads, while benign lesions may be on older felines' bellies, limbs, or heads.

A four-year-old MN DSH presents for evaluation of the feet. On the cat's hind legs, though not painful, he has several soft footpad area swellings. However, the foot pad symmetry is maintained. As of yet, there is no ulceration. The pads seem slightly purplish in color. You discuss diagnostic options with the owner and differentials, including cancer, eosinophilic granuloma complex, something infectious, or other causes of pododermatitis, though uncommon in the cat. You discuss that the treatment varies depending on the diagnosis and recommend a work-up. The owner elects to start with cytology. Cytology shows plasma cells and lymphocytes. Full bloodwork shows an elevated globulin but is otherwise non-remarkable. The cat is FeLV/FIV negative. You state that you still cannot rule in or out specific diseases and recommend a biopsy. The owner opts to hold off and hopes the condition regresses spontaneously. However, she returns in a week because the lesions are now ulcerating and painful, and the popliteal lymph node on the right hind leg is now enlarged. The owner consents to biopsy, and you find plasmacytic dermatitis at the dermis layer and in the adipose tissue. There is edema within the adipose tissue with congested and prominent vasculature. There is erosion/ulceration and exudate in the epidermis, which is acanthotic. Secondary infection is evident by neutrocytosis. This suggests that this cat has what condition?

Plasma cell pododermatitis
Neoplasia
Infectious granuloma
Eosinophilic granuloma complex

Correct answer: Plasma cell pododermatitis

This is a classic presentation for plasma cell pododermatitis. Etiology isn't elucidated. Lesions in some individuals spontaneously regress (though not usually once a secondary infection has developed). Initially, animals will have foot pad swellings, which can be white with cracks. Swelling is soft and initially not painful until they become ulcerative. The lesions can be purple, as can the foot pad in general. In more advanced cases, regional lymphadenopathy may occur. Cytology may show significant numbers of plasma cells but doesn't fully confirm the diagnosis. Biopsy, as described above, does. Treatment is case-based as we do not have evidence-based medicine to address this condition. Initial therapy is recommended to start with doxycycline at 5–10 mg/kg q 12–24 for 30 days past resolution, then wean to the lowest effective dose +/- steroids at 4.4 mg/kg/day for a month after the resolution, then decreasing to the lowest effective dose. Some animals require lifelong therapy to

	recurrence. If severe ulceration occurs, animals may require surgical ent to help improve wound healing.
chlorambu to three m	ve doesn't work, suggested next steps include chrysotherapy or ucil. Most cases with doxy respond in two to three weeks, but it can take up nonths to see full improvement. Recurrence is possible, even for those that n their own.
Other pos granuloma	ssible differentials include cancer, infectious granulomas, or eosinophilic a complex. However, cytology and biopsy help differentiate these entities.

A young foal, seven days old, presents to your emergency hours with a history of acute lameness. You evaluate the foal and appreciate that the right forelimb is warm to the touch, and there is swelling about the carpus. There is evidence of effusion. The joint feels a bit unstable. The foal has had a fever and has been lethargic for the past 12–24 hours. On evaluation, you find no external evidence of a wound. However, the client informs you that just after birth, the foal did develop omphalitis that appeared to respond to treatment well. Radiographs show a widening of the physis and radiolucencies in several carpal bones. You perform arthrocentesis on the affected joint. The results are as follows: The total protein is> 4 g/dL; white blood cell count is > 30,000; neutrophils account for over 80%, and many have degenerative changes. What is your clinical diagnosis for this foal?

Septic arthritis of the carpus
Non-septic arthritis of the carpus
Septic physitis
Cellulitis

Correct answer: Septic arthritis of the carpus

Acute lameness in a foal is a true pediatric emergency. While owners often assume the mare stepped on the animal, this tends to be uncommon. The number one cause of acute neonatal lameness in foals is osteomyelitis/septic arthritis. Differentials include physeal fractures, foot abscesses, muscle/tendon injuries, or long bone fractures. Additional causes of acute lameness in the foal may include hemarthrosis, cellulitis, flexural deformity, ruptured tendon, subsolar/solar bruising, peripheral nerve injury, or laminitis.

Perform a thorough physical examination. If joint effusion is present, presume it to be septic, though remember palpation of effusion can be difficult in the shoulder, hips, and elbows and could be missed on physical exam. Obtain radiographs to determine changes and evaluate for fractures and other abnormalities. Diagnosis of septic arthritis requires radiographs, arthrocentesis, and cultures.

Arthrocentesis:

- Normal values: TP < 2.0 g/dL; WBCs < 1000 cells/mL; neutrophils < 40%
 Septic arthritis: TP > 3 g/dL; WBCs > 20,000 cells/mL; neutrophil counts > 80% +/- degenerative changes
 - 3. Septic physitis, while outside the joint, can still lead to a sympathetic joint

effusion which will have mild to moderate TP increases, variable WBCs, and percent of neutrophils

Ensure effusion is cultured and perform a gram stain. 50% of cultures are negative. But this doesn't mean that the joint isn't septic. 25% of negative cultures will be positive on gram stain. Given the history, obtaining a culture from the umbilical area may also be beneficial in this foal.

Foals can be predisposed to septic arthritis secondary to a failure of passive transfer, prematurity/dysmaturity, poor foaling hygiene, placentitis in the mare, retained fetal membranes, dystocia, or any condition causing septicemia/bacteremia.

Signs of septic arthritis may be simply an acute severe lameness +/- weight bearing +/- more than one joint affected. The joint may be painful, swollen, and or hot to the touch. There may be pitting edema and peri-articular swelling. Painful animals may have elevated heart and respiratory rates. They may or may not have a fever. Some may have increased periods of recumbency and decreased nursing frequencies.

Treatment of septic arthritis includes broadspectrum antibiotics, local drainage and lavage of the joint(s) affected, and local antimicrobial therapy. Adjunctive therapy includes pain management and anti-inflammatory medications. Prognosis is guarded and worse if there is a concurrent systemic illness; multiple joints are affected; osteomyelitis is present, especially if it affects the weight-bearing surface; and if Salmonella species are cultured.

You have a cat who presents for referral for a rhinoscopy because of chronic intermittent nasal discharge that is not fully responsive to antivirals or antibiotics. All of the following would be indications for rhinoscopy, except:

Identify diseases in the mid-nasal area

Identify and remove a nasal foreign body

Biopsy/cytology of mass lesions/mucosa

Identify source of epistaxis or chronic nasal discharge

Correct answer: Identify diseases in the mid-nasal area

Cats with feline herpes or caliciviruses are at risk for developing chronic upper respiratory disease, including chronic rhinitis and sinusitis.

Rhinoscopy is very helpful for a number of reasons, including:

- ID and remove a nasal foreign body
- Biopsy/cytology of mass lesions/mucosa
- ID source of epistaxis or chronic nasal discharge
- Investigate possible causes of nasopharyngeal disease and acute/upper respiratory tract signs, as in this patient
- Permits mucosal nasopharyngeal evaluation and biopsy samples of the nasopharynx but not deeper tissues or sinus evaluation

However, while non-invasive to a degree, if there are any underlying bleeding disorders, it is contraindicated due to the high vascularity of the nasal cavity. Further, if sinusitis is also suspected, not just chronic rhinitis, a CT is likely warranted in addition to the rhinoscopy to classify/determine the extent of damage to the nasal turbinates and the accompanying sinuses. Often, the CT is performed during the same anesthetic event as the rhinoscopy. Finally, if the nasal discharge is mucopurulent and extremely thick, this may limit visibility and prohibit complete examination via rhinoscopy.

Disadvantages include missing mid-nasal area disease, the high cost of the equipment, and failure to detect any pathology underneath and deep into the mucosa.

A three-month-old FI DSH presents to the ER 15 minutes after she snatched her owner's calcium channel blocker that the owner had accidentally dropped on the floor. The kitten is in a phase where everything seems to go in the mouth, and before she can even bend down to look for it, that cat has snatched it up. Thankfully, the owner lives close, and the kitten shows no clinical signs. You want to induce vomiting.

Which of the following is the most effective means to induce vomiting in a feline patient?

Dexmedetomidine 7 µg/kg IM

Apomorphine 0.03 mg/kg IV

3% hydrogen peroxide 2.2 ml/kg or 1 ml/# orally

Ropinirole (Clevor®) 3.75 mg/m² in the eye

Correct answer: Dexmedetomidine 7 µg/kg IM

Cats who ingest calcium channel blockers are at risk of developing bradycardia, hypotension, heart block, non-cardiogenic pulmonary edema, or GI upset. Thus, it is crucial that we induce vomiting before clinical signs manifest.

Dexmedetomidine is the only safe method that can somewhat reliably induce vomiting in cats when administered IM in the shoulder area. If no emesis occurs after 15–20 minutes, Dr. Justine Lee, a veterinary criticalist and veterinary toxicologist and the co-founder of VetGirl, recommends giving a 3.5 μ g/kg IM dose. This drug is beneficial because even if the patient becomes sedated after emesis, it is reversible with atipamezole.

Hydrogen peroxide is contraindicated in cats due to a high risk of developing gastritis or hemorrhagic gastritis (higher risk than in dogs).

Clevor® is a dog-only prescription topical anti-emetic.

Apomorphine is not generally used in cats. It often causes an excitatory reaction, is unpredictable, and is usually unsuccessful in inducing vomiting.

A nine-year-old FS DSH presents with mammary masses for evaluation. There are several clustered around the third gland on the right. You have recommended referral to a surgeon for a unilateral mastectomy, but the owners declined and requested only lumpectomies. You discuss that this isn't ideal given the metastatic rate of mammary cancer in cats and that more than one mass is already present. Lumpectomies only increase the risk of metastasis and reoccurrence without complete mastectomy of that side. The owner understands but has financial concerns and insists this is what she wants. You are preparing for surgery and review what vessels you need to look out for near gland three. These are?

Cranial superficial epigastric vessels

Caudal superficial epigastric vessels

Ventral branches of the intercostals, internal thoracic, and lateral thoracic vessels

Lateral branches of the intercostals, internal thoracic, and lateral thoracic vessels

Correct answer: Cranial superficial epigastric vessels

Major blood vessels to be mindful of when performing surgery associated with the mammary glands of dogs and cats are as follows:

Glands 1 and 2:

Lateral and ventral branches of the intercostals, internal thoracic, and lateral thoracic vessels

Glands 2 and 3:

Cranial superficial epigastric vessels

Glands 4 and 5:

Caudal superficial epigastric vessels

Ideally, in cats, you want to remove the entire chain. This owner is tying your hands. Performing single lumpectomies is not in the patient's best interest as cancer spread has likely occurred already. Ideally, removing the second and third chain and tissue close to the fourth would be most beneficial if not a complete mastectomy.

A very smelly 19-month-old MN Basset hound presents to the office to evaluate his skin. He is not pruritic at all but has very greasy skin, scales, crusts, comedones, and follicular casts, primarily along his dorsum. He hasn't had a history of skin issues in the past. He hasn't had a diet change, no new foods, no recent move or travel outside the area. Which would be a reasonable differential given the dog's breed, age, and clinical findings?



Correct answer: Primary idiopathic seborrhea

Primary idiopathic seborrhea is a keratinization disorder. It is commonly found in American Cocker spaniels, English Springer spaniels, Bassets, Westies, Dachshunds, labs and goldens, and German shepherds.

Given the age of onset and severity of signs yet with no pruritus, this rules out sarcoptic mange, a severely pruritic mite infection. Demodicosis can cause pruritus though it doesn't always, and it should be ruled out in this patient. There is likely a secondary infection (pyoderma/dermatitis) contributing to the scaling and crusting.

While secondary infections are likely present at the time of diagnosis, Malassezia usually causes intense pruritus and is unlikely in this patient.

Thus, the lack of pruritus helps exclude scabies, allergies, and other itch-producing diseases. Because there are no systemic signs, and given the age of the pet, endocrine causes are unlikely.

However, the diagnosis of primary idiopathic seborrhea isn't made until all other possible causes have been ruled out.

Treatment is symptomatic. Treating underlying infections and synthetic vitamin A derivatives (retinoids) may be helpful. Other topical therapies, such as with douxo seborrhea spot-on or spray or similar shampoo products, may be beneficial in minimizing oiliness and lessening the chance of secondary infections.

You are visiting an alpaca farm and have been asked to do pregnancy checks on the hembras (female alpacas). While you are there, you are also asked to observe the males' behavior and make suggestions about the herd makeup. Some can be evaluated without sedation, such as the more social and interactive alpacas. But others are simply too flighty and need sedation.

Which of the following protocols will allow sedation without recumbency?

Xylazine 0.1-0.2 mg/kg, IV

Xylazine 0.3-0.4 mg/kg, IV

Ketamine 4 mg/kg, xylazine 0.4 mg/kg, and butorphanol 0.04 mg/kg

Butorphanol 0.02 mg/kg, IM or IV

Correct answer: Xylazine 0.1–0.2 mg/kg, IV

Xylazine at doses of 0.1–0.2 mg/kg, administered intravenously, can provide adequate sedation without recumbency in alpacas/llamas. However, alpacas often need higher doses than llamas for the same duration of effect. This dose should prevent full recumbency, though it doesn't guarantee they will not kush. Higher doses, xylazine 0.3–0.4 mg/kg, IV, are needed to achieve full recumbency.

Butorphanol at doses of 0.05–0.1 mg/kg, IM, can also be used for sedation. However, butorphanol at 0.02 mg/kg is too low a dose without additional medications to benefit you in sedation.

Finally, ketamine 4 mg/kg, xylazine 0.4 mg/kg, and butorphanol 0.04 mg/kg, when combined IM, can be used for more prolonged procedures and will most likely result in full recumbency.

An owner brings a four-year-old FS Siamese in for increased respiratory rate, weakness, and lethargy. She swears the cat's gums are brown. She did vomit a few times yesterday. The owner doesn't know if she got into anything but says that she is a terror and not like any other cat she's had. She willingly takes pills without food and always grabs things off the ground. On physical exam, you appreciate icteric sclera, muddy mucous membranes, paw edema, and she is dyspneic. You provide her with oxygen therapy and discuss with the owner recommended diagnostics. The owner consents to radiographs, urinalysis, and bloodwork. Bloodwork shows an elevated ALT, ALKP, Tbil, and her PCV/TS is 20%/6.0. She has hematuria and hemoglobinuria. Her blood smear confirmed the low red blood cell count with Heinz bodies.

What are you most concerned with in this patient?

Acute acetaminophen toxicosis
Hepatic lipidosis
Cholangitis
Actute diazepam toxicosis

Correct answer: Acute acetaminophen toxicosis

Given this young cat's history of eating things, you would want to question the owner further to determine if anything known, likely acetaminophen or diazepam, has been dropped/ taken by anyone in the household.

While inflammatory liver disease in cats, most commonly cholangitis/cholangiohepatitis, occurs in cats, none of the choices would lead to methemoglobinuria, dyspnea, anemia, and swelling of the paws, except acetaminophen toxicity.

Acetaminophen toxicity in cats can occur with much lower doses than in dogs because cats are deficient in glucuronyl transferase. This limits the animal's ability to glucuronidate the drug and prevents metabolism after saturation via sulfation metabolism occurs. This leads to toxic metabolites. Cats may show signs at doses as low as 10–40 mg/kg, with up to 100 mg/kg known to be toxic. Cats can get renal and hepatotoxicity though dogs are more likely to develop hepatic necrosis. Methemoglobinemia with a heinz body anemia (change in hemoglobin in RBCs due to oxidative injury) confirms the diagnosis. The muddy mucus membranes arise from this methemoglobinemia.

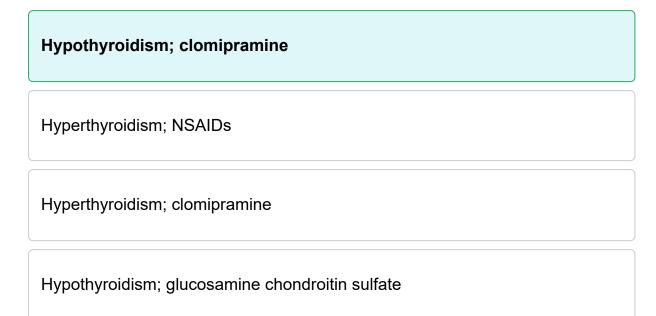
Clinical signs may include weakness, depression, lethargy, trouble breathing, rapid breathing, icterus, vomiting, low body temperatures, dyspnea, paw or facial edema, cyanosis, and even death. Often, the first signs are hematuria and hemoglobinuria.

You can do a rapid bench-top test to differentiate normal blood vs. blood with excess methemoglobin — the abnormal blood will appear a chocolate brown vs. the normal blood. Differentials for methemoglobinemia include other toxicities, such as propylene glycol, nitrates, and local anesthetics.

Treatment is supportive, including oxygen therapy, IV fluid therapy, anti-vomiting, and anti-nausea medications. Transfusions may be needed depending on severity. N-Acetylcysteine (NAC) helps bind the toxin and minimize liver damage. The use of Denamarin as a liver protectant can be beneficial as well.

Hepatic lipidosis or fatty liver is also common in cats but doesn't lead to heinz body anemia or methemoglobinuria.

A four-year-old MN golden retriever with a history of separation anxiety has a history of recent weight gain and obesity, exercise intolerance, no changes in appetite, and even mental dullness. He seems to seek out heat, often found lying on the heating vent. He has a rat tail and bilaterally symmetrical alopecia but is non-pruritic. There is no overt pyoderma. He has no other underlying illness. You want to test him for what ailment? And what medications could he be taking that could interfere with the functional tests?



Correct answer: Hypothyroidism; clomipramine

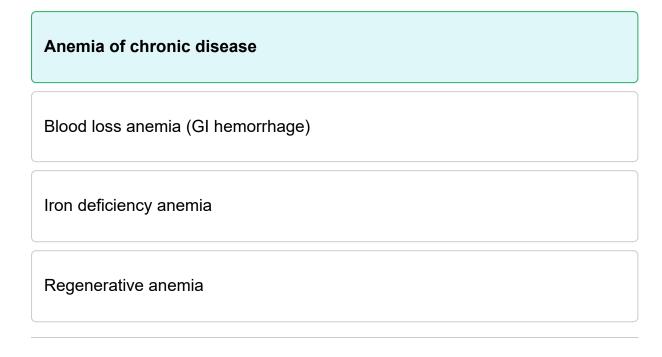
This patient has a classic description that depicts hypothyroidism in dogs. Dogs seem to gain weight suddenly, and owners report no increase in food consumption. They are often heat-seekers, have exercise intolerance, may be depressed or lethargic, and may appear to have a 'tragic look' on their faces. These dogs often have alopecia, commonly truncal and symmetrical but not itchy. Some dogs may develop secondary dermatitis, but not all. This dog has a history of anxiety and takes clomipramine.

Clomipramine, glucocorticoids, aspirin, sulfonamides, phenobarbital, and several other NSAIDs have been shown to interfere with thyroid function testing.

In addition to medications altering thyroid function testing, the presence of an underlying disease can compound testing. All too often, people test for thyroid disease when a patient presents with an underlying illness, such as vomiting or diarrhea, or skin disease. Regardless, animals with underlying conditions can have falsely lowered thyroid levels, called euthyroid sick syndrome. Too many dogs are diagnosed as hypothyroid based on levels taken when sick; thus, the numbers of dogs with this condition are likely lower. Other underlying illnesses will often cause a consistent decrease in the total T3 and T4 levels proportionate to the level of severity of the disease. Dogs sick with non-thyroid illness can have TSH levels increase by 8–10%. Ft4 is less likely affected, but could be elevated or decreased. Thus, avoid testing thyroid function when sick.

Dogs with intestinal neoplasia, up to 64%, can have anemias. You have a 23-kg MN mixed-breed dog with known intestinal cancer treated with palliative therapy. He presents for a recheck of bloodwork and an assessment of his quality of life. His owner describes weakness but not much different than over the past few weeks. He has mildly pale gums and has lost 2 kg, but the owner reports a decent appetite. Otherwise, his physical exam was non-remarkable, save a palpable abdominal mass.

What type of anemia is the most likely cause of his pallor?



Correct answer: Anemia of chronic disease

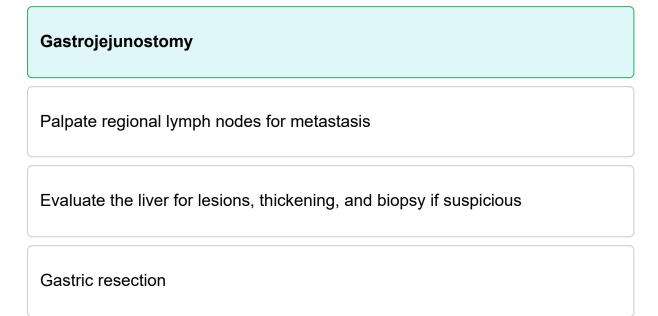
Animals with GI neoplasias may have intestinal bleeding contributing to anemia. However, given the lack of acute change in his overall clinical picture and an overtly normal physical exam (except pallor and weight loss), no tachycardia or bounding/weak pulses, you suspect anemia of chronic disease. This type of anemia occurs with a wide array of disease states. It causes mild to moderate normochromic, normocytic, and non-regenerative anemia. It is the number one reason for anemia in animals.

Iron deficiency anemias are rare in most animal species.

Anemias of chronic disease are the most common type of anemia seen in animals and are non-regenerative by definition. Causes of regenerative anemias include immune-mediated diseases, trauma, and toxins, among other causes.

A ten-year-old MN Belgian Shepherd is referred to your surgical practice for the removal of a gastric tumor. The dog initially presented to the primary care veterinarian with anorexia and weight loss. Radiographs were non-remarkable, and bloodwork showed slight anemia but was otherwise also non-remarkable. The dog was sent home with palliative therapy and was initially doing well, but then started to have hematemesis and melena, and his appetite declined again. His abdominal ultrasound showed mural thickening and loss of normal architecture. Endoscopy showed a submucosal mass without ulceration, but they couldn't biopsy it because of its location. The owners would like a surgical exploratory and removal, if feasible. Chest radiographs to evaluate for metastatic disease were clear.

When performing surgery, the surgeon must be sure to do all of the following, except:



Correct answer: Gastrojejunostomy

All of the answers are technically feasible. However, if an excision and bypass are required, such as a gastrojejunostomy, or cholecystojejunostomy, are warranted, then the prognosis is very, very guarded, and tumor recurrence is very likely with likely clinical signs remaining after surgery and a poor quality of life.

However, if the tumor is localized only to the stomach and gastric resection is possible, it could be curative for adenocarcinomas or leiomyosarcomas (or related benign tumors). Gastric lymphoma, even if solitary, usually requires follow-up with chemotherapy, even with full removal.

Gastric tumors in dogs and cats are rare (<1% of all neoplasias). Cats most commonly get gastric lymphoma, while adenocarcinomas are the most common in dogs. Generally, by the time we identify the disease, metastasis has likely already developed.

Surgical exploratory isn't wrong to determine if the lesion is resectable. However, it is paramount to ensure one palpates and evaluates the entire GI tract and abdominal structures for abnormalities, including the liver, and biopsies any irregularities.

warranted.	 	 	

You are asked to evaluate a three-year-old female alpaca that has been unsuccessfully bred several times. The producer is looking to find out why. You obtain a complete history from the owner. You suspect you know the issue after reading the records and discussing the animal's history with the owner.

Which of the following is a possible cause of the infertility?

She was a twin with a male

Previously corrected angular limb deformity

Infection with Mycoplasma haemolamae

Resolved copper deficiency

Correct answer: She was a twin with a male

New-world camelids are predisposed to congenital and inherited abnormalities. Issues can include choanal atresia (failure of the inner nares to open during development) being the most common. Other abnormalities can include juvenile cataracts, ventricular septal defects, musculoskeletal defects, and angular limb deformities. Tail defects and umbilical hernias also occur.

Urogenital defects seem to be much more common in South American camelids. They can include double cervixes, segmental aplasia of the vagina or uterus, and clitoral hypertrophy related to intersex conditions. Males can have hypospadias, ectopic testicles or retained testicles, and even a corkscrew penis. Either sex can be born with atresia ani.

Freemartinism is a specific problem seen in male/female twins. The female twin, if she shares a placental connection with the male during development, is born sterile due to exposure to masculinizing hormones the male produced. Generally, both twins are chimeras (XX/XY). The male remains fertile, while the female's reproductive tract is underdeveloped and she is rendered infertile.

Mycoplasma haemolamae infections have been reported, and exposure occurs by insect vectors, transplacentally, or via contaminated equipment/needles. Those infected when healthy develop premunity—meaning they are infected but immune, showing organisms in their red blood cells only if stressed or immunosuppressed. This doesn't cause infertility.

 ency results in po ty.	 	

A recently adopted four-MO FI DSH presents to you for a wellness check. The owner said she was told she had been exposed to panleukopenia in utero. She wants her evaluated to ensure she is growing normally and otherwise okay. She describes very mild signs of cerebellar hypoplasia but only primarily evident when she is eating. Which of the following may also be seen with pre-natal or neonatal infections with feline panleukopenia?

Retinal dysplasia and optic nerve hypoplasia/dysplasia
Retinal dysplasia
Optic nerve hypoplasia/dysplasia
Pars plantis

Correct answer: Retinal dysplasia and optic nerve hypoplasia/dysplasia

Feline Panleukopenia Virus (FPV) (AKA Feline Infectious Enteritis, Feline Parvoviral Enteritis) is a parvovirus that causes panleukopenia and enteritis syndromes in wild and domestic feline species globally. Further, it has been found in mink, foxes, and raccoons. It can replicate in ferrets but doesn't cause clinical disease. Usually, infection occurs in cats less than one year of age but can infect any age when unvaccinated. It commonly occurs as outbreaks in shelter-like situations, in barns, feral or stray cat populations, or indoor/outdoor cats without proper vaccination.

If a queen is infected early in pregnancy, she may abort, or the fetus may develop congenital abnormalities. Later in pregnancy, up to one-week post delivery, damage occurs by destroying Purkinje cells and granule precursors in the cerebellum, leading to cerebellar hypoplasia. Signs often begin about two to three weeks of age as kittens start to explore and walk more - with cerebellar ataxia being common. Intention-like tremors are often exacerbated when focusing, like while eating or playing. Most of the time, these patients can do great without complications. However, others may develop hydrocephalus, hydranencephaly, or porencephaly with forebrain changes. Because the virus can also infect non-cerebellar neurons, we can see ocular changes, including:

- Retinal folding
- Retinal dysplasia
- Retinal degeneration
- Optic nerve hypoplasia

Pars plantis represents inflammatory cells in the anterior vitreous in the presence of uveitis. It is a significant finding in uveitis in cats with Feline Immunodeficiency Virus

peracute infection in young kittens is feasible. Some cats may maintain normal white blood cell counts, while others may have leukopenia (neutropenia +/- lymphopenia). A small subset due to secondary bacterial infections may have an elevated white count. Further, anemias, electrolyte changes, and low albumin may also be present.							

A six-month-old MN DSH presented after the owner found him limping up the driveway. He doesn't know what happened but was outside with the cat and heard a yowl. No cars were around, so he knows he wasn't hit by a car and thinks he may have fallen off the patio. He is non-weight bearing lame on the left hind leg. You take radiographs and see a non-displaced, closed, transverse, mid-diaphyseal tibial fracture. No growth plates are damaged, and the fibula appears intact. The owner has financial concerns, and surgery isn't an option. Luckily, this is a good candidate for a closed reduction. You recommend a cast to permit healing and stabilize the fracture. Though you caution that even with strict rest (crated), there is a chance that this method may fail, and surgery may be needed. The owner agrees.

All of the following are true when placing a cast, except:

Apply the cast with the leg held in a slight valgus and flexion

Casts need to span the joint above and below the fracture

Minimize excess padding to prevent loosening of the cast

Ensure that the cast extends distally enough that it can encompass the toes

Correct answer: Apply the cast with the leg held in a slight valgus and flexion When applying a cast, key concepts include:

- 1. Casts must span joints above and below the fracture to ensure stability and healing.
- 2. When applying the cast, hold the affected leg in a slight varus position and flexion.
- 3. Extend the cast distally, including the toes, but leave the toenails of the middle two digits just barely exposed.
- 4. Take care to prevent excess padding. Excess padding loosens the splint after the cast padding has been compressed.

A postmortem evaluation of several recently deceased pigs demonstrates myocarditis, among other findings. You know that several infectious etiologies can lead to myocarditis. All of the following could be a differential, except:

Erysipelothrix rhusiopathiae

FMD (Foot and Mouth Disease virus)

PRRSV (Porcine Reproductive and Respiratory Syndrome Virus)

PRV (Pseudorabies)

Correct answer: Erysipelothrix rhusiopathiae

Erysipelothrix rhusiopathiae is contagious to humans, though uncommon. Erysipelothrix rhusiopathiae, while negatively affecting the heart, has been shown to cause pericarditis, not myocarditis. This organism is capable of infecting pigs of any age and causing significant economic losses. Relevant species in pigs include E. rhusiopathiae primarily, though E. tonsillarum has been isolated from vegetative endocarditis lesions and in chronic arthritis cases. These bacteria are non-sporulating, not acid-fast, nonmotile, facultative intracellular, facultatively anaerobic, and gram-positive rods.

Though rare in people, it is considered zoonotic. The domestic pig is the largest reservoir, with 30–50% of pigs acting as healthy carriers. The organism is housed in the tonsils and other lymphatic tissues. Shedding occurs by clinical or carrier pigs in the urine, feces, saliva, and nasal mucous for long periods.

Transmission arises via the fecal-oral route directly or via a contaminated environment. Disease in pigs is most likely between three months and three years of age. In pigs, it causes an acute, subacute, and chronic form.

The acute form causes septicemia leading to abortions, lethargy, fever, painful joints, depression, laying down, not wanting to get up, stilted gait, reluctance to move, decreased appetite or anorexia, acute sudden death, and a characteristic firm either pink, purple, or red diamond shaped skin lesion. If non-fatal, lesions vanish in four to seven days.

FMD is an all too familiar, very contagious viral disease of wild and domestic animals (cloven-footed) and others. Lesions are confined to the feet, snout, and inside the mouth with vesicles, and all ages are at risk. It has been eradicated from the U.S. on nine occasions, remaining free since 1929. Since the lesions are indistinguishable from other vesicular diseases, they must always be on a differential list when seen.

FMD is an Apththovirus, with distinct immunological types A, O, C, SAT, 1, 2, 3, and Asian 1. One antigen (Virus Infection-Associated Antigen, or VIA) is common among all and helpful in diagnosis. However, new subtypes continue to develop. Transmission is via aerosolization and direct or indirect contact. The spread has been shown to be far-reaching, at as great as 30 miles with the appropriate weather. Many pigs are then amplifier hosts and shed a tremendous amount, contributing to the widespread dissemination. The virus adheres to the respiratory tract's mucosa, where macrophages transport it to the mucosa, epithelium, and myocardium. There it replicates, leading to viremia. Shortly, vesicles develop (in areas with mechanical stress) on the feet, snout, mouth, and tongue in swine. Vesicles may slough over time. Juveniles and neonatal pigs often develop severe myocardial necrosis leading to sudden death, resulting in classic lesions referred to as "tiger-heart" lesions, which can help with diagnosis (mottled myocardium).

Signs include initially lameness, then fever with chomping and slobbering. Abortion/stillbirths may occur or sudden death. Initially, lesions appear blanched, with small foci on various areas. Once readily apparent, they are vesicles or bullae. Mortality is low except in very young pigs.

PPRSV (Porcine Reproductive and Respiratory Syndrome Virus) is an enveloped RNA virus in the Arteriviridae family. The disease has two clinical phases, the first causing reproductive failure and the second, respiratory disease post-weaning. If a naive herd is infected, not all will develop clinical disease, leading to a persistently infected subpopulation and carrier animal state. Since eradicating Classical Swine Fever, PRRSV is considered the most costly disease in the global swine industry.

Disease transmission occurs via direct contact and through contaminated semen. Aerosolization and fomite transmission also contribute to disease spread. It is not zoonotic. Experimentally, insect vector transmission can also occur.

Reproductive disease phase: Stillbirths and mummies incidence may be up to 25—35%, and over 10% may abort. Anorexia and lack of milk production can lead to higher preweaning mortality rates of piglets. This form can last one to four months, depending on the health of the pigs at infection and various facility characteristics.

The respiratory phase tends to occur in suckling piglets, and they may develop thumping respirations and conjunctivitis, with interstitial pneumonia on histopath being common. Piglets may be born viremic and serve as another source of transmission. This post-weaning phase may become chronic, causing decreased total weight gain and 10–25% mortality. Other infections may occur concurrently, including Streptococcus suis, Salmonella, Mycoplasma, and swine influenza, among others.

Gilts, sows, or boars may show reproductive abnormalities, fever, lethargy, waxing and waning, anorexia, and sometimes respiratory distress or vomiting. Mild cyanosis of the ears, vulva, and belly can occur, as well as dyspnea in nursing animals (thumping). Signs may cycle depending on when animals are exposed and shed. Multiple strains may infect a single herd and are not fully cross-protective.

Young, growing, and finishing pigs may show depression, stunted growth, fever, lethargy, and pneumonia. Sneezing and expiratory dyspnea also may occur along with stunted growth.

Control is key, but there isn't a single control strategy that works. Ideally, establishing 'herd immunity' minimizes transmission and subsequent losses. But quarantine of boars for 60-90 days before introducing them to the females in a negative herd is critical. Vaccination may be utilized as a part of a control strategy. Still, it cannot be the only measure as they are not 100% effective. Depopulation and appropriate cleaning measures then, obtaining all new animals PRRSV free is an option. If one doesn't want to depopulate, they can close the herd for a minimum of 200 days,

stabilizing the herd. But the risk of re-infection is high, and biosecurity protocols must be airtight.

Diagnosis most commonly relies on ELISA measuring the IgG antibodies. However, it cannot assess the level of immunity or predict carrier state animals. Titers are evident seven to ten days post-infection and may persist up to 144 days. Test options also include virus isolation, immunohistochemistry, and PCR. Sampling oral fluid from entire pig populations has been used widely as a screening method.

Modified live vaccines provide some protection and may limit shedding, but no treatment exists. Prevention is paramount.

PRV, AKA Aujesky's disease, is a significant disease player in the swine industry. It was eradicated in the U.S. in 2004. It consists of three overlapping clinical syndromes affecting either the nervous, the reproductive, or the respiratory systems. PRV is an alphavirus subfamily of Herpesviridae. It can persist in a carrier (latent state) and easily be destroyed by many disinfectants.

Transmission occurs via contact and exposure to saliva, nasal excretions, urine, and feces. The virus can survive in carrier pigs' tonsils for several weeks and in the CNS for months. Thus, stress can precipitate recrudescence and subsequent re-shedding. It spreads through direct contact, contaminated food/water, and aerosolization.

Clinical signs vary depending on the age of the pigs affected and the immune status of the dam. Younger appear more affected, while older pigs are more resistant.

Sudden death in the very young may be the only clinical sign. Nursing piglets often show neurological signs. You may see very high fevers, conjunctivitis, anorexia, tremoring, depression, vomiting, foaming at the mouth, dog-sitting, paddling, blindness, seizures, coma, and death within one to three days.

Pigs three to nine weeks of age, already weaned, generally have respiratory signs predominate, such as nasal discharge, sneezing +/- cough, and labored breathing. The mortality in nursing pigs can reach 100% but is lower in this age group. Most survive unless they develop secondary bacterial pneumonia.

Ten weeks to market weight morbidity is high and respiratory signs also predominance. They may be febrile, sneeze, cough, have nasal discharge, have depression, or be anorexic. Some will also develop CNS signs. However, most recover in seven to ten days.

Breeding animals may have respiratory disease with full resolution, though, reproductive failure is possible, including mummified fetuses, stillbirths, or weak pigs, in the second or third trimesters.

Swine are the natural domestic animal reservoirs. Vaccinations are highly effective, but all ages are susceptible if not vaccinated. In the commercial U.S. swine industry, the disease has been eliminated. However, a reservoir still exists in some feral populations and is prevalent in other countries. If other species, such as sheep, cattle, dogs, cats, or goats (not horses), are kept in close proximity to pigs, they are susceptible, and death usually occurs in aberrant hosts. Several wild rodents, rabbits, and fur-bearing mammals can become infected. It is not zoonotic.

There is no treatment but weaned and older pigs do tend to recover.

When performing an oral cavity evaluation on your canine patients, it is important to grade the level of disease. We want to assess not just the amount of tartar and gingivitis but many other factors. This permits us to better describe to owners what will be necessary with a Comprehensive Oral Health Assessment and Treatment (COHAT). A four-year-old MN toy poodle presents to you with halitosis. The owner has not brushed the dog's teeth as recommended but gives a greenie/day. You had recommended daily brushing at last year's examination because the pet was a grade 1. Grade 0 and grade 1 scores can be managed at home with appropriate and great home dental care. This includes teeth brushing and products with the Veterinary Oral Health Council seal. Patients with grade 2 should have a COHAT performed to prevent worsening, while those with grades 3 and 4 need a COHAT ASAP.

Which of the following is consistent with the definition/description of grade 3 dental disease?

Moderate to severe gingivitis with moderate calculus formation

Mild to moderate gingivitis with moderate plaque and calculus formation

Severe gingivitis with severe +/- bridging calculus +/- obviously loose teeth

No gingivitis or tartar evident

Correct answer: Moderate to severe gingivitis with moderate calculus formation

- No gingivitis or tartar evident characterizes a dental grade of 0.
- Minimal to mild gingivitis with the mild formation of plaque characterizes a dental grade of 1.
- Mild to moderate gingivitis with a moderate plaque and calculus formation characterizes a dental grade of 2.
- Moderate to severe gingivitis with moderate calculus formation characterizes a dental grade of 3.
- Severe gingivitis with severe +/- bridging calculus +/- obviously loose teeth characterizes a dental grade of 4.

The COHAT terminology should be used in place of dental cleanings, or dental, so that owners fully comprehend the depth and breadth of what is accomplished during a "dental," the benefits of dental radiography (which should be standard of care; not optional), and are informed of ways to prevent worsening disease. Owners who seek out COHATs when their pet has a grade 1 or 2 ensure that the pet doesn't develop severe disease, is at low risk for systemic spread of disease, and can save teeth and

lessen the risk of pain and suffering. Further, the procedure at this stage is also much less costly for the owner.					

It is winter, and many horses in a herd are very pruritic and have dry patchy areas with alopecia. The neck and tail regions are most notably abnormal. Several horses have just come in from exercise when you evaluate them, and you easily identify lice with a hand lens or otoscope (sans cone) as they have crawled out toward the surface. Which of the following is a biting louse associated with horses?

Werneckiella equi equi
Haematopinus asini
Eutrombicula alfreddugesi
Psoroptes equi

Correct answer: Werneckiella equi equi

Pediculosis in horses is caused by two types of biting lice (Bovicola equi and Werneckiella equi equi) and a sucking louse, Haematopinus asini. Infestations are most common in the winter due to the breeding cycle of the species. Lice are obligate ectoparasites, thus, species-specific. Pruritus is a hallmark of infestation, with the tail and neck most commonly affected. They can develop patchy and dry coats and alopecia with crusts and ulcerations. They are visible with a simple hand lens. They will come to the surface and be even more readily visible after exercise.

Trombiculidiasis, chiggers, or harvest mites, cause infection/infestation with crusts and papules commonly on the limbs, neck, and face. They are variably pruritic. If found with a lens, you can see the larval mites of Eutrombicula alfreddugesi (North American species of concern) in the late spring to early fall. They are red-orange larvae and six-legged. You can also find them via skin scraping and impression smears.

Finally, Psoroptes species are the causative agent of "mange" in large animals, including horses, though host specificity remains controversial. Psoroptic mange has been eliminated in the U.S. in horses and sheep, though it remains an issue in cattle. The hallmark of mange is also pruritus, which can be severe. Horses most commonly develop lesions at the mane's base, the tail, ears, and intermandibular regions, then the trunk. These include alopecia, scaling, and crusting. These are not visible with the naked eye or a lens and must be identified on skin scraping/impression smears.

A nine-year-old FS Boxer presents with a recent history of vomiting, decreased appetite, weight loss, lethargy, and intermittent diarrhea. The owner thinks it is pancreatitis and initially wants just to treat it symptomatically but consents to diagnostics. There is scant free fluid. You aspirate expecting it to be a hemoabdomen, but it isn't consistent with blood. His radiographs show a poorly defined increased soft tissue opacity in the right abdominal quadrant, cranially. There isn't enough free fluid on radiographs to show a loss of visceral detail. The bloodwork shows an elevated ALKP and mildly elevated total bilirubin, though you didn't appreciate gross icterus. He is mildly hemoconcentrated, having vomited several times today and not eaten anything since yesterday. A board-certified radiologist performs an abdominal ultrasound that shows a pancreatic mass. Cytology of the abdominal fluid shows a modified transudate without neoplastic cells. Coagulation tests (PT and aPTT) were normal before obtaining a Fine Needle Aspirate (FNA) of the mass. The FNA sample diagnosed exocrine pancreatic carcinoma. The owners consult with the oncology and surgery services about their options.

All of the following are viable treatment options, except:

Chemotherapy
Intraoperative euthanasia
Palliative care
Pancreaticoduodenectomy (Whipple procedure)

Correct answer: Chemotherapy

Most exocrine pancreatic tumors are epithelial in origin, arising from the ductal or acinar epithelium (vs. insulinomas which arise from the endocrine pancreas' islet cells). This condition is rare (< 0.05% of cancers in dogs/cats). An association between pancreatitis and the development of this condition has been researched. Clinical signs can include weight loss, appetite changes, weakness, maldigestion, abdominal distension, effusion, vomiting, and icterus. Radiographs, ultrasound, FNA of the mass, and CT or MRI may help identify metastatic disease before considering surgery. Most patients have advanced disease by the time of clinical diagnosis; thus, 50–70% already have metastatic disease in either local lymph nodes or distant sites. Treatment is surgical with a complete pancreatectomy or pancreaticoduodenectomy (Whipple procedure). But cure rates are low, and complications arise. Chemotherapy and radiation aren't of much use (especially without surgery).

Palliative therapy, such as treating current vomiting, rehydration, addressing pain, and periodic abdominocenteses with intracavitary chemotherapy, may be attempted,

Due to the poor prognosis and high metastasis rates at the time of diagnosis, while surgery can be attempted, intraoperative euthanasia must be brought up to the owners as a possibility before surgery. If the surgeon opens the patient up and					
carcinomatosis or other signs of metastatic disease exist, the owners need to be updated so that they can make an informed decision.					

A four-MO MI DSH presents to the emergency room with hypersalivation, hyperexcitability, tremors, and weakness. The owner had recently applied topical pyrethrin to the pet. However, she accidentally put the dog's product on the pet, not the cat's version. She didn't even realize it until the cat started showing clinical signs, and they rushed her to the ER.

Treatment of this type of toxicity may include all of the following, except:

Diazepam 0.5 mg/kg IV

Bathing in cool water with Dawn® original liquid dish soap

Methocarbamol 55–200mg/kg IV

Intralipid infusion (bolus and CRI or just CIR)

Correct answer: Diazepam 0.5 mg/kg IV

Pyrethrins are common insecticides used in sprays and spot-on therapies in dogs to treat/prevent ear mites, fleas and ticks, and other related organisms. Canine spot-on products often contain 40–50% active ingredients, and it is thought that anything greater than 5–10% can lead to systemic illness in cats due to their glucuronidation metabolism differences.

Exposure can occur when owners apply dog products to cats or when cats are permitted in close contact (grooming, rubbing up against) dogs with applied products. They should be separated long enough for the product to completely dry if used in the same household.

Signs can include drooling, nausea, vomiting, disorientation, tremors, seizures, hyperexcitability, hyperthermia, weakness, tachypnea, or dyspnea.

Tremors most frequently respond to methocarbamol, ideally IV, as oral onset is slower. These tremors are much less responsive to benzodiazepines, and seizure activity is also less well controlled by benzodiazepines. Phenobarbital (4–16 mg/kg IV PRN to effect) or gas anesthesia is much more effective at controlling seizure activity induced by pyrethrin exposure.

Dermal decontamination is absolutely necessary but must be performed after stabilization. once stable enough to bath, bathing in cool water with Dawn®'s original liquid dish soap can help to remove the remaining product and lessen the chance of continued exposure and toxicity. Monitoring needs to include body temperature, hydration status, and sugar levels.

Clinical signs can last for 24–96 hours. The prognosis is usually good with aggressive therapy and decontamination.

Sometimes, however, cost plays a role in care, and some owners cannot afford hospitalization and supportive care. In those animals or in animals whose signs are severe, fail to respond to medications, or are declining, the use of intralipids can be utilized.

An intralipid bolus of 1.5 ml/kg can be administered in severe cases. In milder cases, just the CRI may be utilized 0.25-0.5ml/kg/min. Intralipid use may limit the need for anti-tremoring/anti-seizure medications and shorten hospital courses. While many have used intralipids, they are off-label, and we still lack high-grade evidence supporting their use. Randomized controlled trials ideally are needed to determine more about ideal dosing. Thus, it isn't usually the first thing we reach for though it has been shown to be beneficial.

For owners of captive reptiles, it can be hard to feed them, recognize illness in the early stages, and meet their very specific husbandry requirements. You have been treating a green iguana for several months with a history of radiographic changes that showed decreased cortical thickness and long bone opacity, soft tissue swelling, and periosteal proliferation. Adjustments in diet, husbandry, and enclosures were made.

You are taking further images today to see if the changes have resolved or are improving and if the lizard is responding to therapy. The owner said that previous muscle tremors, anorexia, and unwillingness to walk/stand for long periods had improved dramatically. While the iguana is still not as active as expected, the owner feels there has been a dramatic improvement.

Which of the following was not part of your treatment recommendations to the owner to improve the patient's outcome?

Vitamin D₃ supplementation

Oral or parenteral calcium supplementation

Padded enclosure with substrates that minimize the risk of fractures

Appropriate UVB light access

Correct answer: Vitamin D₃ supplementation

This patient was being treated for metabolic bone disease, specifically for Nutritional Secondary Hyperparathyroidism (NSHP). This metabolic disorder is, sadly, the most common of the Metabolic Bone Disorders (MBDs) in reptiles and is sometimes called "rubber jaw" because of the softness that develops in the jawbones and the facial deformities that may arise. It results from a combination of factors, including the following:

- 1. Improper dietary ratios of calcium-phosphorus. This could result from too little vitamin D_3 sources and/or too little calcium supplementation.
- 2. Insufficient UVB light exposure in diurnal species (insectivorous and herbivorous lizards and chelonians). Those that are carnivorous or eat living prey and those that have a nocturnal lifestyle rely exclusively on the diet for their vitamin D₃ sources. Vitamin D₃ is required to ensure proper calcium and phosphorus regulation in the body. Diurnal species, as described above, require sunshine to synthesize sufficient amounts of D₃ to aid in metabolism that facilitates calcium absorption from the GI tract. These animals require a UVB source when kept indoors, but if feasible, exposure to true sunlight is always

- preferred. Vitamin D supplementation is not recommended because there is a very narrow therapeutic window, and vitamin D toxicity occurs easily.
- 3. Physical hazards. Provide a safe enclosure and habitats with a lot of padding and minimize climbing obstacles and other activities that could increase the risk of pathological fractures until the bones are able to heal and the osteopenia improves.
- 4. Vitamin A deficiency. Oral vitamin A supplementation is also advised, as this is key to renal health. Since negative consequences can affect the kidneys (renal secondary hyperparathyroidism) and the bones, vitamin A supplementation is crucial to recovery.

Recheck radiographs and bloodwork (to evaluate calcium levels and renal values) to monitor improvement. Prognosis with MBDs in reptiles varies with the degree of systemic illness, the severity of bony and/or renal lesions, the presence and locations of fractures, the return to normal ambulation/function, and the owner's willingness to take part in the recovery process. Euthanasia may need to be considered for animals with fractures causing paralysis/paresis that affects their ability to defecate/urinate/lay eggs.

A 14-year-old FS Bichon mix with a history of stage 2 CKD presents to a 24/7 ER. She had a decreasing appetite over the past month. She was switched to Hill's KD diet about two weeks prior. Before that, she had developed diarrhea/soft stool that the owners self-diagnosed as pancreatitis (as she had previously), and the stool improved. However, it is still soft but formed. She had no vomiting and no specific change in urination/drinking. Still, she vomited seven times before her presentation, was panting excessively, and guarding her abdomen. On physical exam, her heart rate was 60, with moderate snappy pulses. She had a very focal left apical II/VI holoystolic heart murmur. Her gums were pale, and her rectal temperature was 98.2°F. She had moderate abdominal pain and nausea with palpation of the abdomen.

Bloodwork showed:

- Crea 3.4 (0.3-1.6)
- BUN 64 (9-30)
- CA 13.3 (ionized normal at 1.33)
- P 8.7 (2.7–5.4)
- Electrolytes were normal
- Alkp 192 (7–115)
- ALT 122 (17–118)
- Urinalysis was obtained after fluids because of an empty bladder. USG 1.011, TNTC cocci, degenerate neutrophils, +++ Blood on dipstick
- Initial BP 160 and, despite fluid therapy, spiked to 220
- Initial lactate 6.9 (X-3); After several hours on fluids with several fluid boluses, decreased to 4.0.
- PCV/TS: 58%/8

You admit the patient and obtain radiographs. Because of the murmur, you want to assess cardiac size and vasculature and evaluate for any occult neoplasia, given her age. Further, you want to assess the abdomen.

This patient received buprenorphine, cerenia, pantoprazole, ondansetron, enrofloxacin for suspected pyelonephritis, four 10 ml/kg boluses over three hours, and was on 5 ml/kg/hr of Plyte. Initially, her temperature normalized, her color returned to normal, and she continued to pant intermittently but would sleep. Later in the evening, however, her color again became pale and almost muddy. Her pulses were weak, and her temperature dropped to 96.3°F. Still, her axillary temperature was 98.1°F, and heat support was removed because her panting worsened dramatically. Her temperature was likely normal, but lack of perfusion and vasoconstriction likely contributed to an inaccurate rectal temperature. She had formed stool that appeared to have melena. She had urinated one time during her stay and had urine removed via cystocentesis. Referral to a specialty practice was declined. PCV/TS only improved to 52%/7.8.

This patient is hemodynamically unstable. An aFAST was performed that showed gastric wall edema and gastric thickening but no free fluid. Cardiac contractility was decreased. Her heart dropped to the 60 seconds again despite aggressive therapy. However, she remained alert and actually seemed to rally and brighten. When evaluating patients during shock resuscitation, we need to determine the success of treatment.

Endpoints of resuscitation include all of the following, except:

Normalization of PCV/TS

Normalization of heart rate

Normalization of respiratory rate

Normalization of blood pressure

Correct answer: Normalization of PCV/TS

While monitoring the PCV/TS can help assess rehydration status or for fluid overload, it doesn't fully demonstrate key endpoints we need to ensure that shock resolves/responds to therapy.

Endpoints of resuscitation must also be evaluated in the context of the patient and clinical signs. Not all patients follow the rule books, as this patient didn't.

Improvements in only a few parameters don't indicate complete resolution of shock.

Endpoints include the normalization of the:

- 1. Heart rate
- 2. Respiratory rate
- 3. Blood pressure
- 4. Improvements in pulse quality
- 5. Improvements in blood lactate concentration
- 6. Improvement of capillary refill times

Further, while some parameters may normalize, the patient may still have poor oxygen delivery at a subcellular and cellular level. Thus, additional parameters include base excess, lactate, and hemoglobin saturation. Once physical exam parameters improve/ normalize, biochemical endpoints can be evaluated.

Despite aggressive therapy, this patient's low dose micro CRI of dobutamine to improve cardiac output (per a criticalist's recommendations) and intensive management failed to respond as expected. Failing to address the underlying cause rapidly enough or missing the whole picture (unable to obtain an ultrasound until morning) means something may have been missing. It may mean that full resuscitation isn't feasible. Was this a bad pyelonephritis and pancreatitis with gastric wall edema? Was the renal pelvis mineralization clinically significant in this patient? Was there cancer hidden? These owners elected humane euthanasia because of her lack of improvement/stabilization. The renal mineralization, the only noted radiographic abnormality by the radiologist, and normal cardiac size/vasculature, do not provide sufficient information.

You have a bitch who is about to whelp but is having a dystocia. She requires a c-section. The owner does not plan to breed her again and would like her spayed. It would be appropriate to do an en-bloc c-section in which of the following scenarios?

The mother is stable, with normal bloodwork
Fetal hypoxia
Fetal bradycardia
Fetal distress

Correct answer: The mother is stable, with normal bloodwork

Animals may require a C-section for various reasons:

- 1. Certain breeds often require them, including Boston terriers, French and English bulldogs (French bulldogs more frequently require this), some mastiff breeds, and Scottish terriers
 - 2. Animals with previous c-sections may require them for future deliveries
 - 3. Brachycephalic and small-breed dogs may need them more often than largebreed dogs
 - 4. Dystocias (due to malpositioned, maldeveloped, or oversized fetuses)
 - 5. Small pelvic canals
 - 6. Uterine inertia
 - 7. Hypocalcemia (prepartum preeclampsia) is common in animals without prenatal care and not fed an appropriate puppy food or supplemented correctly during pregnancy
 - 8. Fetal putrification

If the owners do not want to save the uterus and plan to spay the patient during c-section, en bloc OHE (Ovariohysterectomy) can be considered. This means that the uterus is removed before a hysterotomy (incision in the uterus) is made to remove the neonates. This can be done if the mother is not in distress, her electrolytes and other parameters are stable, and the fetuses aren't in distress. If, however, they are in distress, a uterine incision and delivery of the puppies should be undertaken before spaying the patient. Even if the owner has not elected OHE, removal en bloc of the uterus may be needed if the uterus is of questionable health.

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All of the following antibiotics have been associated with antibiotic dysbiosis and enterotoxemia in guinea pigs, except:

Nitroimidazoles
Beta-lactams
Macrolides
Lincosamides

Correct answer: Nitroimidazoles

Antibiotics that can be safely used in guinea pigs include metronidazole (a nitroimidazole antibiotic), fluoroquinolones (such as enrofloxacin), chloramphenicol, trimethoprim-sulfamethoxazole, and a few others. However, beta-lactams, like the penicillins and related derivatives, macrolide antibiotics like azithromycin, and lincosamides like clindamycin can be lethal and must be avoided. Beyond appropriate antimicrobial stewardship, it is essential to know and understand the species you work with to ensure safe antibiotic choices and to only reach for them when absolutely necessary.

Guinea pigs are susceptible to changes in gut bacteria, with an increase in the gram negatives and a decrease in the gram positives. In some species, this leads to dysbiosis and may trigger diarrhea. However, in guinea pigs, it can lead to lethal enterotoxemia, because of an overgrowth of Clostridial organisms. We can see C. difficile develop, and the toxin production causes a systemic wide response. If it develops, high-fiber diets may be used to prevent ileus. Cholestyramine may help bind the toxins, but these antibiotics must not be used to prevent this life-threatening illness

Clostridium pilliforme does cause disease in guinea pigs, but not enterotoxemia. Instead, it causes Tyzzer's disease, though this occurs more commonly in small rodents. It can lead to diarrhea, poor coat quality, and depression. Transmission occurs not by antibiotic dysbiosis and overgrowth but by the fecal-oral route. Some animals may remain asymptomatic carriers and, when immunosuppressed, demonstrate clinical signs. Because it is an intracellular organism, premortem diagnosis is difficult. Prevention is key, as treatment is usually not effective.

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You are evaluating an 18-year-old cat who presented with a 12-hour history of hematuria, pollakiuria, and stranguria. The owners feel she was otherwise healthy and had been eating and drinking normally until today. They report she had an ivy ingestion three to four months ago and was seen at another emergency hospital. Her bloodwork at that time was said to be normal. The patient is somewhat fractious, so interpreting physical exam abdominal palpation for pain is challenging. You perform radiographs that show small kidneys bilaterally but are otherwise unremarkable, and there is no evidence of cardiac disease. Bloodwork and urine results are as follows: Bun 66 (15-32); Crea 2.6 (0.8-1.6); WBCS 40.0 (5.5-19.50); Neuts 35.99 (3.12-12.58), Eos 1.99 (0.06-1.93), but blood smear shows varied neutrophil morphology and immature neutrophil cells and a normal manual eosinophil count. You suspect the machine was misreading some of the neutrophils. HCT 33%; PCV/TS: 33/7.0; Urinalysis: USG 1.018; WBCs 15-30/hpf; RBCs 10-20/HPF; Rods and cocci in both chains and singularly multiple granular casts, and some squamous epithelial cells. A urine culture is pending. What are you most concerned about in this patient?

Chronic pyelonephritis
Renal lymphoma
Acute pyelonephritis
Transitional cell carcinoma

Correct answer: Chronic pyelonephritis

Given the patient's age, lack of systemic clinical signs, the findings on bloodwork (azotemia and leukocytosis with a left shift) and the findings on a sterile urine sample, chronic pyelonephritis is the most likely etiology. Taken together with the small kidneys bilaterally, this suggests a degree of chronic renal change and is more supportive of chronic vs. acute pyelonephritis. While infections in cats occur very infrequently, they are common in cats that are very young, immunosuppressed, or have underlying kidney disease. Further, renal lymphoma in cats is often associated with FeLV infection and manifests with bilateral renomegaly, not small kidneys. We cannot fully rule out Transitional Cell Carcinoma (TCC) in this patient without imaging. However, most patients with TCC have clinical signs for weeks to months, not a sudden acute onset of hematuria and pollakiuria. No transitional cells were noted, but an abdominal ultrasound would be needed to evaluate the kidneys and rest of the urinary tract system to determine if there is an underlying trigger for the obvious kidney infection.

This patient should be hospitalized on IV fluids and IV antibiotics to help flush out the kidneys and start antibiotics pending urine culture. Starting a broad-spectrum

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You have a client who calls after finding a litter of kittens without a mother. She plans to bottle-feed and hand-raise the kittens. She saw the mother give birth yesterday but hasn't seen the mom since. The kittens have been crying for hours. How long does she need to stimulate the babies to urinate and defecate?

First three weeks of life	
First two weeks of life	
First one week of life	
First month of life	

Correct answer: First three weeks of life

Normally, mom's grooming helps to stimulate infants to go to the bathroom. However, without a mom's gentle tongue, humans need to stimulate neonates to go to the bathroom, generally for the first three weeks of life. You can use a cotton swab, finger, or tissue and softly rub the genital area. Rubbing the anal area accomplishes the same thing for stool. Normal infants have loose but not liquid stools. If they aren't defecating and you are concerned about constipation, you can always take the tip of a thermometer and place it in the rectum. This will often stimulate them to go.

A previous sheep farmer has elected to switch production to dairy cattle farming. He had no issues with the sheep in the environment. Still, since the cattle have been housed on the same lot, he has had numerous young growing cattle, more than adults, with vague clinical signs such as tenesmus, diarrhea, and weight loss, and then several have progressed to ascites. One had a prolapsed rectum. Several animals have also developed neurological changes. The producer requests that you do some diagnostics on some of the animals showing clinical signs to see if you can identify a source of the problem. He is concerned it may be improperly mixed in the feed rations or infectious. Bloodwork shows elevated liver enzymes. Some animals have GDH, SDH, and LDH elevations, but others have only GGT and ALT changes. Gross pathology in some who have succumbed or been euthanized due to signs of liver failure includes fibrosis, bile duct proliferation, and megalocytosis. On postmortem, all cattle who died had ascites, extremely thin body conditions scorewise, and more than one had a prolapsed rectum. The livers were small; some were firm with pale brown to yellow coloring. You discuss with the farmer possible causes and suspect an environmental source. You request to evaluate the habitat, and the farmer consents. You want to see if you can identify the source and develop a management strategy that will help prevent animal exposure. What are you looking for as the potential cause?

Common groundsel (Senecio vulgaris)
Cocklebur (Xanthium orientale)
Cottonseed (Gossypium spp.)
Whitebrush <i>(Aloysia gratissima)</i>

Correct answer: Common groundsel (Senecio vulgaris)

Plants causing pyrrolizidine alkaloid toxicosis (AKA Seneciosis, Senecio Poisoning, Ragwort Toxicity) (PA), such as is seen with various Senecio species, leads to liver failure. Numerous species of plants and multiple genera contain the PA compounds of concern. Usually, the plants of concern aren't intentionally eaten, not very palatable. Thus, we see chronic, delayed, and progressive toxicity and changes leading eventually to liver failure. The fact that sheep previously grazed the same area without issue can be attributed to their being much less susceptible to the toxic effects.

Sadly, various plants cause liver toxicity. Two common species of concern include Senecia species, which contaminate first-cut alfalfa, and Amsinckia intermedia, common with oat hay fields. The key hallmark change seen with PA toxicity is hepatic megalocytosis (in combination with a small, fibrotic liver). Though aflatoxicosis can

also cause this, thus, it is not considered pathognomonic. Still, while other plants cause liver toxicity, it doesn't present the same way. These patients will also have non-specific changes, including biliary hyperplasia early on in the disease with fibrosis and hepatocyte necrosis.

Various species can develop PA toxicity, including horses and dogs.

With Whitebrush (Aloysia gratissima), we generally see fatty degeneration.

With Cocklebur (Xanthium orientale), we see hemorrhagic, centrilobular necrosis.

We see liver necrosis and cardiac lesions in Cottonseed (Gossypium spp.) secondary to gossypol pigment. With this toxin, liver damage occurs secondary to cardiac failure and is not a primary liver problem.

Identifying the plant of concern and developing a management strategy to prevent growth and or prevent consumption will be paramount to prevent future outbreaks/issues.

A ten-year-old MN DSH presents because of a lesion on his ventrum that the owner noticed a few weeks ago. It started as pea-sized, but today it started to ulcerate, and it seems to have grown a bit. The owner stated that it began as a single reddish-black nodule easily compressible and circumscribed.

Which of the following is the treatment of choice?

Excisional removal
Cryosurgery
Radiation therapy
Chemotherapy

Correct answer: Excisional removal

In cats, all tumors should be considered malignant and evaluated ASAP. This owner, however, describes to you a classic hemangioma. They are rarely seen in cats and can be found on the extremities, head, or abdomen. They start as single or multiple small red-to-black nodules and are often circumscribed and compressible. They can be ulcerated and appear first as a blood blister. They are benign, but because they ulcerate and can grow large, excision is the treatment of choice. Sometimes, those tumors on a distal extremity may be difficult to remove, and radiation or cryosurgery may be warranted. Because these lesions are benign, chemotherapy isn't necessary.

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All of the following would be considered appropriate primary or adjunctive treatment options in managing acute lead toxicosis in cattle, except:

d-Penicillamine

Thiamine

Calcium disodium EDTA (calcium versenate)

Rumenotomy to remove sources of lead

Correct answer: d-Penicillamine

Cattle with acute lead toxicity may show GI signs such as drooling and bruxism and neurological signs such as blindness, eyelid twitching, muscle tremors, ataxia, or seizures. This usually occurs in younger animals, and signs occur about one to two days post-ingestion. Those with subacute poisoning, usually older cattle, will have signs of dullness, rumen stasis, anorexia, head pressing, blindness, and constipation, followed by diarrhea, bruxism, incoordination, and hyperesthesia. Chronic toxicity can manifest in various ways, including a combo of acute/subacute disease. Chronic disease swallowing reflexes may be a problem, and aspiration pneumonia may ensue. Infertility may also be a sign of chronic disease in males or females.

The presence of GI, along with neurological signs, occurs in about 60% of cattle ingestions and helps differentiate lead toxicity from other common cattle neurological diseases.

Treatment in food-producing animals with lead toxicity is not recommended. A combination of a poor prognosis (poor response to therapy), level of clinical illness, herd management mentality (vs. individual animal), and tissue residue severity with both public health and economic ramifications for the animal/producer make it difficult to recommend treatment in cattle used in food production. For small animals, where tissue residues and human health risks aren't a concern, depending on the severity and acute vs. chronic exposure, treatment may be warranted and prove fruitful.

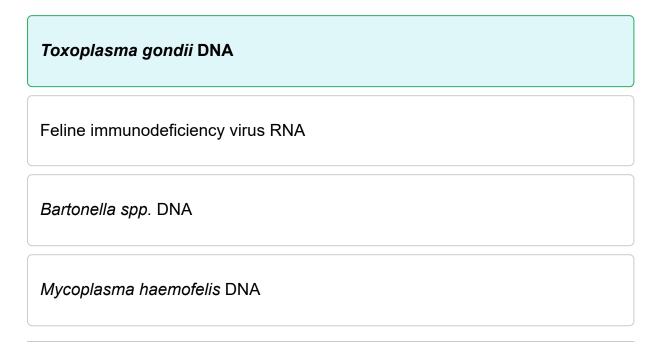
No approved treatment on-label exists for chelation in cattle. Of the available chelators, d-penicillamine, dimercaprol, and calcium sodium EDTA, the latter is the most effective in cattle. Treatment is given for a few days, then an off period is needed, and post-treatment, blood lead levels, and kidney function should be evaluated. Care must be made to ensure mineral deficiencies do not arise, as it will also chelate other things like zinc. Oral supplementation of zinc may be needed.

Supplementation with thiamine has been shown in small and large animals to aid in acute toxicities, lessening clinical signs.

performed to remove the exposure. Magnesium sulfate laxatives may be used to form insoluble compounds of lead sulfides.					
Some patients may require supportive care and nutritional management depending on the degree of neurologic and other abnormalities.					

You are looking into starting a blood transfusion donation program for cat blood. You run a local ER and are having no trouble keeping/getting canine blood products, but feline products are hard to come by these days. You want to ensure you screen the pets properly for infectious diseases before committing to using that patient for blood donation. You want to ensure you develop a screening protocol in compliance with recognized experts, such as the AAFP guidelines, as well as the most up-to-date evidence-based clinical medicine information related to organisms transmissible via blood transfusions. Further, knowing the area where you live, the endemic diseases, and the prevalence rates can help guide your screening test selections.

All of the following would be considered core organisms to test for, except:



Correct answer: Toxoplasma gondii DNA

In addition to proper blood typing, all cats, due to the naturally occurring alloantibodies in some blood types and the severe risk of potentially lethal reactions without typing, typing, and crossmatching, have to be built into your program. Crossmatching may be skipped in dire straights if there is no previous blood transfusion in the patient's history or if they have had a transfusion two or more days earlier.

Which organisms should be tested in addition to the key pathogens varies with the clinical practice location. Knowledge of endemic concerns and epidemiology associated with those diseases is critical in deciding what other organisms to test for in your practice situation.

These primary pathogens should be included regardless of practice location.

- 1 Feline leukemia virus DNA
- 2. Feline Leukemia virus RNA
- 3. Feline immunodeficiency virus DNA
- 4. Bartonella species DNA
- 5. Mycoplasma haemofelis DNA (though inactivated during storage of whole blood for one week)

Additional screening should be based on incidence and prevalence data in your area.

- 1. 'Candidatus Mycoplasma haemominutum' (survives a week in stored blood); (low pathogenicity relative to other mycoplasma species)
- 2. 'Candidatus Mycoplasma turicensis' (low pathogenicity relative to other mycoplasma species)
- 3. Anaplasma phagocytophilum (If they reside in areas where Ixodes spp. ticks are of threat)
- 4. Anaplasma platys (If residing in areas with Rhipicephalus spp. ticks)
- 5. Cytauxzoon felis
- 6. Babesia felis
- 7. Ehrlichia canis
- 8. Leishmania infantum
- 9. Neorickettsia risticii

The 2021 AAFP ISFM Consensus Guidelines on the Collection and Administration of Blood Products does not recommend testing for coronavirus, Rickettsia felis, or Toxoplasma gondii, as transmission from blood products hasn't been documented. If you look at the reference by Jane E. Sykes' Greene's Infectious Diseases of the Dog and Cat Fifth Edition, they list coronavirus as a primary pathogen to evaluate for, with a publication date of 2023. So, the consensus about testing for coronavirus obviously isn't 100% across the board. However, if it isn't transmitted through blood or is already part of a pre-established lab profile, there isn't necessarily harm in evaluating for its presence. Care needs to be taken with the interpretation of results. But the interpretation of PCR test results is a question unto itself.

interpretation of PCR test results is a question unto itself.

A cat presents to you with signs that you think are consistent with hypothyroidism. Despite knowing that it is very rare in cats, we know it can occur for all of the following reasons, except:

Increased response by the thyroid gland to TSH
latrogenic
Dyshormonogenesis
Thyroid dysgenesis

Correct answer: Increased response by the thyroid gland to TSH

Clinical signs of hypothyroidism in cats with significant clinical disease may include lethargy, dullness, hypothermia, decreased appetite, bradycardia (uncommonly), non-pruritic, and seborrhea sicca. Obesity can also occur, especially in those with the iatrogenic form. Unlike dogs, who may develop bilateral focal alopecia, if cats develop alopecia, they tend to get focal areas of alopecia over the caudal hocks, craniolateral carpi, dorsal, and lateral tail bases. Many cats with iatrogenic have none or mild signs. Cats with juvenile-onset/congenital forms have significant signs, including severe lethargy, mental dullness, constipation, poor appetite, disproportionate dwarfism, and bradycardia.

Some congenital forms of hypothyroidism may develop, though rare in cats. These include dyshormonogenesis (intrathyroidal defects in thyroid hormone biosynthesis), thyroid dysgenesis, and failure of the thyroid gland to respond to TSH (not an increased response).

Iatrogenic remains the primary cause of hypothyroidism in cats. Usually, a consequence of overtreating hyperthyroid cats is either via radioiodine therapy, antithyroid drug (methimazole) administration, or thyroidectomy.

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A four-MO FI Border Collie is adopted by an older couple who didn't do their research before getting this breed. They also hadn't had a puppy in a long time and had forgotten what it was like. The puppy is crate trained and comfortable in it, so there are no issues when they aren't home. However, the puppy is being destructive, and the owners are frustrated and have been using punishment to no avail, unsurprisingly. Behaviors include chewing on the furniture right in front of them, stealing napkins off the table, grabbing stuff off the counters and the newest — digging up plants and veggies in the garden. You discuss possible behavior modification options with the owner and educate them about the breed and appropriate age-related behaviors.

While anxiety or phobias may contribute to the behaviors described, they are all ageappropriate and, thus, you would advise all of the following, except:

Keep the dog confined at home when they cannot watch the dog

Provide daily exercise such as regular walks

Reward-based, positive training (e.g., cooperative care training, clicker training)

Use feeder toys and consider freezing a tasty treat in a kong or similar toy

Correct answer: Keep the dog confined at home when they cannot watch the dog

Sometimes time outs may be needed. This goes for kids, parents, and even dogs. However, this should be as a last resort to keep the pet safe, not as the go-to way to limit destructive behaviors. This puppy is just that, a puppy. Age-appropriate behaviors include chewing, digging, stealing, and others. They need to be taught using positive reinforcement, not punishment, what behaviors are acceptable and what aren't, and provided with an outlet and something to do instead of those behaviors. Providing feeder toys to slow down meals and make them work for their kibble, freezing kongs with tasty treats in them, and reward-based training keep them occupied and stimulated. Regular exercise (not just sending them out in a fence in the yard, but walking on a leash for 20–30 minutes at least once per day can help). For dogs that dig, one may need to separate them from that area with a barrier and even provide them with their own safe place where you allow them to dig. For dogs that chew, redirecting that behavior to a favorite toy or another safe object may also be beneficial. With this puppy, most likely, it is just being a puppy. The breed requires a lot of exercise and mental stimulation. These owners need to find a way to encourage healthy behaviors, stimulate the dog's mind and body and permit it to still engage in normal puppy learning methods in the environment.

peaceful co-existence.						