(asymptomatic) mitral valve regurgitation. Relevant inclusion criteria for the trial that demonstrated this effect were a vertebral heart sum > 10.5, an echocardiographic left atrial–aortic ratio > 1.6, and left ventricular enlargement.

- ACE inhibition may have a positive effect on the time to development of stage C HF in canine patients with left atrial enlargement due to mitral valve regurgitation.

- Evidence that medical therapy slows the progression of HCM is lacking.

### Technician Tips

Teaching owners to keep a log of their pet’s testing respiratory rate can allow early detection of HF decompensation so that medications can be adjusted and hopefully hospitalization for acute HF can be avoided.

### Client Education

Management of the veterinary patient with chronic HF requires careful monitoring and relatively frequent adjustment of medical therapy (see client education sheet: How to Count Respirations and Monitor Respiratory Effort)

### SUGGESTED READING


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**Editor:** Meg M. Sleeper, VMD, DACVIM

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### Heart Murmur, Incidental Finding

#### Basic Information

**Definition**

A heart murmur that is detected in the process of an examination that was not initially directed at the cardiovascular system

**Synonym**

Asymptomatic heart murmur

**Epidemiology**

**Species, Age, Sex**

Any species, all ages, both sexes

**Genetics, Breed Predisposition**

Predispositions mirror those of the causative cardiac diseases (pp. 263, 505, 657, 658, 764, 844, and 948).

**Risk Factors**

- Structural heart disease
- Anemia
- Youth

**Clinical Presentation**

**History, Chief Complaint**

- By definition: identified in patients that are presented for noncardiovascular concerns, such as annual wellness exams, noncardiac medical concerns, or preanesthetic evaluation.
- Although no historical signs are associated with the murmur, misleading or overlapping signs are common, including cough, exercise intolerance, and others, which can be caused by unrelated comorbidities.

**Physical Exam Findings**

- Heart murmur (by definition), which is described according to timing, grade, and point of maximal intensity (p. 414).
- Auscultatory features of murmurs that are nonpathologic (see Differential Diagnosis below) classically meet the six S criteria, which are typically systolic, soft (grade 1-2/6), sensitive (prone to change in intensity with heart rate or body posture), short (midsystolic), single (unaccompanied by other abnormal sounds), and small (not widely radiating).

**Etiology and Pathophysiology**

- A heart murmur is caused by turbulent blood flow in the heart (p. 414).
- Identifying the timing, location, and intensity of the murmur may be straightforward or challenging; uncertainty favors pursuing diagnostic testing.
- The presence of a heart murmur does not warrant treatment. Rather, determining its cause (definitively or presumptively) can lead to an assessment of whether treatment is indicated.

### Diagnosis

**Diagnostic Overview**

First, an incidentally detected heart murmur is pursued through careful characterization of the murmur’s timing, grade, and point of maximal intensity. Second, these characteristics, combined with the patient’s signalment, may provide a strong suspicion of a likely underlying cause. If so and the veterinarian’s tentative diagnosis is of a benign process, the client is satisfied with this opinion without confirmation, and the animal is not to be used for breeding nor subjected to cardiovascular stress, diagnostic testing is not essential. Otherwise, diagnostic testing should be pursued.

**Differential Diagnosis**

Murmurs may be nonpathologic (the heart is structurally normal) or pathologic (caused by a structural heart lesion):

- Nonpathologic (benign) murmurs are further described as functional if a plausible physiologic cause is detectable (e.g., anemia) or as innocent if no cardiac or extracardiac cause for the murmur can be identified.
- Pathologic murmurs can be caused by any cardiac disorder of any degree and do not automatically indicate a severe condition.

**Initial Database**

- Thoracic radiographs may be considered as the initial diagnostic test in small- to medium-breed dogs with systolic murmurs that are loudest over the mitral valve region.
- An echocardiogram should be considered for any adult animal with one or more of the following: uncertain or unusual murmur characteristics, murmur characteristics suggesting a form of heart disease that requires initiation of treatment, large-breed dog (auscultation and thoracic radiographs have low specificity for individual cardiac disorders), impending cardiovascular stress (e.g., plane travel, general anesthesia), breeding prospects, or owner who wishes to have confirmation of the cause of the murmur.

*An echocardiogram should be considered for puppies and kittens with a murmur that is grade 3/6 or louder, that is diastolic or continuous, that obscures the second (or both) heart sounds, that radiates to the carotid region or is loudest over the left apex or right hemithorax, or that is a direct relative of an animal with congenital heart disease.*

- NT-pro-BNP testing in cats can raise or lower the likelihood of structural heart disease (notably cardiomyopathy) as the cause of the murmur.

### Treatment

**Treatment Overview**

Because a murmur is a physical finding alone, no treatment is warranted.

**Acute and Chronic Treatment**

Initiation of treatment in the absence of a diagnosis is not appropriate. It can lead to administration of medications a patient does not need (or that are contraindicated), cause unnecessary expense, and cause adverse treatment effects.
ADDITIONAL SUGGESTED READINGS


RELATED CLIENT EDUCATION SHEETS

Consent to Perform Echocardiography
Dilated Cardiomyopathy
Heart Failure
How to Count Respirations and Monitor Respiratory Effort
Mitra/Tricuspid Regurgitation Due to Myxomatous Heart Valve Disease
Possible Complications
Overinterpretation or underinterpretation of incidentally detected heart murmurs can lead to failure to provide an accurate treatment plan and prognosis.

PROGNOSIS & OUTCOME
Because incidentally detected murmurs occur in animals without associated clinical signs, the prognosis is often fair to good. Many disorders are progressive, but some (notably patent ductus arteriosus) lend themselves to being cured. The exact prognosis therefore depends on establishing the underlying cause and its degree of severity.

PEARLS & CONSIDERATIONS
Comments
• 25%-69% of cats with heart murmurs have no detectable heart disease.
• Treatment of a heart murmur is never indicated. The murmur is a clue, and the cause to which the clue is pointing may or may not benefit from treatment.

Technician Tips
Cats routinely have heart murmurs that are heart-rate dependent or that can vary in intensity between anesthesia and being awake. These characteristics are typical of physiologic murmurs, but it is impossible to be certain of the murmur’s cause (pathologic or nonpathologic) without an echocardiogram.

SUGGESTED READING

Heart Murmur, Physiologic

BASIC INFORMATION

Definition
Heart murmurs not associated with cardiac disease

Synonyms
Innocent murmurs, flow murmurs, nonpathologic heart murmurs

Epidemiology
SPECIES, AGE, SEX
Physiologic heart murmurs are common in puppies and kittens, and these generally disappear by 4-6 months of age. Other causes for murmurs unrelated to heart disease can be detected at any age.

GENETICS, BREED PREDISPOSITION
• Hound dogs (e.g., greyhound, Italian greyhounds, saluki) and, in general, athletic dog breeds are particularly prone to developing physiologic heart murmurs.
• Boxer dogs

RISK FACTORS
• Athleticism
• Anemia
• Other high cardiac output conditions (anxiety, hyperthyroidism, fever)

ASSOCIATED DISORDERS
Commonly associated with severe anemia

Clinical Presentation
HISTORY, CHIEF COMPLAINT
• Physiologic murmurs in puppies/kittens or athletic dogs are found during routine checkups.
• Patients with murmurs caused by anemia or fever can show clinical signs associated to these underlying conditions.

PHYSICAL EXAM FINDINGS
• These murmurs are more easily auscultated over the left heart base, occur during systole, and are usually soft (<III/VI).
• Pale mucous membranes may be evident in patients with physiologic heart murmurs caused by anemia.
• Fever may cause a physiologic murmur.

Etiology and Pathophysiology
• The genesis of a murmur is affected by multiple factors; structural heart disease creates turbulence and/or increases blood velocity due to valvular leakage (regurgitation), abnormal shunts, or obstructive lesions (p. 414).
• Physiologic heart murmurs result from a relatively high cardiac output. It is hypothesized that young patients have a relatively high stroke volume for their great vessels, causing physiologic murmurs. As these patients grow, their great vessels enlarge and the murmur disappears, usually by 6 months of age.
• Changes in blood properties, such as its viscosity or density, can also lead to heart murmurs in normal hearts. In patients with anemia, the combination of decreased blood viscosity due to a low hematocrit and an increased stroke volume can result in a physiologic murmur.
• Greyhounds and other athletic hunting dogs may have a soft, basilar systolic murmur that is physiologic. An echocardiogram, which is necessary to rule out a pathologic cause for the murmur, may demonstrate transaortic velocities that are slightly increased but still in the normal range and a normal cardiac structure.
• Although boxer dogs are predisposed to subaortic stenosis (SAS), these dogs also have an increased prevalence of physiologic murmurs that are thought to be due to a relatively smaller left ventricular outflow tract without other changes consistent with SAS.
• Emotional stress also increases cardiac output and should be taken into consideration when auscultating and/or echoing these patients.

DIAGNOSIS

Diagnostic Overview
Physiologic heart murmurs cannot be diagnosed solely by auscultation, and an echocardiogram is necessary to rule out structural heart disease. However, the echocardiogram may be delayed if there is evidence supporting a cause for physiologic murmur (e.g., if severe anemia exists, echocardiogram may be postponed to see if resolution of anemia results in resolution of murmur).

Differential Diagnosis
Other causes of heart murmurs: congenital (pulmonic or subaortic stenosis, ventricular septal defects, atroventricular valve stenosis), acquired (degenerative valvular disease, secondary valvular regurgitation due to dilated, hypertrophic, or restrictive cardiomyopathy, bacterial endocarditis).

Initial Database
• Echocardiogram
• Hematocrit

TREATMENT

Treatment Overview
• No treatment is necessary for pediatric or athletic murmur.
• Patients with physiologic murmurs due to anemia or fever need treatment for the underlying systemic condition.
Hypercalcemia, Idiopathic Feline

BASIC INFORMATION

Definition
This poorly understood condition is the most common cause of increased ionized calcium (iCa) in cats.

Epidemiology
SPECIES, AGE, SEX
Cats of any age (often 5-10 years) and either sex

GENETICS, BREED PREDISPOSITION
Long-haired cats appear to be overrepresented.

RISK FACTORS
Genetics, diet, or the use of urinary acidifiers

ASSOCIATED DISORDERS
Calcium oxalate urolithiasis, chronic kidney disease (CKD)

Advanced or Confirmatory Testing
• Ionized calcium (i.e., biologically active component of the total serum calcium): normal or low with CKD, increased with most other causes of hypercalcemia (e.g., PHPTH, hypercalcemia of malignancy, vitamin D intoxication)
• Serum PTH and PTHrP concentrations during hypercalcemia
  - PTH should be undetectable in response to hypercalcemia.
  - PTH values within or above reference range are consistent with PHPTH.
• Undetectable PTH and detectable PTHrP concentrations are consistent with hypercalcemia of malignancy.
• Serum vitamin D concentrations: if suspect intoxication (p. 164)
• Cervical ultrasound
  - Parathyroid glands should be ≈1.3-3.3 mm in greatest width (dogs and cats).
  - In dogs with PHPTH, a mass is typically identified involving one or more parathyroid gland(s), usually 4-8 mm in greatest diameter.
• Dogs with renal secondary hyperparathyroidism have enlargement of two, three, or all four parathyroid glands.
• Additional testing based on abnormalities identified (e.g., fine-needle aspiration of enlarged lymph nodes, fungal serology)

TREATMENT

Treatment Overview
Successful treatment of underlying cause lowers serum calcium. If (Ca • PO₄) is > 60, additional measures may be required. Rapid reduction in serum calcium, even with extremely increased values (15-23 mg/dL) is not necessary if (Ca • PO₄) is < 60, which is typical of PHPTH. Even when calcium is within reference range, if Ca • PO₄ is increased, nephron damage may ensue.

Acute General Treatment
Primary (most efficacious):
• IV fluid therapy (calcium free; avoid lactated Ringer’s solution)
  - Dilution of serum calcium and phosphorus concentrations, improved glomerular filtration rate
  - Twice maintenance plus dehydration deficit should be administered over the first 24 hours, assuming no heart disease, oliguria, or other factor predisposing to intolerance of volume load; adjust according to clinical signs.
• Furosemide 2-3 mg/kg IV q 4-8h. Calciuric diuretic (unlike thiazide diuretics or spironolactone) is not recommended for pets with renal insufficiency.
• Glucocorticoids (prednisone or dexamethasone): decrease intestinal calcium absorption, increase renal calcium excretion. Diagnostic samples (e.g., lymph node aspirate, bone marrow aspirate, liver biopsy) should be obtained before treatment because steroids may mask lymphoma.
  - Secondary therapies (more expensive and not often required):
    • Bisphosphonates
    • Calcitonin
    • Plicamycin
    • Cinacalcet

Chronic Treatment
Treat inciting cause

Possible Complications
Overcorrection (hypocalcemia), urolithiasis, nephron damage (if Ca • PO₄ > 60)

Recommended Monitoring
• Serum total and ionized calcium concentrations
• Renal parameters
• Serum electrolytes

PROGNOSIS & OUTCOME

• Varies; depends on ability to achieve normocalcemia and correct underlying cause
• Excellent for PHPTH

PEARLS & CONSIDERATIONS

Comments
• Remember, renal failure is not caused by hypercalcemia alone.
• Correcting total calcium concentration for hypoalbuminemia or hyperalbuminemia is not reliable (instead, measure serum ionized calcium concentrations directly).
• Oral consumption of calcium alone does not cause hypercalcemia.
• Hypercalcemic dogs that are ill are not likely to have PHPTH.

Technician Tips
• Urolithiasis related to hypercalcemia can cause urinary obstruction. Straining to urinate is an emergent condition.
• Hypercalcemic dogs should always have drinking water available and should be given ample opportunity to urinate.

SUGGESTED READING

AUTHOR: Edward C. Feldman, DVM, DACVIM
EDITOR: Leah A. Cohn, DVM, PhD, DACVIM

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Clinical Presentation

HISTORY, CHIEF COMPLAINT

- Usually an incidental finding (e.g., geriatric screening) or associated with vague clinical signs such as weight loss, diarrhea, constipation, vomiting, or anorexia
- The modest degree of hypercalcemia typical of the disorder is seldom associated with the most worrisome adverse effects of hypercalcemia (e.g., tissue mineralization).
- Sometimes, signs related to calcium oxalate urolithiasis (e.g., dysuria, peruria [p. 1014]) or concurrent CKD (e.g., polyuria/ polydipsia [pp. 167 and 169]) are noted.

PHYSICAL EXAM FINDINGS

No specific physical exam findings. Calcium oxalate urolithiasis can cause signs of urethral obstruction in some affected cats.

Etiology and Pathophysiology

- Extracellular total calcium fractions include biologically active iCa (=52%), protein-bound bound calcium (=40%), and calcium complexed to other molecules (=8%). Calcium balance is closely controlled in health through intestinal absorption, renal excretion, and redistribution from bone.
- As the name implies, the cause of ionized hypercalcemia in affected cats remains unknown.

DIAGNOSIS

Diagnostic Overview

Typically, total calcium is measured first, and if above the upper end of the reference range, iCa is measured. If that too is above the reference range, attempts should be made to identify a cause of hypercalcemia. Idiopathic hypercalcemia is a diagnosis of exclusion.

Differential Diagnosis

Hypercalcemia (pp. 491 and 1232)

Initial Database

- Serum biochemistry profile: increased total calcium; phosphorus within reference range
  - Concurrent CKD associated with azotemia, hyperphosphatemia
- Ionized calcium: usually mild to moderate increase (80% between 1.5 and 1.75 mmol/L; 1.4 mmol/L is the upper end of the reference range)
  - If iCa cannot be measured quickly in house, sample should be collected anaerobically and transported on ice.
  - Exposure of sample to air can lead to loss of CO₂, resulting in decreased iCa.
  - Lactic acid accumulation alters the pH of stored samples, resulting in increased iCa.
  - CBC: unremarkable
  - Urinalysis: variable urine specific gravity, possible calcium oxalate crystalluria
  - Total thyroxine (T₄): unremarkable
  - Thoracic and abdominal imaging: rule out neoplasia

Advanced or Confirmatory Testing

Serum parathyroid hormone (PTH), parathyroid hormone–related protein (PTHrP), vitamin D profile:
- PTH: below or near the lower end of the reference range
- PTHrP: typically below limits of detection
- Vitamin D: 25(OH)D₃ and 1,25(OH)₂D₃ within reference range

TREATMENT

Treatment Overview

Because the degree of hypercalcemia is typically modest, emergent efforts to reduce calcium are not required. After other causes of hypercalcemia have been ruled out, dietary therapy is typically begun. If unsuccessful, medical management is attempted. Concurrent urolithiasis and/or CKD must be addressed, if present.

Acute General Treatment

Rarely, calcium oxalate urolithiasis results in urethral obstruction, requiring emergency intervention (p. 1009)

Chronic Treatment

- Many cats can be managed with dietary therapy alone.
- If ionized hypercalcemia persists after a 6-week diet trial, medical therapy with glucocorticoids or bisphosphonate drugs is recommended.
  - Prednisolone (not prednisone) 0.5-1 mg/kg PO q 12-24h. Avoid use until diagnostic testing is complete.
  - Alendronate 5-20 mg/CAT PO q 7 days. Begin with lower dose, and titrate up as needed. Administer after a 12-hour fast. Pills should not be cut because they can be highly irritating to the oral and esophageal surfaces. Follow pill with 5-10 mL of water to reduce risk of esophageal stricture. Liquid formulations are available but may not be palatable.
  - Occasionally, a combination of prednisolone and alendronate is required to control iCa.

Nutrition/Diet

- High-fiber diet and/or psyllium supplementation recommended
- Wet/canned foods preferred
- Oxalate prevention diets useful for cats with no evidence of CKD
- Renal diets are appropriate for cats with concurrent azotemia.

Possible Complications

- Uncontrolled hypercalcemia may result in calcium oxalate urolithiasis.

• Alendronate may cause esophageal stricture or irritation of mucous membranes.
• In humans, alendronate may cause osteonecrosis of the mandible and maxilla; if dental work is required, it should be completed before starting alendronate.

Recommended Monitoring

Recheck iCa 6 weeks after starting diet trial or 1-2 weeks after any change in medical therapy. Once controlled, iCa should be rechecked q 4-6 months. Serum chemistry profile (azotemia) and urinalysis (crystalluria) should be checked q 6-12 months.

PROGNOSIS & OUTCOME

With treatment, excellent. Without treatment, urolithiasis remains a concern.

PEARLS & CONSIDERATIONS

Comments

- Other causes of hypercalcemia may be associated with life-threatening disease and should be ruled out before instituting treatment for idiopathic hypercalcemia.
- Severe hypercalcemia is seldom caused by idiopathic hypercalcemia.
- Renal damage associated with hypercalcemia is related to the calcium × phosphorus product more than to the iCa. Because hypercalcemia is mild and phosphorus is within reference range, kidney damage is unlikely with idiopathic hypercalcemia alone.
- It is possible for a cat to have both CKD and idiopathic hypercalcemia, which can confuse the diagnosis (e.g., CKD can cause increased total calcium but normal iCa).
- Use of formulas to adjust calcium concentration based on albumin is not appropriate for cats with hypercalcemia. Instead, ionized calcium concentrations should be measured directly.

Technician Tips

Demonstrate how to properly administer medications, including giving water afterward to minimize the risk of esophageal stricture with alendronate.

Client Education

Proper administration of medications

SUGGESTED READING


AUTHOR: Leah A. Cohn, DVM, PhD, DACVIM
EDITOR: Etienne Côté, DVM, DACVIM
ADDITIONAL SUGGESTED READINGS


Hypernatremia

BASIC INFORMATION

Definition
A serum sodium (Na⁺) concentration above the reference range; caused by net water loss (most common) or Na⁺ gain

Epidemiology
SPECIES, AGE, SEX
No species, age, or sex predisposition

GENETICS, BREED PREDISPOSITION
Essential adipsic hypernatremia rarely reported in schnauzers, other dog breeds, and cats; may have a genetic basis

RISK FACTORS
• Diuresis in the absence of adequate available water replacement
• Excessive water loss from nonrenal sources (e.g., vomiting, diarrhea, burns)
• Acute administration/consumption of large amounts of Na⁺ (e.g., sea water consumption)

ASSOCIATED DISORDERS
Essential adipsic hypernatremia, diabetes insipidus, central nervous system (CNS) damage

Clinical Presentation
DISEASE FORMS/SUBTYPES
• Can be acute or chronic; accumulation of idogenic osmols in chronic hypernatremia impact treatment
• Categorized by volume status as hypovolemic, normovolemic, or hypervolemic

Recommended Monitoring
• Monitor plasma TGs 4-8 weeks after initiation of low-fat diet; then every 6-12 months.
• Monitor hematologic/biochemical parameters with fibrates, niacin, or lovastatin.

PROGNOSIS & OUTCOME
• Successful management depends on adequate control of underlying disease(s) and reduction of plasma lipid concentrations.
• Cats with peripheral neuropathies generally have clinical signs resolve within 4-12 weeks of instituting diet change.

PEARLS & CONSIDERATIONS
Comments
• Hyperlipidemia in patients fasted > 12 hours is abnormal.
• Lipemic plasma is an indication of hypertriglyceridemia, not hypercholesterolemia.
• Hyperglycemia is a more sensitive indicator of hypertriglyceridemia, not hypercholesterolemia.

DIAGNOSIS

Diagnostic Overview
Hypernatremia may be suspected in depressed animals with conditions known to predispose to hypernatremia, or it can be an incidental finding on serum biochemical profile. Signs of hypernatremia may not be apparent until Na⁺ > 175-180 mEq/L.

Acute hyperosmolality causes brain cells to shrink because intracellular water is pulled into the extracellular fluid space, resulting in rupture of vessels and intracranial bleeding.

If hypernatremia comes about more slowly, the brain can adapt through production of idogenic osmols, which hold water volume in the brain cells.

Overly rapid correction of long-standing hypernatremia causes water to be pulled into the brain cells by idogenic osmols, resulting in brain swelling and neurologic damage.

Causes of hypernatremia (p. 1237)
• Pure water deficit: normovolemic hypernatremia (e.g., water deprivation [especially with diabetes insipidus], adipsia)
• Hypotonic fluid loss (most common): hypovolemic hypernatremia (e.g., diabetes mellitus, postobstructive diuresis, gastrointestinal [GI] fluid loss, burns, chronic kidney disease)
• Increased Na⁺ retention or intake: hypervolemic hypernatremia (e.g., hypertonic enema solutions, sea water consumption, excess hypertonic saline infusion)

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ADDITIONAL SUGGESTED READINGS


Fletcher JM: Diagnosis and management of hyperlipidemia. In Proceedings from the 14th annual Southwest Veterinary Symposium, 2016, Fort Worth, TX.


RELATED CLIENT EDUCATION SHEETS

Consent to Perform Abdominal Ultrasound
How to Change a Pet’s Diet
Differential Diagnosis
- Encephalopathic signs: hypoglycemia, hypernatremia, hepatic encephalopathy, uremia, intoxications, hypoxia, CNS disorders
- Hypernatremia: pseudohypernatremia occurs occasionally in hyperproteinemic or hyperlipidemic animals. Confirm true hypernatremia with direct selective electrode measure.

Initial Database
- Review history for water consumption/thirst, urine production, possible salt ingestion/administration
- Serum biochemical profile
  - Na⁺ above upper reference range (by definition; usually Na⁺ > 157 mEq/L)
  - Hyperchloremia (common)
  - Azotemia (may accompany hypovolemia or kidney disease)
  - Hyperphosphatemia (may accompany kidney disease or sodium phosphate enema use)
  - Increased albumin in hemoconcentrated state
  - Serum osmolality (measured or calculated); always increased
- CBC: may show evidence of hemoconcentration
- Urinalysis, with urine osmolality (if available): hyposthenuria (e.g., diabetes insipidus), isosthenuria (e.g., kidney disease), or concentrated urine (e.g., salt intoxication, GI losses)

Advanced Diagnostic Testing
Additional testing is aimed at identification of the underlying cause of hypernatremia; choice of test depends on suspected cause. Common tests:
- Abdominal imaging: cause of vomiting or diarrhea, evaluation of kidneys and adrenal glands
- Brain imaging by MRI or CT: if hypothalamic lesion suspected
- Tests to confirm endocrinopathies, if indicated: diabetes insipidus (p. 250), hyperaldosteronism, diabetes mellitus (p. 251)

TREATMENT

Treatment Overview
Acute hypernatremia (<24-hour duration) can be corrected rapidly, but longer-standing hypernatremia must be corrected slowly (often over 48-72 hours at < 8-12 mEq/L per 24 hours). Because frequent measures of serum Na⁺ are required, animals with severe hypernatremia should be treated at 24-hour care facilities capable of monitoring electrolytes in real time.

Acute General Treatment
- See Hypernatremia Algorithm (p. 1428).
- For hypernatremia of short duration (<24 hours), rapid correction is appropriate (1.5-2 mEq/L/h) using no or low Na⁺ fluids (e.g., 5% dextrose, 0.45% sodium chloride, one-half strength lactated Ringer's solution)
- For hypernatremia lasting for > 24 hours, correct no more quickly than 0.5 mEq/L/h (12 mEq/L/day). This rate may be difficult to achieve with very-low-sodium fluids; keep in mind that even fluids with a Na⁺ concentration of 40 mEq/L (e.g., Normosol M, Plasma-Lyte 56) can provide necessary water with a lower Na⁺ content than the patient's serum. Frequent (i.e., q 2-4h) rechecks of Na⁺ are essential.
- For animals that are not vomiting and mentally appropriate, enteral water supplementation is useful.

Chronic Treatment
Address underlying cause of hypernatremia.

Possible Complications
Coma, seizures, and death

Recommended Monitoring
- During correction of chronic, severe hypernatremia, monitor serum Na⁺ q 2-4h to be sure correction is not overly rapid.
- Repeat neurologic evaluations at least daily; signs of overly rapid correction may not be apparent for 48 hours or more after treatment.

PROGNOSIS & OUTCOME

Comments
- Prognosis depends on underlying cause as well as appropriate treatment. Often, hypernatremia is completely reversible if treated appropriately in a timely manner.
- Guarded to grave after coma occurs

PEARLS & CONSIDERATIONS

Comments
- Hypernatremia is more often the result of water loss rather than Na⁺ gain.
- For slow-onset hypernatremia, correct slowly; for rapid onset of hypernatremia, correct rapidly.
- Hyperaldosteronism is rare and causes hypertension more often than hypernatremia (excess Na⁺ pulls fluid into vascular space).

Prevention
- Provide ample access to water for any animal with polyuria or salt access.
- Do not allow dogs to drink sea water.
- Mix generous amounts of water in moist food for animals with adipsia.

Technician Tips
Any animal with polyuria should be provided access to water at all times (or IV fluids if GI/oral intake is not allowed) during any hospital stay, even if brief.

Client Education
Stress the importance of free-choice water for polydipsic pets.

SUGGESTED READING

AUTHOR: Michael Schaer, DVM, DACVIM, DACVECC
EDITOR: Leah A. Cohn, DVM, PhD, DACVIM

GENETICS, BREED PREDISPOSITION
- Any breed
- Keeshond: inherited (autosomal dominant); a genetic test is available (http://ahdc.vet.cornell.edu/docs/PHPTInstructions.pdf).
- Hereditary neonatal PHPT has been reported in two German shepherd dogs.

Clinical Presentation
HISTORY, CHIEF COMPLAINT
- Polyuria/polydipsia (=50% dogs; ≈10% cats)
- Lower urinary tract signs (caused by infection or cystic calculi), including pollakiuria, stranguria, and hematuria (=50% of dogs)
- Weakness, lethargy (=40%-50% of dogs and cats)
- Inappetence (=25%-30% of dogs; ≈40% of cats), vomiting (=10% of dogs; ≈40% of cats)
- Some (=30% dogs) have no clinical signs; hypercalcemia is an incidental finding.

PHYSICAL EXAM FINDINGS
- Physical exam: typically unremarkable

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