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CARDIOLOGY & ENDOCRINOLOGY

Finding the Balance in Your Patients with Cardiovascular and Renal Disease

***Audrey K. Cook**, BVM&S, DACVIM, DECVIM*
***Ashley B. Saunders**, DVM, DACVIM (Cardiology)*
Texas A&M University

Cardiovascular and renal disease are commonly diagnosed in cats and dogs; incidence increases with patient age. Each condition is irreversible and progressive. These conditions are particularly challenging when they occur simultaneously; what is good for one system may be counterproductive for the other. For example, fluid therapy is routinely administered to patients with acute or substantial renal compromise but may be problematic for patients with cardiac disease; and multiple medications administered to patients with cardiovascular disease can affect the kidneys, electrolytes, and blood pressure. In this article, we describe the physiologic interactions between these 2 systems, disease classifications, techniques for detecting one condition in the presence of the other, and strategies for managing patients with these co-existing conditions.

DETECTING HEART DISEASE
Most heart disease in dogs is unmasked by clues detected during physical examination (eg, murmur, arrhythmia, or jugular pulses).

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PHYSIOLOGIC LINK BETWEEN HEART AND KIDNEYS

Both the heart and the kidneys play a role in volume regulation and can directly or indirectly influence endocrine responses that affect fluid balance. The kidneys are one of the body's primary "volume monitors." Glomerular filtration rate (GFR) is a surrogate indicator of extracellular volume status. If an animal is dehydrated or acutely volume depleted, renal perfusion will drop and GFR will decrease. This response triggers the release of renin from the juxtaglomerular apparatus in the kidney, the starting point for the renin-angiotensin-aldosterone system (RAAS) (**FIGURE 1**).

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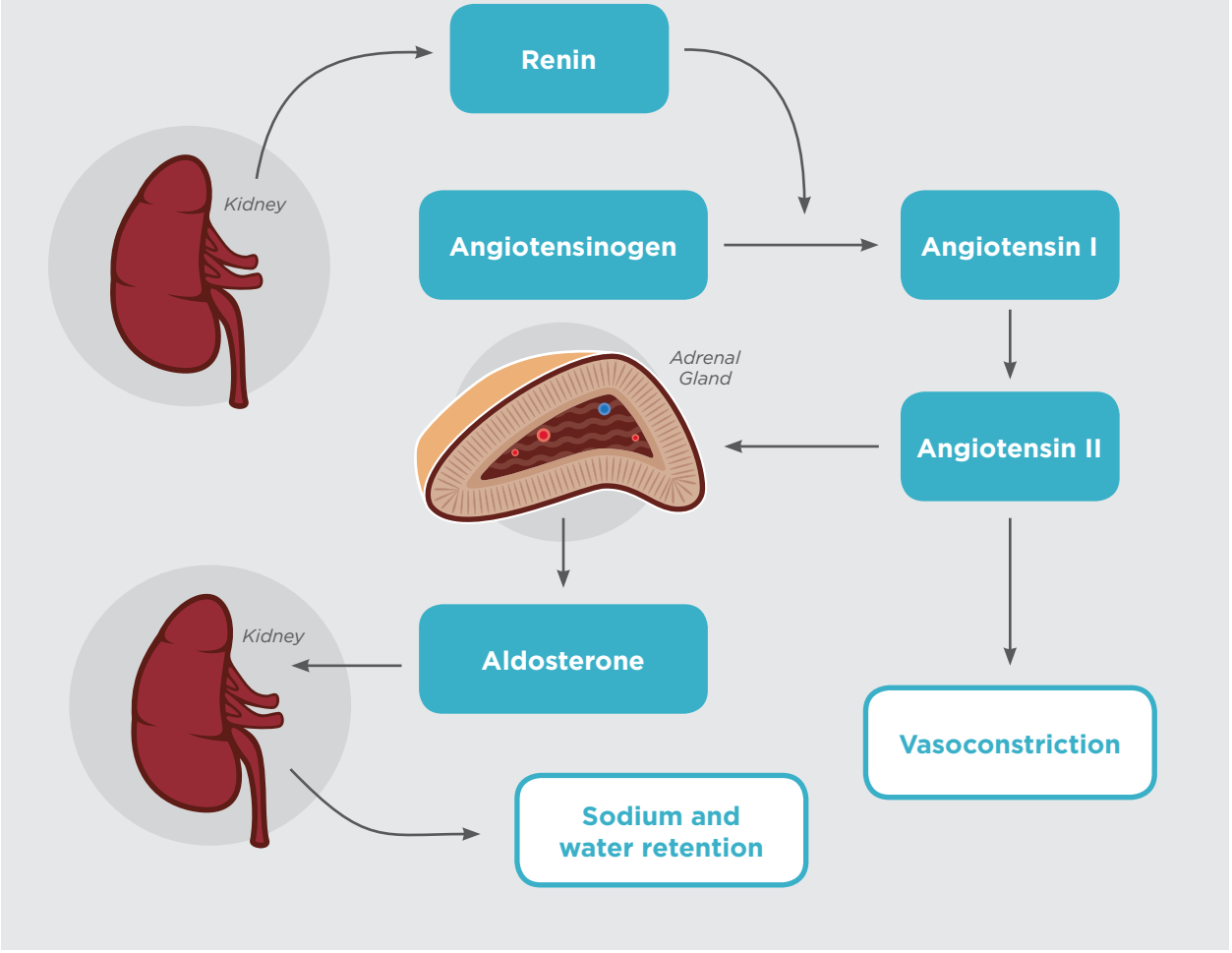
FIGURE 1. Renin-angiotensin-aldosterone system.



The RAAS

This system, the release of renin, angiotensin II, and aldosterone, is one of the most potent endocrine systems in the body and plays a key role in survival from acute injury or illness. Through widespread vasoconstriction triggered by angiotensin II and aggressive kidney reclamation of sodium and water, the RAAS helps restore and maintain blood pressure and volume.

Although the RAAS is a life-saving response in emergent circumstances, it can become maladaptive over time if the GFR decrease is not due to a decrease in blood volume. For example, a dog with dilated cardiomyopathy and reduced cardiac output will deliver less blood to the kidneys, despite an excess in total body volume. Decreased renal perfusion activates the RAAS, leading to vasoconstriction and inappropriate sodium and fluid retention. Vasoconstriction and volume retention may help restore GFR but adversely affect the heart and contribute to congestive heart failure (CHF).

Other Interactions

Although the RAAS is one of the major ways in which the kidneys and heart interact, they interact in other ways as well. Common processes associated with renal disease (eg, anemia, systemic hypertension, hypokalemia, or a hypercoagulable state) can negatively affect the cardiovascular system. Medical therapies designed to support one system may be deleterious to the other. For example, diuretic therapy to manage CHF in an azotemic animal with renal disease may further decrease GFR through volume depletion. In patients with renal disease, changes in blood flow and glomerular filtration may affect drug metabolism and clearance of drugs given for cardiovascular disease (eg, digoxin), resulting in increased risk for toxicity.

CLASSIFICATION OF CARDIOVASCULAR–RENAL DISORDERS

In 2015, an international group of 16 board-certified veterinary experts reviewed the cardiovascular–renal syndrome.[1] They defined cardiovascular–renal disorders as "disease, toxin, or drug-induced structural and/or functional damage to the kidney and/or cardiovascular system, leading to disruption of the normal interactions between these systems, to the ongoing detriment of one or both." The group proposed the following veterinary classifications: primarily cardiovascular, primarily renal, and concurrent disease.

Primarily Cardiovascular Disease Leading to Kidney Dysfunction

Mechanisms by which cardiovascular disease can lead to kidney dysfunction include activation of the RAAS and the sympathetic nervous system or glomerular injury secondary to deposition of antigen–antibody complexes.

Examples include

- Systemic hypertension leading to glomerular disease
- Cardiac shock, low cardiac output, and systemic hypotension leading to decreased renal perfusion, azotemia, and acute kidney injury (AKI)
- Heartworm infection leading to glomerulonephritis
- Caval syndrome leading to AKI

Primarily Renal Disease Leading to Cardiovascular Dysfunction

Mechanisms by which renal disease can lead to cardiovascular dysfunction involve fluid volume, hemodynamic status, electrolyte disorders, and altered clearance rates for cardiac drugs.

Examples include

- Kidney-mediated systemic hypertension, leading to increased afterload, left ventricular hypertrophy, and worsening mitral or aortic valve insufficiency
- Volume overload, leading to congestion or systemic hypertension
- Hypokalemia or hyperkalemia, leading to cardiac arrhythmias
- Uremic hypodipsia, anorexia, or emesis, leading to volume depletion and reduced cardiac output and perfusion
- Activation of the RAAS, leading to sodium and water retention and cardiac and vascular remodeling or congestion
- Anemia secondary to chronic kidney disease (CKD), leading to volume overload and reduced cardiac tissue oxygenation

Concurrent Disease of Both Systems

This classification applies to cardiovascular and renal disease that develop independently or arise from a common trigger.

Examples of common triggers include:

- Septic or neoplastic emboli leading to renal and cardiac infarction
- Gastric dilation and volvulus leading to cardiac arrhythmias and azotemia

RECOGNIZING CARDIOVASCULAR AND RENAL DISEASE

Cardiac and renal disease develop differently in dogs and cats, thereby limiting our ability to generalize among species. In dogs, common cardiac conditions include degenerative mitral valve disease and dilated cardiomyopathy; in cats, they are hypertrophic cardiomyopathy and systemic hypertension. In dogs, common renal conditions include glomerular disease, pyelonephritis, and acute tubular injury; in cats, they include idiopathic tubulo-interstitial disease.[2]

Recognizing Cardiovascular Disease in Dogs and Cats with Renal Disease

Cardiovascular disease is indicated by a history of a respiratory problem, collapse, abdominal distension, heart murmur, arrhythmia, muffled heart sounds, or abnormal pulses. Appropriate diagnostics to assess the cardiovascular system (eg, echocardiography, electrocardiography, blood pressure measurement, and thoracic radiography, with or without a heartworm test) should be accompanied by a systemic health evaluation, including a complete blood count, biochemical profile (with or without thyroid testing), and urinalysis.

Dogs

Most heart disease in dogs is unmasked by clues detected during physical examination (eg, murmur, arrhythmia, or jugular pulses). At a minimum, if a murmur is heard, 2-view thoracic radiography

should be performed to assess heart size and pulmonary vasculature before fluid therapy is begun. Echocardiography is recommended for dogs with radiographic evidence of cardiomegaly and for breeds at risk for dilated cardiomyopathy. Electrocardiography is indicated to further evaluate arrhythmias.

Cats

Cats may have substantial preclinical heart disease without any obvious abnormalities detected during physical examination. Even for cats with significant cardiomyopathy, thoracic radiographs may be unremarkable. Detection of a murmur, gallop rhythm, arrhythmia, or radiographic cardiomegaly in a cat should prompt further evaluation of heart disease by echocardiography. However, it is impractical to perform echocardiography for every cat before starting fluid therapy. Measuring N-terminal pro B-type natriuretic peptide (NT-proBNP) may be helpful; elevated values suggest a higher likelihood of underlying heart disease warranting further evaluation. This biomarker has been studied in cats with heart disease and can identify cats with moderate to severe echocardiographic changes.[3,4] Although NT-proBNP concentrations can be elevated in cats with hypertension, hyperthyroidism, and severe azotemia, concentrations are not significantly elevated in normotensive cats with mild to moderate (stages 1 to 3) CKD and are not useful indicators of hypertension.[5,6]

Recognizing Renal Disease in Dogs and Cats with Cardiovascular Disease

In dogs and cats, a routine cardiovascular workup and systemic health examination will probably identify most concurrent renal compromise, although some abnormalities may be subtle. For example, a dog may have International Renal Interest Society stage 1 or 2 CKD (**TABLE 1**)[7] but still have a serum creatinine concentration within the reference range (<1.6 mg/dL [144 mcmol/L]). The only evidence of intrinsic renal disease may be poorly concentrated urine (specific gravity <1.020). Note that cats and dogs receiving diuretic therapy for heart failure will have a low urine specific gravity; thus, urine specific gravity is best evaluated before diuretic therapy is initiated.

For patients with known renal compromise (eg, azotemia, proteinuria), the need for cardiovascular diagnostics and imaging should be carefully considered. For dogs and cats with all forms of kidney disease, measurement of systemic blood pressure is always indicated to identify hypertension that can cause end-organ damage.

Dogs

Sometimes, a useful biomarker for early CKD in dogs is serum symmetric dimethylarginine (SDMA). This marker correlates well with measurements of GFR and usually increases before serum creatinine levels suggest renal compromise.[8] Alternatively, a dog with well-

TABLE 1 International Renal Interest Society Staging Scheme for Chronic Kidney Disease in Cats and Dogs[7]

STAGE	SERUM CREATININE	COMMENTS
1	<1.6 mg/dL (cat) <1.4 mg/dL (dog)	No clinical signs but evidence of renal issues, such as poorly concentrated urine, proteinuria, and abnormal renal palpation or images
2	1.6–2.8 mg/dL (cat) 1.4–2.0 mg/dL (dog)	Mild renal azotemia; minimal clinical signs
3	2.9–5.0 mg/dL (cat) 2.1–5.0 mg/dL (dog)	Moderate renal azotemia; variable clinical signs
4	>5.0 mg/dL (cat) >5.0 mg/dL (dog)	Severe renal azotemia; substantial compromise

concentrated urine but substantial proteinuria may have renal disease that might be missed without further testing (eg, urine protein:creatinine ratio). For dogs with proteinuria, heartworm testing is appropriate.

Cats

Because cats naturally have well-concentrated urine, early CKD is often overlooked. Specific gravity <1.035 strongly suggests CKD in this species, and further staging with renal imaging and urine protein:creatinine ratio, with or without urine culture, is indicated. In cats with muscle wasting, serum SDMA concentrations may be a more accurate reflection of renal function than creatinine values.[8]

MANAGING PATIENTS WITH CONCURRENT CARDIOVASCULAR AND RENAL DISEASE

Balancing management of patients with concurrent cardiovascular and renal disease is challenging. You may need to prioritize one organ system above the other, bearing in mind that overall treatment goals are to maintain appetite and body condition, decrease clinical signs, and provide a good quality of life for the patient.

Dogs

If the Primary Condition is Cardiovascular Disease

Understanding the characteristics of common heart diseases in dogs can help predict which complications are more likely. For example, dogs with dilated cardiomyopathy have reduced ventricular systolic function, leading to reduced cardiac output and possible systemic hypotension, which may reduce GFR and trigger renin release. Therapeutic goals for all dogs with reduced ventricular systolic function, including dilated cardiomyopathy, are to improve ventricular function with positive inotropes to maintain adequate cardiac output, blood pressure, and renal perfusion. For dogs with CHF, medical therapies should be considered carefully; diuretics should be prescribed at the lowest dose necessary to resolve clinical signs. Recommended therapy for CHF in dogs typically consists of furosemide, pimobendan, and an angiotensin-converting enzyme (ACE) inhibitor.[9] However, multiple medications used to manage cardiovascular disease (eg, diuretics, ACE inhibitors, and angiotensin-receptor blockers) can affect the RAAS and the kidneys, alter electrolytes, and influence blood pressure. For some patients, ACE inhibitors may need to be decreased or discontinued if poorly tolerated and/or if they substantially increase serum creatinine levels.

Therefore, kidney function and electrolytes should be assessed before starting cardiac medications, and creatinine and electrolytes should be re-assessed within 10 to 14 days. Electrolyte changes can affect renal function and contribute to arrhythmias and should be identified and addressed promptly (eg, parenteral or oral potassium supplementation for hypokalemia).

When multiple cardiovascular medications are started simultaneously, patients with concurrent kidney disease are vulnerable to acute azotemia because of sudden changes in volume status and derangements in renal autoregulatory mechanisms. There is little consensus on how much of a serum creatinine increase is acceptable, although most clinicians accept a <30% increase from baseline.[10] If creatinine levels increase substantially (>30%) within the first 2 weeks of initial administration or during long-term use of an ACE inhibitor, the drug should be discontinued or its dose decreased. In some instances, azotemia in a patient with heart failure is tolerated and no adjustments are needed.

If the Primary Condition is Renal Disease

For dogs with AKI and degenerative mitral valve disease with normal heart size, fluid therapy should be administered to address the renal injury and optimize the chances of recovery, while closely monitoring for fluid overload. For dogs with primarily renal disease that show signs of volume overload during fluid therapy, fluids should be discontinued until the volume overload is resolved. Diuretics can be administered if necessary. Often, the first indication of volume overload is increased resting respiratory rate, which should be checked every hour during fluid administration. Heart rate may also increase if fluids are poorly tolerated. Monitoring for increased body weight can also be helpful.

Cats

If the Primary Condition is Cardiovascular Disease

Medical therapy for heart disease in cats is not well defined. ACE inhibitors, which block profibrotic effects of angiotensin II and aldosterone, have an inconsistent effect.[11] In a small retrospective evaluation of cats with preclinical and clinical

hypertrophic cardiomyopathy, enalapril did not adversely affect blood pressure or creatinine levels.[12]

Therapy for CHF typically consists of furosemide and an ACE inhibitor to reduce fluid accumulation.[13] Complications that can be encountered while managing heart failure and that contribute to reduced renal perfusion include systemic hypotension and dehydration/volume contraction associated with diuretic therapy. Several retrospective studies of cats with CHF from multiple forms of cardiomyopathy with and without systolic dysfunction have reported use of inodilator therapy (pimobendan).[14–16] Although this drug did seem to prolong survival, caution is recommended for its use in cats with known or suspected left ventricular outflow tract obstruction.[15] Whether pimobendan affects renal perfusion has yet to be investigated, but its use did not significantly affect creatinine concentrations.

Diuretic therapy can cause hypokalemia in cats and may be exacerbated by hyporexia. Low or borderline serum potassium concentrations should be addressed promptly with oral potassium gluconate because potassium depletion will affect renal function, impairing urine concentrating ability and possibly causing muscle weakness and acute myopathy.

If the Primary Condition is Renal Disease

In cats, fluid therapy for CKD may quickly unmask occult heart disease, regardless of administration route. SC fluid administration is not inherently safer than IV administration because fluids still enter the vascular compartment and cannot be discontinued if problems occur. For severely azotemic cats, long-term SC fluid therapy may help maintain well-being; however, for cats with modest CKD, it is best avoided because the inevitable sodium loading predisposes these cats to volume overload and can increase systemic blood pressure, irrespective of underlying cardiac function.

If the Dog or Cat has Concurrent Cardiovascular and Renal Disease

For dogs and cats with overt cardiac and renal disease, fluid therapy should be administered with caution. Fluid deficits should be estimated carefully and replaced over 24 hours. Concurrent maintenance fluid needs should be met with a lower sodium fluid, such as Normosol-M with 5% dextrose (hospira.com/en/products_and_services/iv_solutions/NORMOSOL_M_AND_DEXTROSE) or 0.45% saline[17] and discontinued as soon as possible. Fresh water should be provided at all times.

Systemic Hypertension in Dogs and Cats

High blood pressure is important to identify, manage, and monitor for patients with both cardiovascular and renal disease. Systolic blood pressure >160 mm Hg is considered abnormal, and pressure >180 mm Hg increases risk for damage to the eyes, brain, kidneys, and cardiovascular system.[18,19] In one study, systemic hypertension at initial CKD diagnosis was documented for ≈20% of cats.[20] Among cats with systemic hypertension, azotemia has been reported for 62% and left ventricular hypertrophy for 59%.[21,22] Persistently elevated systemic blood pressure and evidence of target organ damage warrant vasodilator therapy, typically with amlodipine, a dihydropyridine calcium-channel blocker. ACE inhibition results in dilation of the renal efferent arterioles, but the effects are generally insufficient for ACE inhibitors to serve as sole therapy for systemic hypertension. However, evidence supports the routine addition of an ACE inhibitor for all patients receiving long-term amlodipine therapy to counter the anticipated activation of the RAAS.[23] The combined use of amlodipine and an ACE inhibitor is certainly appropriate when blood pressure remains >160 mmHg or the urine creatinine:protein ratio indicates proteinuria; however, these 2 drugs should not be started simultaneously because severe azotemia can arise.

QUESTIONS TO GUIDE MANAGEMENT DECISIONS AND RECOMMENDATIONS

- **Is heart disease or kidney disease causing the clinical signs**? Prioritize the disease causing the most severe clinical signs.
- **Are both biochemistry analysis and thoracic radiography needed?** Usually yes, because the information they provide is often complementary. For example, when azotemia is present, radiographs will help indicate concurrent presence of active CHF (eg, pulmonary venous congestion, pulmonary edema, pleural effusion) and therefore will help guide decisions about diuretic dose reductions. If radiographs demonstrate evidence of CHF, information regarding current renal and electrolyte values can help guide decisions about increasing the diuretic dose, optimizing positive inotropic therapy, and reducing sodium intake.

- **What does urine specific gravity indicate in a patient receiving diuretics**? Although this measure can help identify renal disease, it will be low in patients receiving diuretics and cannot be used to differentiate prerenal and renal azotemia. For patients receiving diuretics, use surrogate markers of hydration status (body weight, total protein, albumin) instead.
- **Is blood pressure accurate and repeatable (a serious challenge in some cats)?** Are the blood pressure readings abnormally high or low? If too high, consider further workup and evaluation for systemic hypertension. If too low, determine whether the patient is clinically weak and the cardiac output low or whether the current medications are driving the blood pressure too low.
- **Which medications interact and what are the potential adverse effects?**
  - Many medications used to manage cardiac disease are vasodilators that can have an effect on blood pressure (eg, ACE inhibitors [eg, enalapril, benazepril], inodilators [eg, pimobendan], dihydropyridine calcium-channel blockers [eg, amlodipine], ß-blockers, and some antiarrhythmics [eg, procainamide]). High doses of a diuretic can lower blood pressure by reducing intravascular blood volume and further stimulating the RAAS.
  - Risk for azotemia and electrolyte abnormalities increases with combined use of ACE inhibitors and diuretics.
  - Administration of a nonsteroidal anti-inflammatory drug in patients with cardiovascular or renal disease may cause acute azotemia. Efferent arteriole vasodilation induced by an ACE inhibitor combined with afferent arteriole vasoconstriction induced by nonsteroidal anti-inflammatory drug–induced cyclooxygenase inhibition can result in reduced GFR and subsequent azotemia.
  - Changes in renal function can affect medication clearance, resulting in drug toxicity.
- **Is azotemia mild or severe?**
  - Mild azotemia, in the absence of clinical signs, may be well tolerated.
  - If azotemia worsens or is no longer mild, ACE inhibitors should be used at lower doses or discontinued. If a high diuretic dose is resulting in dehydration or worsening azotemia, consider whether it can be reduced.
  - Is fluid therapy carefully considered and appropriate? Fluid therapy for CKD can easily overload a diseased cardiovascular system. Prolonged fluid replacement is often poorly tolerated by patients with CKD because of poor renal perfusion and a limited ability to excrete surplus volume.
- **Is the patient eating and drinking enough?** A good appetite supports a good quality of life. Find palatable diets, avoid high-salt foods, and try appetite stimulants. In cats with concurrent cardiac and renal disease, feeding tubes may improve nutritional status and make administering multiple medications easier.

TAKEAWAYS:

- When you diagnose pathology in the cardiovascular or renal system, especially in an older animal, look for concurrent disease in the other system.
- If both systems are involved, focus your management strategy on the system causing the most severe clinical signs.
- Because therapy for one system may be counterproductive for the other, monitor the patient closely.
- According to history, physical examination findings, and diagnostic test results, adjust therapy (eg, fluids, electrolytes, drug dosages) as needed. TVP

Audrey K. Cook

Audrey K. Cook is a Diplomate of both ACVIM and ECVIM and is Board Certified in Feline Practice (ABVP). She is currently an associate professor of small animal internal medicine at Texas A&M, with particular interests in endocrinology, gastroenterology, feline medicine and endoscopy. Dr. Cook routinely speaks at national meetings and is a recipient of the Texas A&M University Distinguished Achievement Award in Teaching.

Ashley B. Saunders

Ashley B. Saunders, DVM, Diplomate ACVIM (Cardiology), is an associate professor of cardiology at Texas A&M University's College of Veterinary Medicine and Biomedical Sciences. Her interests include interventional cardiology and advanced imaging, heart failure management, and innovative teaching. She is a Montague Center for Teaching Excellence Scholar and has been awarded a Texas A&M University Distinguished Achievement Award in Teaching.

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CONTINUING EDUCATION

Finding the Balance in Your Patients with Cardiovascular and Renal Disease

LEARNING OBJECTIVES

After reading this article, participants will be able to explain the complex physiology between the heart and kidneys, classify cardiovascular and renal disorders according to primary disease process, create a diagnostic plan, and make management recommendations based on clinical considerations in an individual canine or feline patient.

TOPIC OVERVIEW

This article discusses cardio-renal disease in dogs and cats including diagnosis and management strategies.

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1. **Examples of cardiovascular disease processes that lead to kidney dysfunction include**
   a. Systemic hypertension
   b. Heartworm disease
   c. Low cardiac output
   d. All

2. **A biomarker that is useful for detecting early chronic kidney disease in dogs and cats and that correlates well with measurements of glomerular filtration rate is**
   a. Angiotensin II
   b. Cardiac troponin I
   c. N-terminal pro b-type natriuretic peptide (NT-proBNP)
   d. Serum symmetric dimethylarginine (SDMA)

3. **Idiopathic tubulo-interstitial disease is the most common renal pathology in cats.**
   a. True
   b. False

4. **You are presented with a 10-year-old Miniature Poodle with degenerative valve disease and stable CHF managed with benazepril, furosemide, and pimobendan. Blood pressure is within normal range. The most recent biochemistry panel documents azotemia. Select the ideal next step for this dog:**
   a. Decrease the pimobendan
   b. Decrease the furosemide
   c. Increase the benazepril
   d. Administer subcutaneous fluid therapy

5. **Systolic blood pressure increases the risk for target organ damage when it is**
   a. <120 mmHg
   b. 120–140 mmHg
   c. 140–160 mmHg
   d. >180 mmHg

6. **Nonsteroidal anti-inflammatory drugs cause _____ of the afferent arterioles of the kidneys, and ACE inhibitors cause _____of the efferent arterioles; the combined effect can reduce glomerular filtration rate.**
   a. Vasoconstriction, vasoconstriction
   b. Vasoconstriction, vasodilation
   c. Vasodilation, vasodilation
   d. Vasodilation, vasoconstriction

7. **Which of the following is a useful screening test for preclinical heart disease in cats?**
   a. Cardiac auscultation
   b. Measurement of systolic blood pressure
   c. Measurement of N-terminal pro b-type natriuretic peptide (NT-proBNP)
   d. Electrocardiography (ECG)

8. **Subcutaneous fluid therapy is superior to intravenous fluid therapy in cats with renal disease and concurrent cardiac disease.**
   a. True
   b. False

9. **The electrolyte disorder most often associated with diuretic use is**
   a. Hypokalemia
   b. Hypomagnesemia
   c. Hyperkalemia
   d. Hypernatremia

10. **Which of the following serum biochemical parameters should be rechecked within 14 days of starting an ACE inhibitor?**
    a. Phosphorus
    b. Sodium
    c. Creatinine
    d. Albumin

NOTE Questions online may differ from those here; answers are available once CE test is taken at **vetfolio.com/journal-ce**. Tests are valid for 2 years from date of approval.