Practical Approach to the Small Animal Emergency Patient

Michael Verschoor-Kirss





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USA

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Dedicated to my wife, the best thing veterinary medicine ever gave me, and our daughter, the best gift we gave each other.

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Preface

Welcome to the occasionally chaotic world of veterinary emergency medicine! Thank you for allowing me to be your guide for a portion of your journey as an emergency clinician. I remember starting off as an intern and struggling to find a resource that answered the questions I had on the clinic floor. My hope is that this book can serve as that resource for you. And to be honest, it also encompasses the questions I still have on a daily basis as a senior clinician!

The other inspiration for this book was the many conversations I've had with new graduates, junior emergency doctors, and interns about cases over almost a decade of emergency and critical care practice. I realized that they were all incredibly smart individuals who knew almost all of the answers, but what they didn't know were the questions they needed to ask. My goal is to help provide you with the questions and thought processes for managing the common emergencies you will see in practice.

There are pros and cons to this being a sole-author publication. This book reflects my opinion, clinical practice, and interpretation of primary literature. While you will see a large number of publications cited here, what you actually read is my interpretation of those studies. My expectation is that many people will disagree with certain aspects of what I've written and so always feel free to reach out with questions or concerns.

Finally, the goal of this book is not to be 100% comprehensive in its scope of disease. I could never include all of the information required to manage every single patient you will see. But even if this helps you manage one patient on your own, then I will consider this a success. If you can competently manage the common conditions in this book, you'll be well on your way to success in the ER. Ready to start? Then let's go!

Thanks for all you do,

MICHAEL
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Practical Approaches for the Emergent Patient

PART I

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The Triage Exam

CHAPTER 1

CRITICAL QUESTIONS

1. Could this pet have a life-threatening illness or injury based on its signalment and presenting complaint?
2. Is this pet in shock?
3. Are they having difficulty breathing?

INTRODUCTION

The triage exam starts when you, a technician, assistant, or receptionist, first interact with a pet and owner in the hospital. It is a combination of the presenting complaint, physical exam, and point of care lab work/imaging that you acquire prior to speaking with the owner. The goal of the triage exam is to detect and diagnose life-threatening issues that would lead to patient morbidity and mortality without immediate intervention. However, your triage exam is also helpful in guiding the first conversation with a pet owner because it can give you an idea as to how sick a pet is and what initial diagnostics and treatments may be necessary.

THE TRIAGE HISTORY

The triage history is never complete, and you're frequently receiving it secondhand. While occasionally a pet owner can quickly give you a definitive diagnosis (i.e. "my pet was just hit by a car"), many times the presenting complaint is vaguer, such as "he/she's just really lethargic." Therefore, it is important for both you and your staff to effectively detect the presenting complaints that may require emergent intervention.

Important questions to consider for pets who may benefit from a triage physical exam and/or emergent diagnostic testing:

1. Can the pet walk?
   The inability to ambulate will include many cardiovascularly unstable, intoxicated, and neurologically impaired animals. Pets with this presenting complaint should be immediately assessed using the triage physical exam (see below) to evaluate for shock and stability. Pets who have a history of "collapse" should similarly be evaluated, EVEN if they are currently walking. We will talk more specifically about the approach to the "down" pet and gurney patient in a later chapter.
2. Is the pet having difficulty breathing?
   Difficulty breathing can indicate upper airway obstruction, pulmonary pathology, pleural space disease, and metabolic instability. All pets with difficulty breathing should be evaluated using the triage physical exam, as the inability to appropriately ventilate and oxygenate can rapidly spiral and progress to cardiac arrest.
3. Can the pet urinate?
   This is most important in male cats, whose presenting complaint can widely vary when experiencing a urethral obstruction. Since this condition can trigger life-threatening sequelae, including cardiac arrest, it should always be confirmed or denied via either triage imaging or physical exam.

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4. Does the pet have concerning comorbidities?
Most owners will share if their pet has previously diagnosed chronic diseases but differentiating those that could be life-threatening (i.e. congestive heart failure [CHF]) and those that are less concerning (i.e. Lyme disease) can be challenging. While many patients presenting complaints are typically related to their chronic disease or medication, as we'll discuss later, you cannot always assume the two are related.

5. Does the patient have a history of seizures? If so, how many?
Many first-time seizure patients (as long as they have only had one) do not require emergent intervention. However, if a patient has had cluster seizures (>2 seizures in 24 hours) or if the seizure is believed to be the result of a toxic ingestion, they should be evaluated emergently. Furthermore, patients having seizures in the waiting room or with the owner (even though non-life-threatening) tend to increase the overall anxiety level and may make discussing diagnostics and treatments more challenging. Further information about seizure management can be found in Chapter 16.

6. Has the pet ingested anything foreign or toxic, and if so, when?
Quickly assessing these pets may allow you to induce emesis that can either allow for rapid discharge in the case of an ingested foreign body or reduce the absorbed dose of a toxin. See also Chapter 20 for further information on the management of the toxic ingestion.

7. Is there any history of recent or acute abdominal distention? These cases should be immediately evaluated using the triage exam to rule out cavitary effusions or gastric dilatation/volvulus (GDV).

THE TRIAGE PHYSICAL EXAM

Your evaluation on triage is NOT a complete physical exam. The presence of abnormalities, such as flank alopecia and dental disease, is less critical than determining cardiovascular stability.

The goals of the triage physical exam are to:

1. Identify signs of shock with the intent of guiding diagnostics to understand the underlying cause for shock.
2. Identify a suspected cause for respiratory distress (if any) to help with early intervention.
3. Identify other underlying disease processes (i.e. urinary obstruction) that would require further diagnostics and immediate intervention.

The first 30 seconds of the triage exam are geared at answering the following questions:

1. Is my patient alive?
2. Does my patient have a patent airway and functioning respiratory system?
3. Is my patient in shock?

QUESTION 1

Is my patient alive?

While many patients (especially cats) can present apparently moribund, there are several disease processes, such as urinary obstruction and decompensated shock, that can be rapidly reversed with appropriate treatment. Your initial assessment should involve visualization of any spontaneous

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respiratory efforts and either pulse palpation, cardiac auscultation, or palpation of the apex beat of the heart, which is especially helpful in cats. If there is any doubt as to these findings within 30 seconds, cardiopulmonary resuscitation (CPR) should be immediately initiated. Further information on CPR can be found in Chapter 24.

QUESTION 2

Is the airway intact and are the lungs functional?

We'll delve deeper into assessment of respiratory distress in a later chapter, but there are a couple of key points to make here. Broadly, respiratory distress can be divided into an upper and lower component. While upper respiratory/airway disease can be more rapidly fatal (i.e. brachycephalic obstructive airway syndrome [BOAS] crisis), lower airway disease in the long term carries a poorer prognosis. Either way, correct identification and treatment for *either* localization is critical for effective stabilization. Therefore, consider the following questions:

1. Do I hear audible (without a stethoscope) respiratory noise? This is more commonly associated with upper respiratory diseases such as laryngeal paralysis, BOAS, or a nasopharyngeal polyp (among others).
2. Is the patient using accessory muscles to breathe, orthopneic, agonal, or have cyanotic mucous membranes? Regardless of etiology, these signs are representative of imminent respiratory arrest, and endotracheal intubation should be initiated.
3. Is the signalment and history consistent with CHF, asthma, pleural effusion, pneumonia, laryngeal paralysis, tracheal collapse, or BOAS? These seven differential diagnoses represent the most common emergency presentations for true (nontraumatic) respiratory distress. There are usually both signalment and physical exam clues that can help rule in or rule out certain diseases on this list, though we'll discuss these further in Chapter 15.
4. Have I done point of care ultrasound (POCUS)? This is ALWAYS an indicated test for pets in respiratory distress, as it can begin differentiating between cardiac, pulmonary, and noncardiopulmonary causes for dyspnea.

   Further details on the management of the dyspneic patient can be found in Chapter 15.

QUESTION 3

Is my patient in shock?

Appropriate identification of shock and its underlying etiology is crucial for the rapid stabilization of any sick patient. Physical exam findings of shock can include pale mucous membranes, abnormal heart rate (high or low), dull mentation, reluctance to walk, and poor pulse quality. In cats, a low temperature is highly correlated with both sepsis and mortality (Pontiero et al. 2025). A high shock index (heart rate divided by blood pressure) is a more sensitive indicator of shock in both dogs and cats (Porter et al. 2013, Fadel et al. 2025) but requires measurement of a systolic blood pressure, which may not be immediately available. POCUS is useful for identifying cavitary effusions that are frequently a source of shock, and in skilled hands, it can also be helpful in evaluating for a cardiogenic source of shock. Furthermore, even in many seemingly stable older patients, it is not uncommon to find significant abnormalities on POCUS.

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MISCELLANEOUS PRESENTING COMPLAINTS

1. Is this a urethral obstruction? Every male cat presenting with vague or poorly differentiated signs should have its bladder palpated to rule out a urethral obstruction. See also Chapter 13.
2. Is this pet mentally appropriate? While dull mentation can be a sign of shock/systemic illness, in cases of seizures or central nervous system disease, being prepared for additional seizures through placement of an IV catheter is helpful for treating any additional events. See also Chapter 16.
3. Is there any history of acute abdominal distention? Evaluating these pets via POCUS is helpful in rapidly ruling in/out peritoneal effusion as well as stratifying the risk of GDV.
4. Has the pet recently ingested a toxin or foreign body that may benefit from induction of emesis, and is it safe to do so? See also Chapter 20.

SECONDARY SURVEY

The secondary survey should occur after any immediately life-threatening conditions have been identified and addressed. This exam involves taking formal vital parameters, assessment for musculoskeletal disease, a full neurologic exam, pain levels, degree of dehydration, etc.

USE OF TRIAGE SCORING TOOLS

Currently there is only one triage scoring system in veterinary medicine that uses a body systems approach toward identifying and stratifying illness severity (Ruys et al. 2012). Interestingly, this paper showed poor agreement (<50%) between systematic and intuitive triage, though its methodology provides a helpful framework in patient assessment as shown in Figure 1.1.

There are other scores, such as the Animal Trauma Triage score, that use perfusion, cardiac, respiratory, neurologic, and musculoskeletal categories to evaluate disease severity (Rockar et al. 1994) and the VetCOT score (Chik et al. 2021) that also includes ionized calcium and lactate.

CONCLUSIONS AND FINAL CHECKLIST

The goal of the triage exam is to identify life-threatening abnormalities to immediately institute stabilizing measures. Using history, signalment, your physical exam, and POCUS, you should run through the following checklist in your mind:

1. Is this patient alive?
2. Based on presenting complaint and signalment (especially age), could they have a life-threatening disease?
3. Are they in respiratory distress and does it localize to the upper respiratory tract, lower respiratory system, or pleural space?
4. Are they in shock and why?

While we'll talk about the appropriate stabilizing treatment based on specific disease processes in later chapters, some of the diagnostic and therapeutic tools you have quickly available for the crashing patient are shown in Table 1.1.

As a final figure, I've included my own mental approach to the triage exam as a flowchart in Figure 1.2.

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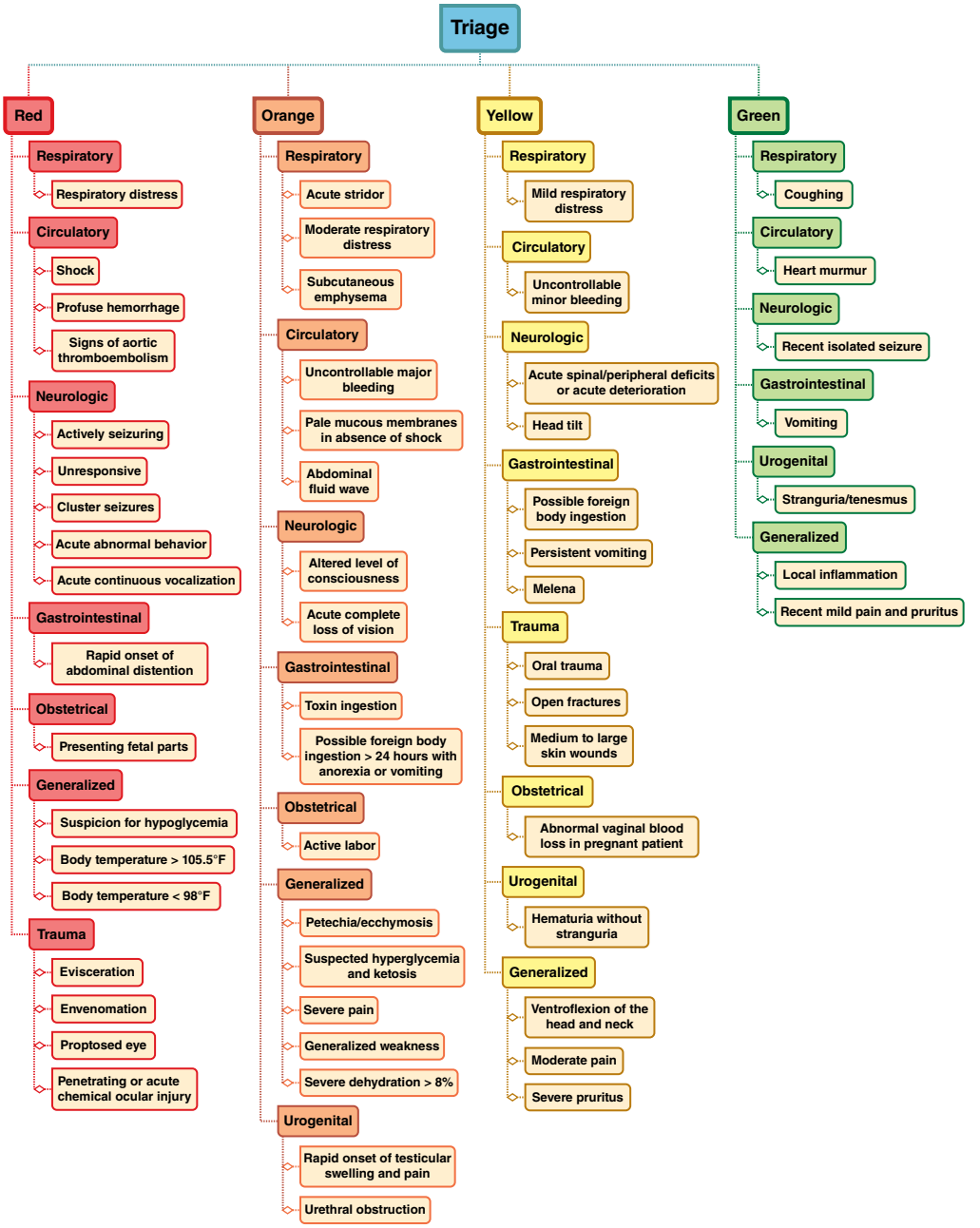


FIGURE 1.1 A systematic approach to assessing disease severity at triage. *Source*: Thomvosky and Ilie (2024)/Canadian Veterinary Medical Association. Used with permission.

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Table 1.1 Initial diagnostic and therapeutic options for the emergent small animal patient. Table from the author.

Diagnostic tools	Treatment options
Point of care ultrasound	Intravenous catheter/fluid bolus (see Chapter 4)
Point of care electrolytes/chemistry	Sedation and/or analgesia (see Chapter 5)
Blood pressure	Thoracocentesis
Electrocardiogram (see Chapter 22)	Diuretics (see Chapter 22)
Packed cell volume/total solids	Intubation (see Chapter 15)
Blood glucose/lactate	Oxygen therapy (see Chapter 15)
Pulse oximetry (see Chapter 15)	—

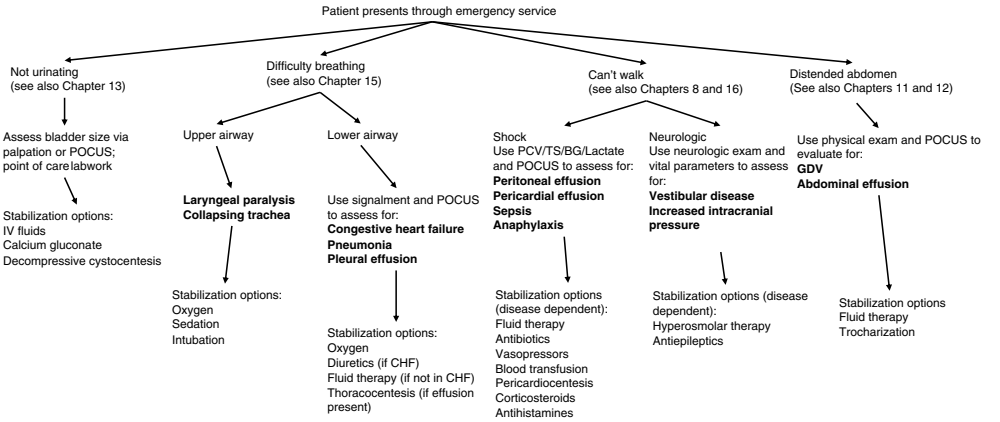


FIGURE 1.2 The author's initial triage flowchart including concerning presenting complaint, diagnostic options, can't miss diagnoses (bolded), and stabilization options. Figure from the author.

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Rational Lab Work Selection and "Diagnostic Stewardship"

CHAPTER 2

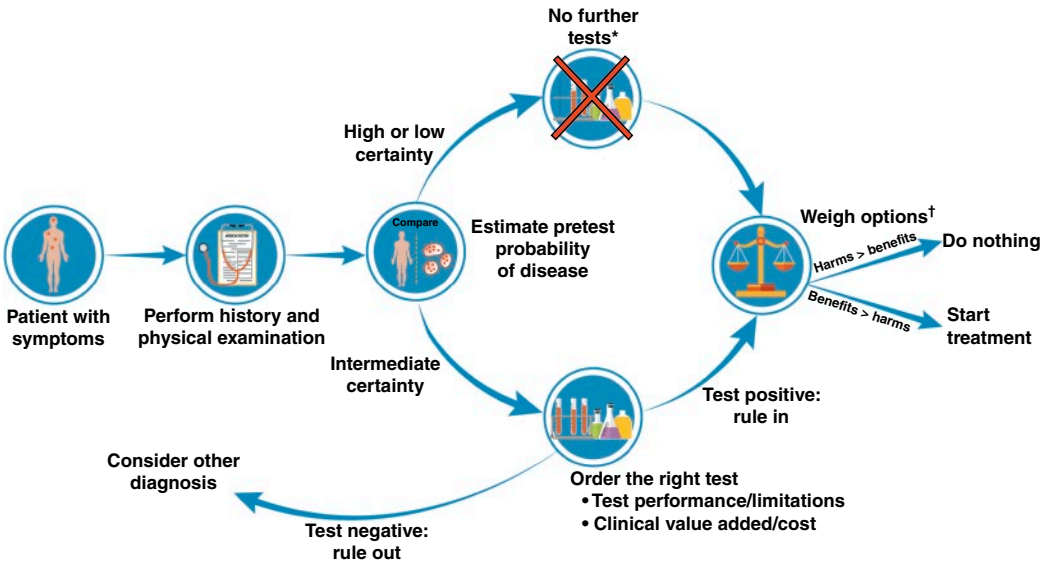


FIGURE 2.1 Decision pathway for diagnostic testing emphasizing the utility of rule in and rule out testing. *Source*: Morjaria and Chapin (2020)/with permission of Elsevier.

CRITICAL QUESTIONS

1. What information am I looking for with these tests?
2. Will the test I am submitting answer the question I am asking?
3. How quickly do I need these answers?

INTRODUCTION

There is a surprising dearth of veterinary research evaluating both the utility of submitting lab work in a sick patient and the risk/benefit of in-house versus send out testing. The majority of published studies focus on healthy patients (Dell'Osa and Jaensch 2016), preoperative populations (Alef et al. 2008, Díaz et al. 2021), or those requiring nonsteroidal anti-inflammatory medications (Chalifoux et al. 2021). Therefore, the majority of this chapter will represent more "expert opinion" (both mine and others) rather than evidence-based medicine.

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Each clinic will have a different selection of in-house and send out laboratory panels available. These can range from a simple blood gas or mini chemistry up to a full complete blood count (CBC), chemistry profile, thyroid level, urinalysis, cortisol panel (and more!). While it can be tempting to run a combination of in-house and send out lab work on every patient just "to be sure," that approach is needlessly wasteful both of clinic resources and owner finances. Furthermore, the use of these screening or rule out tests, as I describe them below, can lead to a phenomenon known as "overdiagnosis," where the overexuberant pursuit of incidental findings causes direct or indirect patient harm (McKenzie 2016).

Therefore, the decision of which lab panels (if any) to run can be boiled down to two of our critical questions: what information do I need, and how quickly do I need it?

If you can satisfactorily answer these two questions, then the solution to which panels to submit (and how to submit them) becomes much more straightforward. However, I think it is also important to point out that sometimes your initial panels will lead to additional testing, which is okay! You're not expected to submit every test you need the first time, and if you are, then that's probably because you're over-submitting tests, which, as discussed above, can also be potentially harmful.

WHAT INFORMATION DO I WANT FROM THESE RESULTS?

Answering this question will not only help guide the tests you submit but also help you inform an owner as to *why* such testing is important, making it much more likely they will allow you to do them (Janke et al. 2021). Owners that understand how a test will help answer an important clinical question for their pet and potentially change treatment are much more likely to agree to it. If you force yourself to answer this question, you are also inadvertently coming up with a differential diagnosis in your mind that can be helpful when considering treatment options and prognosis. In fact, your list of REALISTIC differential diagnoses (based on history, physical exam, etc.) should be driving test selection, not the other way around. The goal of a test is NOT necessarily to provide you the answer, but rather to raise or lower your index of suspicion for a specific disease process. This approach, known as Bayesian reasoning (McKenzie 2021), is an interesting clinical reasoning construct that goes beyond the scope of this book (see Cumming et al. 2022 in additional reading) but also encompasses important factors such as diagnostic test accuracy in its framework. For example, screening a young, normal-acting dog in New England for *Borreliosis* is low yield because you have a low pretest probability of them having that disease (given lack of clinical signs), and so even a positive result would likely not be a reason to treat.

Before picking a test panel to perform, the first step is knowing what results you are going to get with each one. Unfortunately, this can vary substantially between hospital, reference laboratory, and point of care (POC) analyzer. Sadly, many reference laboratories create confusion by making 5+ different chemistry panels, and a recent search of test panels from a large reference laboratory showed about 240 different CBC/chemistry panel combinations! Knowing which panel will give you the results you need, i.e. I don't need a creatine kinase for this patient, can narrow down the options to what will truly be helpful.

I've included a brief summary of what different lab tests might include. For simplicity, I've omitted send out infectious disease testing, such as vector-borne disease and leptospirosis because many of these are region-specific. Similarly, thyroid and other endocrine testing are generally "second line" tests for the emergency clinician, though they are included on many panels.

1. Complete blood count: WBC with differential, RBC, Hgb, HCT, MCV, MCH, MCHC, RDW, platelet count, MPV. Certain panels may or may not have a reticulocyte count, which is crucial for anemic pets. Make sure if you have a pet you suspect of anemia, then you pick

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the right panel. Unfortunately, there are many disadvantages to an in-house CBC, including misclassification of circulating blast cells, platelet clumping (artificial thrombocytopenia), and others. If you strongly suspect there will be something abnormal on your CBC, it is always best to submit to a reference lab for a blood smear review.

2. Chemistry panel: Na, K, Cl, HCO_3, Anion gap, Na : K, BUN, creatinine, Ca, P, Mg, total protein, Alb, Glob, A/G, glucose, ALT, AST, ALP, GGT, bilirubin, Chol, CK, Amylase. As noted above, there can be WIDE differences between what is available on different chemistry panels. While some are obvious, i.e. a renal panel, some are less so, such as the difference between a preoperative panel and an adult wellness panel, so make sure the test you submit is answering the question you're asking.
3. Urinalysis: Color, turbidity, pH, protein, glucose, ketones, bilirubin, blood, specific gravity, sediment exam. While in-house urinalysis, including urine sediment, is becoming more available, as with a blood smear, you should make sure results are valid. Furthermore, I have found many inexperienced clinicians will misinterpret the numerical values for glucosuria and ketonuria, so make sure you know the correlation to the subjective (1+, 2+, etc.) scale.

Once you know the results you will get on each panel, you have to think about the values you expect to be abnormal. The history and physical exam can frequently guide this thought process. For example, a patient with a history of chronic kidney disease and acute vomiting is most likely due to a progression of their kidney disease. Or a patient with recent chemotherapy who is lethargic and febrile may now be neutropenic. On physical exam, a patient with pale mucous membranes could be anemic, shocky, or both, while a visibly icteric patient may benefit from testing geared at differentiating a prehepatic, hepatic, or posthepatic hyperbilirubinemia. Again, asking this question will force you to have a reasonable list of differential diagnoses in your mind that a laboratory test can help raise or lower your index of suspicion for.

This mechanism of thinking around diagnostic testing is what could be referred to as a "rule in" approach and is similar to the Bayesian method discussed above. For a set of differentials, you have a clinical suspicion for a certain set of disease processes based on your physical exam/history, each with a certain pretest probability of being correct. You then use a lab test to verify or confirm (i.e. increased posttest probability) which of those diagnoses is most likely correct to guide treatment. Unfortunately, these "rule in" tests also have a close cousin, the "rule out" tests, which are where you are running tests to make sure your patient *doesn't* have a particular disease process. Again, while it may be tempting to run lab work on everything that comes in through the emergency room, that blanket approach is ultimately inappropriate and will limit your clinical reasoning to a point where, if an owner can't afford lab work, you will have nothing to offer them.

The goal of a "rule out" test is to pick up on disease processes that you may not have initially suspected from your physical exam/history or that can masquerade as other more benign diseases. However, as an astute clinician, you also have to take into consideration the likelihood and risk factors for either of the above scenarios to be true. You can do that by asking yourself two simple questions:

1. How sick is my patient *really?*
2. How old is my patient?

The answer to the first question goes back to our recent discussion around the triage exam. If there are warning signs from that exam (i.e. high heart rate and especially low body temperature in cats) or if the patient, based on your clinical gestalt, appears sicker than you would expect for its presenting complaint/physical exam findings, then those are the patients I will push for rule

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out laboratory testing. For the more stable patient, a delay in diagnosis is generally not prognosis altering in the majority of cases.

The best way I can illustrate this point is through two canine cases with the same presenting complaint of acute vomiting. Both dogs are 1-year-old Labradors who have been previously healthy. The first Labrador gets up to greet me in the exam room and walks around while I'm trying to examine it. The second Labrador is lying down, and I have to lift them up to palpate their abdomen. Both physical exams are identical. Of those two patients, I would clearly be more likely to pursue "rule out" lab work on the second rather than the first, as she seems clinically "sicker." I would also be more likely to find something *if it was there to begin with* in that second dog. While that is not to say that the first Labrador couldn't go on to develop electrolyte derangements from a hemorrhagic gastroenteritis if they were to get sicker or not respond to therapy. However, that delay is unlikely to be catastrophic. My point is that it isn't quite as mission critical to immediately rule in/out a disease process with the first dog *and* you're less likely to find what you're looking for in a "healthier" dog. When speaking to clients, I will use both of these points when discussing the pros and cons of doing this kind of rule out testing. Most clients are very understanding and will be okay even if their pet does go on to require further testing.

To answer our second question, we have to think about the role that age plays as a risk factor when it comes to disease. While age is a number in that you can have a very healthy 13-year-old dog and a very unhealthy 1-year-old, age does confer several immutable factors when it comes to disease. The first is that older pets are much more likely to have systemic organ dysfunction (i.e. liver/kidney disease) than in younger dogs. The second is that they will have much less physiologic reserve to fight off a disease than a younger pet. Something as simple as a urinary tract infection could cause only minor signs in a 1-year-old dog, but could cause fever and lethargy in an 11-year-old. For those reasons, I am much more likely to push for "rule out" testing in an older pet for diagnostic reasons, prognostic purposes, and because it may have treatment implications.

HOW QUICKLY DO I NEED THESE RESULTS?

The decision to run POC versus send out laboratory tests is challenging because there is a tradeoff in both directions. The advantage of POC labs is a faster answer, thus allowing you to make faster diagnosis and treatment decisions (Godfrey 2024). While no veterinary studies exist on this subject, there are several human studies that have shown both a decreased length of stay and decreased time to intervention with POC testing (Rahsepar et al. 2022, Alter 2021, Florkowski et al. 2017). The disadvantages are they tend to be a bit costlier, are less thorough than send out labs, and tend to have worse quality control. The advantages of send out labs are better quality control, more extensive testing available, and cost (comparable panels are generally cheaper). The disadvantage is that you may have to wait for results. Therefore, the first question to ask is: when will I get these results? Some larger specialty and university hospitals will have in-house reference laboratories that can turn around results within 3–4 hours. However, if your hospital has to wait for results overnight or over a weekend, your decision will likely be very different.

The second question to ask when considering your required timeline for results is to ask yourself how your treatment plan will change depending on those results. For example, a cat with a urethral obstruction cannot wait any period of time for you to know whether its potassium is high because you need to know to give calcium gluconate. Conversely, the stable older cat with one episode of vomiting can probably wait several hours (or overnight) for results. The bottom line is that if test results are NOT going to change your treatment approach, i.e. hospital admission, fluid therapy, procedure, then you can likely wait for send out testing. Furthermore, if a lab result isn't going to change your treatment approach at all, then strongly consider whether or not to submit it at all.

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CONCLUSIONS

The approach to rational diagnostic testing selection has several components. The first is whether to do any tests at all, which you should base on your history, physical exam, risk factors, and differential diagnosis list. Again, the goal of this testing should be to increase or decrease your index of suspicion for a particular disease. The second is which tests to do, which you should guide based on your rule in differential diagnosis list. The third is whether to do in-house or send out testing, which you should decide based on illness severity and the implications those results would have for your treatment plan. These concepts are what are referred to in human medicine as "diagnostic stewardship" and encompass the use of the right test, for the right patient, at the right time to improve patient outcomes (Fatemi and Bergl 2022) and is conceptually illustrated in Figure 2.1. And while these decisions can be challenging to the novice practitioner, if you go through and answer the questions above, you will be able to acquire your own clinical gestalt about lab testing and will be less likely to err on something you truly can't miss. You may even find yourself "getting lucky" and picking up on things that others may have missed!

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Selecting Appropriate Imaging Studies

CHAPTER 3

CRITICAL QUESTIONS

1. How likely is it that imaging will give me useful information?
2. How will the results of an imaging study CHANGE my management of this patient?
3. When should I reach for computed tomography (CT) in the emergency setting?
4. What is the downside of "over imaging?"

INTRODUCTION

We'll encounter similar "diagnostic stewardship" issues with imaging as we did in Chapter 2 on lab testing. While we'll cover more details on the pros and cons of certain imaging modalities for specific disease processes in subsequent chapters, the goal here is to establish general principles in deciding whether or not to do imaging in the first place.

As an emergency clinician, we generally have easy access to two imaging modalities: radiographs and ultrasound, though we'll briefly discuss computed tomography (CT) as well. There are advantages to each modality in terms of accessibility, patient stress, and reliability of results.

WILL THIS IMAGING STUDY BE HELPFUL?

Just as with laboratory testing, the goal of an imaging study is to provide a diagnosis that informs a treatment decision. To achieve the first half of that goal, the clinician must understand how likely an imaging study is to provide them both useful AND accurate information.

Oftentimes, there are clues in the history to justify imaging in the presenting complaint, such as coughing, difficulty breathing, or vomiting. However, it is important to put these presenting complaints in context of both signalment and duration of clinical signs when considering whether to pursue imaging. For example, the differential diagnoses for a febrile 1-year-old dog coughing for 24 hours are very different than the 11-year-old dog coughing for 3 months. In addition, considering past medical history can also be helpful in guiding decision-making around imaging. A cat with a history of foreign body surgery who presents for multiple episodes of vomiting is more likely to warrant imaging than the older cat who vomited one time. Presenting complaints that include difficulty breathing or coughing with or without histories of cardiopulmonary disease may be candidates for thoracic imaging. Similarly, pets with presenting complaints of vomiting/diarrhea or inappetence with or without a history of gastrointestinal disease would be candidates for abdominal imaging. Patients with clinical signs referenceable to neither body cavity (especially the chest) are unlikely to have rewarding imaging studies (Moss et al. 2025).

The physical exam usually doesn't provide a specific diagnosis but can be helpful in providing a sense of urgency as to the necessity of imaging. For example, a vomiting pet with abdominal pain almost certainly warrants emergent imaging to rule out foreign body obstruction or peritonitis. Conversely, a young pet who is coughing but still bright, eating, and breathing normally likely does not require imaging.

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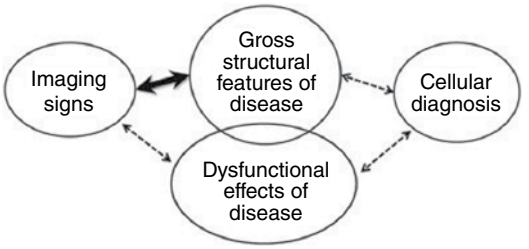


FIGURE 3.1 A helpful imaging study requires the disease to have a gross anatomical change. It cannot directly evaluate for "functional" diseases or provide a cytologic diagnosis. *Source*: Lamb (2016) with permission of Elsevier.

The integrated use of history and physical exam to guide the decision to perform an imaging study is the concept involving "pretest probability," or how likely it is a pet has a specific condition before a test is done, as discussed in Chapter 2. Taken to the extreme, if there was a situation where the pretest probability for a condition was 100%, doing an imaging test would be completely superfluous because the diagnosis has already been made! The higher the pretest probability, the more likely it is that a positive imaging test will provide it as an accurate diagnosis, which would be labeled as "posttest probability" (Lamb and Nelson 2015). For example, in a young vomiting dog with abdominal pain, you likely already have a high pretest suspicion for a gastrointestinal foreign body. When the ultrasound shows a foreign object with orad intestinal distention, the posttest probability is nearly 100%. Providing an appropriate history (or direct consultation with a radiologist) is so crucial in obtaining an accurate diagnosis, as that collaboration allows both parties to mentally rank their own pretest and posttest probabilities (Arruda Bergamaschi et al. 2023). Judging the true pretest probability is challenging for the novice clinicians, but with practice and repetition, it does become easier to incorporate all historical and physical exam factors to determine what disease process truly is "most likely."

Unfortunately, there is a second important concept to consider the limitations of an imaging study as shown in Figure 3.1. One has to consider how likely it is that the study will be able to diagnose the condition you're looking for. More specifically, how likely is it that the disease you are looking for will have visible structural changes on imaging, as it cannot evaluate function or microscopic disease.

Many diseases (especially in the respiratory tract) do not show significant or pathognomonic changes on imaging studies that correlate to a patient's clinical signs (Osathanon et al. 2022, Lamb 2016). Always consider that if the disease you have highest on your differential (highest pretest probability) is NOT one with significant visible changes, perhaps an imaging study will be unrewarding and plan accordingly.

Combining both of these concepts, the clinician must then assess the accuracy of the imaging study being performed. Unfortunately, multiple studies have shown that most imaging tests only have a moderate sensitivity/specificity and positive/negative predictive value for detecting changes, especially when measurements are involved (Lamb and Nelson 2015). Furthermore, in many cases, there can be a lack of correlation between imaging findings and those found at necropsy (Cohen et al. 2023). Therefore, when choosing to do an imaging study, consider how accurate the test is at giving you a true result and place it in an appropriate clinical context.

HOW WOULD IMAGING CHANGE MY MANAGEMENT?

This is probably one of the hardest questions for a clinician to answer because it requires them to honestly rank a differential diagnosis list and incorporate the owners' goals of care. But first, we have to understand how imaging can change a treatment protocol. As we'll see later, our list of

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treatment options for any disease is actually shorter than we would expect in terms of both medical and surgical management.

For abdominal imaging on an emergent basis, the biggest change an ultrasound or radiographs would make in our treatment approach is whether or not surgery is required. While we'll talk later about risk factors for surgical disease in the abdomen, if a foreign body, gastric dilatation and volvulus (GDV), hemoabdomen, or septic abdomen is a rule in (not a rule out) diagnosis, then abdominal imaging is absolutely warranted. This is not to say that abdominal imaging does not have a role in diagnosing nonsurgical disease, but more to consider what nonsurgical disease you are evaluating for and how your treatment approach would differ as a result.

Imaging of the chest can sometimes be more nuanced as you have to balance the risk of a patient destabilizing due to the stress of radiography versus the benefit of the information a radiograph will provide. In general, chest radiographs are most helpful in evaluating whether antibiotics, steroids, or diuretics are the most appropriate pathways forward. Thankfully, as we'll see in Chapter 15 when we discuss diagnosis/management of the dyspneic patient, point of care ultrasound will prove an incredibly helpful, lower risk diagnostic tool in answering some of those questions.

PELVIC AND EXTREMITY FILMS

The utility of pelvic and extremity films for diagnosing disease in the emergency room is a perfect conclusion to this section because it beautifully incorporates both of our core questions. The goal of these studies is to diagnose fractures because those are definitely going to be managed differently than a soft tissue injury in terms of either internal or external coaptation. However, we also have to take into account the history, signalment, and physical exam to determine the likelihood of a fracture being present. For a further discussion on imaging in the trauma patient, you can refer to Chapter 10.

In the history, we have to ask ourselves is there an injury bad enough to cause a fracture, such as a motor vehicle accident or gunshot wound. In older pets, it is always important to ask whether or not a pathologic fracture could be present. Finally, but in all honesty, most importantly, is there an area of pain I can localize on my physical exam. If you can't palpate an obvious fracture or area of discrete pain on physical exam, the chances of finding a clinically significant injury (i.e. fracture) are likely low. The exception to this is in pediatric patients where imaging should almost always be employed to evaluate for growth plate fractures. Some risk factors that should increase your inclination toward extremity radiographs are summarized in Table 3.1.

Table 3.1 Clinical scenarios where extremity radiographs are most likely to be helpful.

High-risk indications for extremity radiographs
Significant blunt force trauma (i.e. HBC)
Palpable bone pain/crepitus or swelling
Non-weight-bearing lameness in pediatric patient
+/– joint swelling
Fever + bone pain in pediatric patient

WHAT IS THE DOWNSIDE OF IMAGING?

Besides the obvious downsides of a test not providing helpful or accurate information, there are other risks with imaging as well. Many pets will need to be sedated for imaging, and inappropriately weighing the risk/benefit of sedation versus the information to be gained has the potential to lead to catastrophe. Second, running large numbers of tests can drain owner finances, leaving you with a diagnosis (hopefully) but no ability to treat it. Finally, the more tests a clinician does, the higher the likelihood of encountering incidental findings. Imaging is not immune to these with a rough incidence rate of 8–11% of CT cases having at least one "incidentaloma" (Caspanello et al. 2023). The problem with these findings is twofold. First, while they should be generally communicated to an owner, that runs the risk of diverting focus from the primary

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problem. And second, they can sometimes distract the clinician from addressing the primary disease process and can trigger unnecessary/potentially harmful interventions (Caspanello et al. 2023). For example, the finding of liver and splenic nodules in an older patient is frequently non-pathologic (or not related to the primary disease state) but can occasionally lead to unnecessary and expensive tissue sampling.

WHAT IS THE ROLE OF CT IN THE EMERGENCY ROOM?

I chose to place CT here, because I think it illustrates several of the concepts we've talked about above. As an imaging modality, CT can be more sensitive and specific for detecting disease, but it is not without its drawbacks. Beyond the logistical factors that CT is more costly, less available, and requires more resources (sedation or general anesthesia); since it is more "detailed" than other modalities, it is also more prone to picking up incidental findings.

For the truly "emergent" patient (i.e. one you are responsible for managing until an inpatient doctor takes over), a CT scan is almost never critical except in cases where it would lead to a surgical intervention. The vast majority of these cases involve vertebral or skull fractures, where emergent stabilization/fixation translates to improved outcomes. Almost every other "emergent" disease can be managed or temporized without the use of advanced imaging.

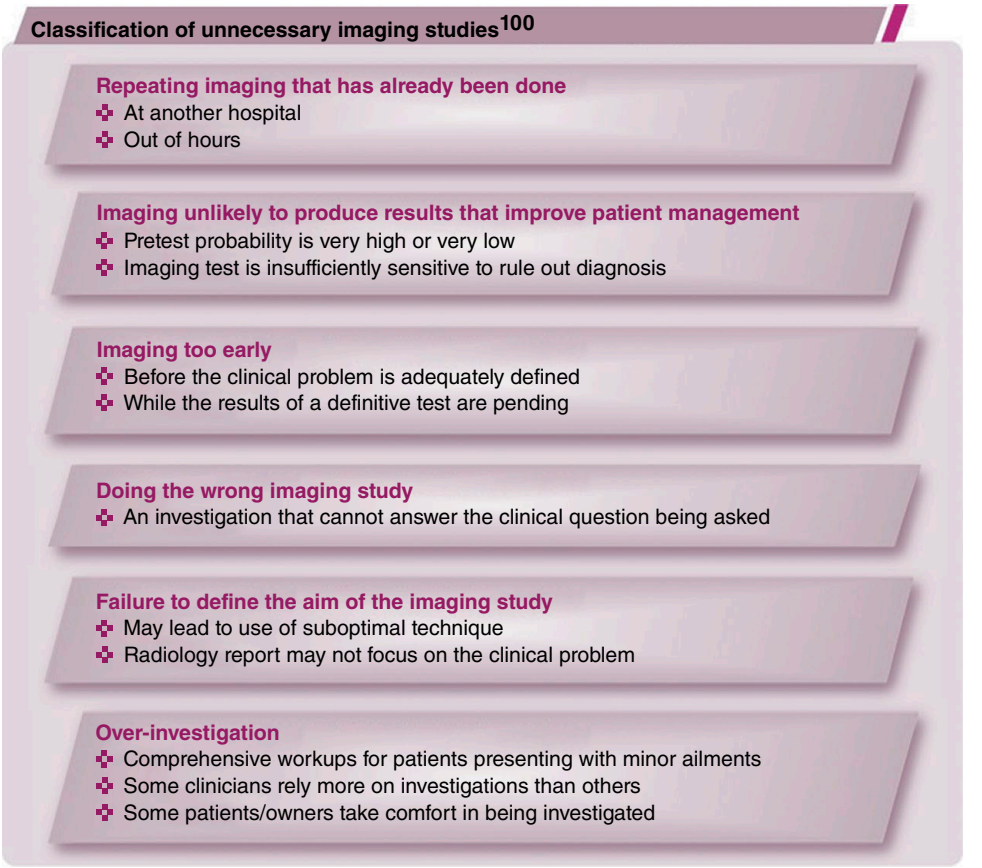


FIGURE 3.2 Factors to consider when determining the clinical utility of a diagnostic imaging test. *Source*: Lamb and David (2012), International Cat Care Veterinary Society and Feline Veterinary Medical Association.

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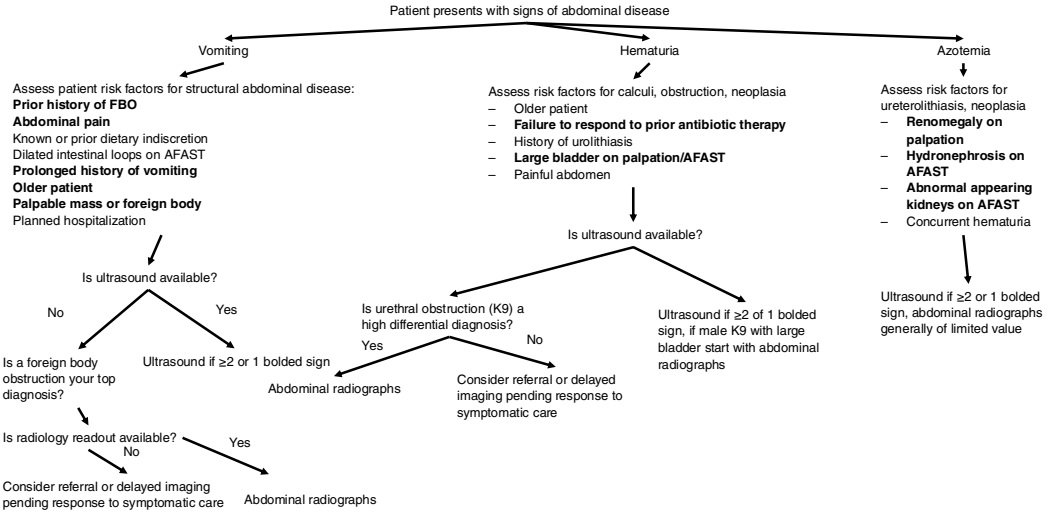


FIGURE 3.3 A flowchart to guide decision-making around abdominal imaging based on patient risk factors. See also Chapter 14. Figure courtesy of the author.

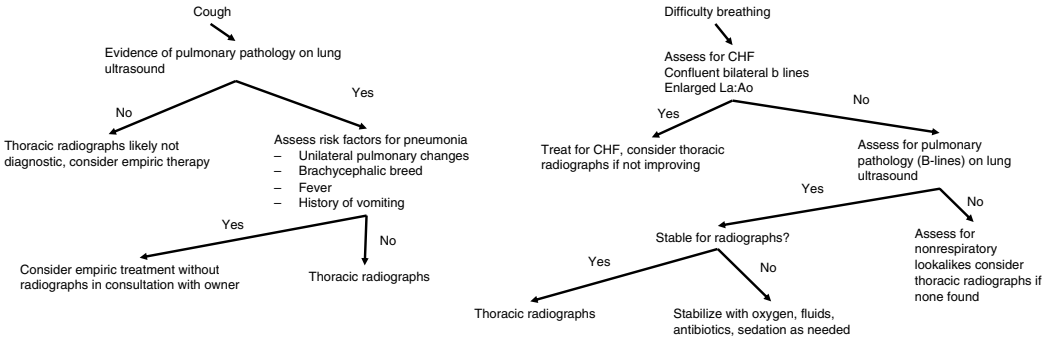


FIGURE 3.4 A diagnostic flowchart for thoracic radiographs using point of care ultrasound to help limit/avoid nondiagnostic or potentially hazardous studies. See also Chapter 15. Figure courtesy of the author.

CONCLUSIONS

It may seem that this whole chapter is all about dissuading you from ever doing any imaging studies; it is not! The goal of this chapter is to help you choose imaging studies that are likely to yield an *accurate/helpful* result, and improve patient outcome through better treatment selection. Considering *and* avoiding the causes of unnecessary and inappropriate imaging, as shown in Figure 3.2, will help you select appropriate and useful imaging studies. We will apply these general concepts in more detail to specific disease processes in later chapters.

As a final note on this chapter, I've included my own mental flowcharts in Figures 3.3 and 3.4 when it comes to considerations surrounding abdominal and thoracic imaging. Cost consideration is important as well, so for owners with financial concerns, I try to consider how different my treatment approach would be between two differential diagnoses (i.e. both would get antibiotics) and whether or not I can treat multiple diseases simultaneously.

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Intravenous Fluid Therapy: Yes, No, How Much, How Fast

CHAPTER 4

CRITICAL QUESTIONS

1. Is my patient dehydrated or hypovolemic based on history, physical exam, and laboratory parameters?
2. How much fluid does my patient need, and how fast do I need to give it?
3. Does it matter which fluid I give?
4. Does my patient have heart disease significant enough to risk developing congestive heart failure with fluid therapy?
5. Is my patient high risk for fluid overload?
6. Are electrolyte abnormalities present, and why are they there?

INTRODUCTION

Fluid therapy has the potential to be a lifesaving treatment, but it is also a drug with inherent risks and adverse effects. Therefore, when considering giving fluids, you should consider whether or not your patient will benefit from that therapy and what the potential downside is in using them.

There are three main goals of intravenous fluid therapy: to correct intravascular volume deficits (hypovolemia), improve extracellular/interstitial volume deficits (dehydration), and rectify electrolyte abnormalities. If your patient isn't hypovolemic, dehydrated, or has electrolyte abnormalities based on your history, physical exam, and point of care lab work, then they may not benefit from this treatment.

ASSESSING FLUID REQUIREMENT: DEHYDRATION

Dehydration represents a depletion of fluid from the extracellular space. In taking a patient history, ask whether or not the patient has had any reason to become dehydrated: are they presenting for vomiting/diarrhea? Have they been drinking and urinating excessively (and now stopped drinking)? This history is then combined with the physical exam, evaluating for signs of dehydration including tacky mucous membranes, ocular hydration (sunken globe, dehydrated cornea), and increased skin turgor as summarized in Table 4.1 to generate a rough idea as to the degree of dehydration (Goggs et al. 2008, Goucher et al. 2019).

However, beyond history and physical exam, there are several additional objective markers we can sometimes use to assess hydration status, including increased urine specific gravity (which can be obtained point of care), increased packed cell volume, and increased total protein (Boyd 1981, Atata et al. 2019).

The only potential caveat to the above assessment is that the only study assessing the correlation between physical exam, laboratory findings, and hydration status (as assessed by weight gain post fluid therapy) found no correlation between any parameters! (Hansen

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Table 4.1 The physical examination findings seen with different degrees of dehydration.

Level of dehydration (%)	Clinical signs
<5	Not detectable
5–6	Subtle loss of skin elasticity
6–10	Definite delay in return of skin to normal position
	Eyes possibly sunken in orbits
	Possibly dry mucous membranes
10–12	Tented skin stands in place
	Eyes sunken in orbits
	Dry mucous membranes
12–15	As for 10–12%, plus possible signs of shock (e.g. tachycardia, cool extremities, rapid and weak pulses, prolonged capillary refill time)

Source: Goggs et al. (2008) with permission of John Wiley & Sons.

and DeFrancesco 2002). However, if you critically examine that paper and read the authors' discussion, there are a fair number of confounding factors that may explain their results. You should not rely on any one factor or symptom but rather take the patient as a whole when determining the need for fluid therapy.

ASSESSING FLUID REQUIREMENT: HYPOVOLEMIA

Hypovolemia refers to a fluid deficit within the intravascular space. Unlike with dehydration, history is of less benefit for detecting hypovolemia because the source of intravascular fluid loss (i.e. hemorrhage) is not always externally apparent.

Physical exam findings consistent with hypovolemic shock are those we discussed earlier in the triage exam chapter. These will include alterations in heart rate (usually high in dogs, can be low in cats), mucous membrane color (usually pale), capillary refill time, and pulse quality (Johnson 2016).

In addition to the history and physical exam, quantitative markers of hypovolemia include low mean arterial blood pressure, high blood lactate, and a high shock index value. Additional (though slightly more advanced) parameters would include point of care ultrasound evaluation of the heart for increased cardiac contractility and decreased ventricular/atrial size (Campbell and Kittleson 2007, Fine et al. 2010) as is shown in Figure 4.1.

DEHYDRATION: HOW MUCH FLUID TO GIVE AND HOW FAST?

The short answer to this question is until you've corrected the dehydration, shock, or electrolyte abnormalities! However, in reality, the amount of fluid to give is dependent on the degree of dehydration or shock AND the underlying disease process.

For the dehydrated patient, it's important to assess the degree of dehydration (Table 4.2) – the higher the degree of dehydration present, the more fluids the pet will need. For some rough numbers, I will roughly use 1.25x maintenance fluid rate for patients with <5% dehydration, 1.5x maintenance for 5% dehydration, 2–2.5x maintenance for 5–7% dehydration, and 3x maintenance for 10% dehydration. There are multiple ways of calculating a maintenance fluid rate (Hopper et al. 2018), though I prefer to use a simple 60 ml/kg/d as my baseline rate. Ultimately, what is more important than your initial fluid rate is ensuring you modify it in response to the patient's condition.

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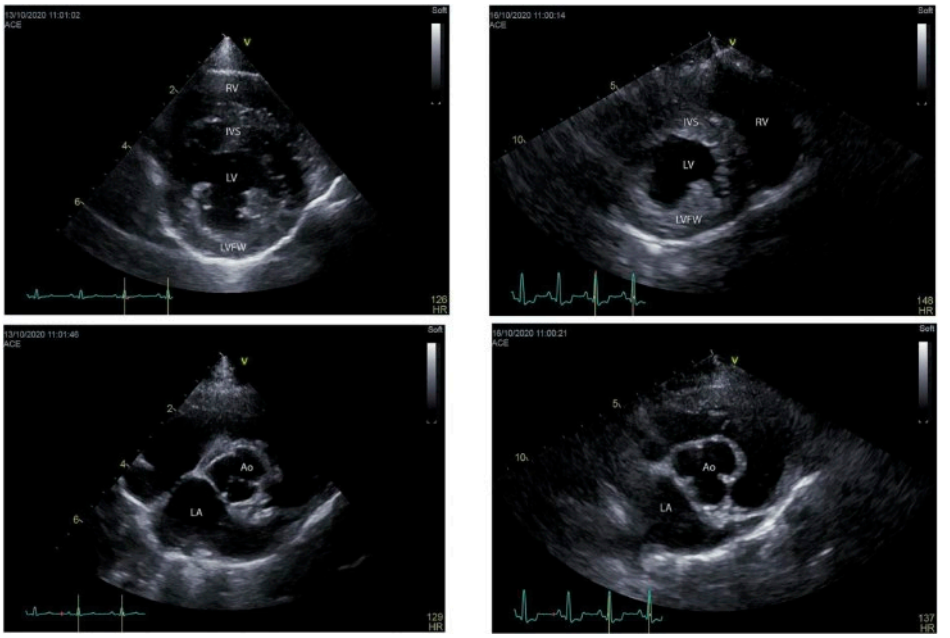


FIGURE 4.1 Echocardiographic images of a (a) normal and (b) hypovolemic heart. *Source*: Boysen et al. (2021)/Frontiers Media S.A./CC BY 4.0.

Table 4.2 Calculated fluid rates for a 25-kg dog using the "maintenance multiplier" and fluid deficit approach corrected over 24 (1) and 12 (2) hours.

Degree of dehydration (%)	Maintenance multiplier approach (ml/hr)	Fluid deficit approach[1] (ml/hr)	Fluid deficit approach[2] (ml/hr)
3	78	31	63
5	94	52	104
7	125	73	146
10	188	104	208

You can also calculate out the patient's fluid deficit using the equation:

Fluid deficit (liters) = body weight (kilograms) × percent dehydration (as a decimal). This deficit would then be corrected over 12–24 hours. Using this approach will result in different fluid rates depending on the time period for correction fluid rates than the approach I've outlined above as summarized in Table 4.2. However, both approaches neglect the fact that, as dehydration is corrected, (and you treat the underlying condition), a patient will likely start voluntarily taking food and water. There have been no controlled studies evaluating the superiority of one approach over the other.

If a patient is going to need surgery for something like a foreign body, then a faster fluid resuscitation plan will likely make anesthesia safer. For these patients, I will consider giving a portion of their dehydration deficit over 1 hour prior to anesthesia.

Secondly, it's also important to understand whether or not the pet has ongoing losses that may increase that "normal" or basal maintenance fluid requirement. For example, pets with poorly controlled diabetes mellitus or renal insufficiency will have a higher baseline maintenance fluid requirement than a healthy vomiting pet. The final consideration for the dehydrated pet is whether

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or not you expect ongoing losses, such as a post obstructive diuresis with a blocked cat or persistent vomiting/diarrhea.

More important than what your selection of an initial fluid rate is how you monitor your patient after starting them. Periodically evaluating the hydration parameters discussed above is vital in ensuring that your plan is achieving your desired goals.

HYPOVOLEMIA: HOW MUCH AND HOW FAST

The approach to the hypovolemic or shocky patient is different as their resuscitation is more time critical since the longer a patient is in shock, the higher the risk for organ dysfunction and mortality. Current recommendations for fluid administration are ¼ shock dose (15–20 ml/kg) as a bolus given over approximately 15 minutes. However, more important is what you do AFTER the bolus is complete. Evaluating your patient for improvement in perfusion parameters, such as heart rate, lactate, blood pressure, mucous membrane color/capillary refill time, and mentation, is critical in determining whether you've given "enough" fluid or need to consider reaching for another bolus, vasopressors, or blood products. The decision as to your second line therapy will depend on the underlying etiology for shock, as a hemorrhaging patient would benefit from a transfusion whereas the septic patient may need a vasopressor.

DOES FLUID SELECTION MATTER?

The honest answer is that in the vast majority of cases, your selection of replacement fluid (i.e. Lactated Ringers, Plasmalyte, Normosol, 0.9% saline) likely has little influence on any patient-centered outcome in the majority of routine cases (Klein-Richers et al. 2022, Banzhaf et al. 2025). The exceptions to this rule would be patients with severely altered sodium levels, cardiac disease, and liver failure (Rudloff and Hopper 2021). My own personal practice is to use a balanced isotonic crystalloid (usually Lactated Ringers) for all aspects of maintenance/replacement fluid therapy. This avoids certain hypothetical concerns about non-physiologic sodium, chloride, and potassium levels that may occur with other fluid types, such as 0.9% saline. Since I typically do not reach for synthetic colloids (and there are a large number of good references discussing them), they aren't going to be covered here. We'll discuss the use of hypertonic solutions (hypertonic saline) in later chapters on trauma and hemoabdomen management.

FLUIDS WITH A HEART MURMUR

Giving fluids to a patient with a heart murmur is one of the most challenging and scary decisions for the junior clinician. In reality, the concern and question are whether or not the patient has severe enough heart disease that giving fluids would push them into congestive heart failure. Thankfully, there are several clues we can obtain from our history, physical exam, and point of care imaging that can help us answer this important question.

If you auscultate a heart murmur in a patient, make sure to ask if that murmur has ever been noticed before and whether or not it has been worked up. The soft murmur a pet has had their entire life is less likely to represent end-stage heart disease. Similarly, if a dog had an echocardiogram that diagnosed mild disease *and the murmur hasn't progressed*, in all likelihood that dog doesn't have clinically significant heart disease. Furthermore, in cats with heart murmurs, over 50% will not have any structural heart disease! (Ferasin et al. 2022) Unfortunately, the converse is also true, as most clinicians have experienced that the absence of a heart murmur does not rule out structural cardiac disease in cats, as 15–22% of cats with hypertrophic cardiomyopathy lack a murmur (Rush et al. 2002, Payne et al. 2010). Finally, ask if the pet is on any current cardiac

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medications. If they are already on heart medications, especially ones for heart failure that immediately raises a red flag that that pet has enough disease that you should be judicious with fluid administration.

For dogs with degenerative mitral valve disease, murmur intensity will roughly correlate with disease severity. If a dog with a soft murmur who you suspect based on signalment (or know from your history) has degenerative mitral valve disease is unlikely to go into heart failure with reasonable fluid therapy.

The use of cardiac biomarkers (specifically NT-proBNP) is tempting to evaluate the risk of cardiac disease but comes with several drawbacks. Firstly, while there is an assay currently in development for dogs (Harr et al. 2022), the only point of care test commercially available is for use in cats. Secondly, while that point of care test has been shown helpful in predicting the presence and absence of cardiac disease in cats WITH heart murmurs, it performs worse in cats without abnormalities on auscultation (Lu et al. 2021, Machen et al. 2014, Harris et al. 2017)

Finally, point of care ultrasound can be very helpful in identifying pets (especially cats) who have more significant cardiac disease than their history or physical exam would suggest. A normal left atrial size in the presence of a heart murmur indicates a low risk for developing left-sided congestive heart failure from standard fluid therapy. Similarly, if you chose to do a chest X-ray, a normal heart size usually indicates a low risk for end stage disease.

Ultimately, the decision to give fluids to a pet with a heart murmur comes down to a risk/benefit analysis. As discussed above, factors to consider when evaluating that risk include signalment, history and prior workup of the murmur, medication history, presumptive diagnosis, laboratory findings, and imaging (Côté et al. 2015). If this analysis indicates a perceived low risk of advanced heart disease, then the patient likely will tolerate a reasonable amount of isotonic crystalloid. If the analysis favors a benefit of fluid therapy based on the underlying (noncardiac) disease process, but with a perceived HIGH risk of advanced cardiac disease, one can consider a low rate (50–100% of normal maintenance) of 0.45% NaCl as the lower sodium content favors faster distribution outside the vascular space reducing the risk of congestion (Pouchelon et al. 2015). And finally, regardless of the approach taken, it is always important to monitor for signs of congestive heart failure including changes in murmur intensity, new gallop sound, tachypnea/dyspnea, crackles, pleural effusion, and new onset of pulmonary edema on lung ultrasound. I've summarized the risk assessment and considerations for fluid therapy in Figure 4.2.

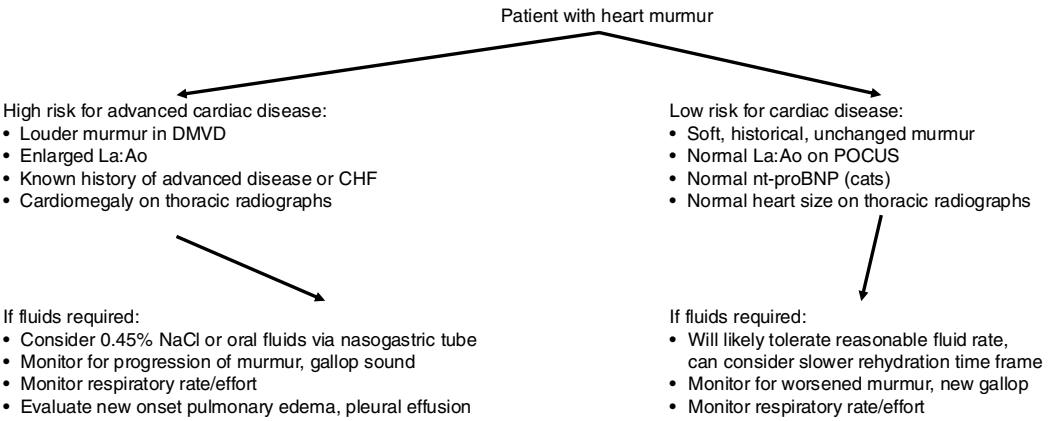


FIGURE 4.2 How to evaluate and manage risk factors for fluid therapy in patients with potential cardiac disease. Figure courtesy of the author.

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NONCARDIAC RISKS OF FLUID ADMINISTRATION

Beyond congestive heart failure, inappropriate or excessive fluid administration can cause adverse effects known collectively as fluid overload. This syndrome is best defined as the development of tissue/organ edema, effusion, or weight gain in a patient receiving intravenous fluids. (Hansen 2021). Fluid overload is caused by a combination of fluid therapy and an underlying disease state leading to decreased fluid excretion (Thomovsky et al. 2016). Diseases that predispose a patient to developing fluid overload would include oliguric/anuric renal failure, sepsis, hypoalbuminemia, and patients with systemic inflammation (O'Connor and Prowle 2015). Clinical signs associated with fluid overload can include inappropriate weight gain, interstitial tissue edema, serous nasal discharge, and tachypnea due to pulmonary edema (Pardo et al. 2024). Adverse effects of fluid overload include impaired renal, cardiopulmonary, and hepatic function as well as gastrointestinal ileus and poor wound healing (Thomovsky et al. 2016, O'Connor and Prowle 2015, Hansen 2021). There are currently two veterinary studies examining the adverse effects of fluid overload. The first showed an increased mortality in a diverse population of critically ill dogs with fluid overload (Cavanagh et al. 2016), while the second smaller study in feline urethral obstruction showed increased duration of hospitalization and cost but no difference in mortality (Ostroski et al. 2017).

CORRECTING ELECTROLYTE ABNORMALITIES

The calculations and tables for correcting sodium, potassium, and bicarbonate abnormalities are well covered in many other references, and I won't repeat them here. Since crystalloid fluids contain many of the same constituent ions as plasma, albeit in differing concentrations, they can and will alter the concentrations of sodium, chloride, and potassium (among others). However, that ignores the important role the kidney and hormonal (i.e. Renin–angiotensin–aldosterone and antidiuretic hormone) systems also play in regulating plasma ion balance. Therefore, the first question you should ask is based on my provisional diagnosis for the cause of these electrolyte abnormalities, are those systems functional? For example, giving saline to an Addisonian will only produce mild changes in the sodium because there is no aldosterone to force the kidney to reabsorb it. Treating the underlying disease condition that has caused these changes is exponentially more important than attempting to correct any abnormalities with fluids alone. In almost every case, the body is exponentially better at regulating its own electrolytes than our crude attempts at manipulating it. Secondly, always ask if the treatments you are instituting are going to change any electrolytes. This is most relevant in the setting of diabetic ketoacidosis where high doses of insulin have the potential to dramatically change phosphorus and potassium levels. Ultimately, when correcting any electrolyte abnormality, the most important thing to remember is to recheck those values to make sure you are making progress and haven't overshot the mark.

SPECIAL CASES

There are certain disease processes where fluid therapy may differ from the basic principles outlined above, and the detrimental effects of overly aggressive treatment are more pronounced. The first is in acute kidney injury (see also Chapter 18), where the goal is to more rapid rehydration (within 6 hours) with more frequent assessment of urine output, body weight, other signs of overhydration to prevent progressive renal dysfunction (Langston and Gordon 2021, Segev et al. 2024). The second is in hemorrhage and trauma, where overly aggressive fluid therapy can promote a coagulopathic state and lead to worsened bleeding (Palmer 2017). In these patients, earlier use of blood products (if fluid is required) likely leads to better outcomes (Hall and Drobatz 2021). Finally, in patients with sepsis/septic shock, poor vascular tone and concurrent hypoalbuminemia are risk factors for fluid overload, with recommended early use of vasopressors if patients fail to respond to initial fluid loading (Montealegre and Lyons 2021).

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CONCLUSIONS

The decision to give intravenous fluids should be based on an assessed need (either dehydration or hypovolemia) while avoiding the deleterious effects of inappropriate administration (fluid overload). The amount and rate of fluid administration depend on the severity of the underlying condition. Fluid choice/selection is only critical in a limited number of cases. Patients with heart murmurs and cardiac disease can receive fluids, provided you've done an appropriate risk/benefit analysis and appropriately monitor their respiratory status.

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Analgesia and Sedation

CHAPTER 5

CRITICAL QUESTIONS

1. Will my patient benefit from pain medications?
2. What is the risk/benefit ratio of sedating (or not sedating) this patient?
3. Have I adequately resuscitated the patient prior to sedation?
4. Is pain or anxiety *contributing* to the instability of my patient?
5. What effect will these medications have on the patient?
6. What are the goals for my sedation protocol?
7. How can I sedate the patient with a heart murmur?

INTRODUCTION

As with all decision-making in emergency medicine, choices around sedation and analgesia involve a detailed risk/benefit analysis. As the incidence of pain in the emergent or critically ill patient ranges between 37 and 54% (Wiese et al. 2005, Rousseau-Blass et al. 2020), appropriately identifying risk factors for adverse outcomes while simultaneously balancing the benefit of pain management and sedation is a crucial skill for the emergency clinician. The majority of clinicians feel comfortable prescribing a variety of opioid and non-opioid analgesics, and there are a number of excellent texts that already discuss their use (see additional reading section). Therefore, the majority of this chapter will focus on where many junior doctors struggle: the safe and appropriate use of sedation and chemical restraint.

BENEFITS OF EFFECTIVE ANALGESIA

The goal of the emergency clinician is to stabilize every patient as completely as we can. But what does that actually mean? In reality, it means improving our patient's condition from presentation, and it can be as simple as treating a urinary tract infection with antibiotics to treating hemorrhagic shock with a blood transfusion. Frequently, we use various markers such as heart rate, temperature, and lactate (among others) to track whether or not we are achieving our resuscitation goals. Besides our duty to alleviate animal suffering, unmanaged pain will cause perturbations in these values making it challenging to determine if we have appropriately stabilized our patient's condition.

Ineffective analgesia also increases a patient's overall metabolic work as their body and brain focus on pain, which diverts energy required for other important physiologic functions. (Hansen 2005, Simon et al. 2017). Newer clinicians will often prescribe inadequate analgesia because they are concerned about "unmasking" or causing cardiovascular instability. While inappropriately high doses of pain medications will undoubtedly cause problems such as respiratory depression with opioids, it is also important to realize that if your patient's perceived "stability" is due to a high sympathetic tone from pain, then that is even worse (Mathews 2018). In fact, if appropriate doses of analgesia cause a deleterious change, then in fact, your patient was unstable to begin with. As long as the clinician is ready to appropriately respond to changes after giving pain medications, the benefit of appropriate analgesia far outweighs the risk.

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ASSESSING THE NEED FOR ANALGESIA

Certain presenting complaints, such as trauma, automatically lend themselves to a pain assessment but always take care to assess for pain in patients with more vague presenting complaints, such as lethargy, since spinal pain is a frequent contributor to these signs.

There are multiple scoring systems available in veterinary medicine to assess for pain that can also be helpful as adjuncts to your history or physical exam. The majority of them, such as the short form Glasgow composite pain score (Holton et al. 2001, Reid et al. 2007) and Colorado State Acute Pain Scale among others (reviewed in Gruen et al. 2022, Monteiro et al. 2023) use a combination of behavioral and palpation clues to evaluate the presence and severity of pain. Beyond identifying pain in untreated patients, these scoring systems can also allow you to track and reevaluate the efficacy of your initial analgesia.

THE PERILS OF INADEQUATE SEDATION

Anxiety, similar to pain, will provoke a compensatory (though ultimately detrimental) stress response that can be linked to gastrointestinal complications, wound healing issues, altered pain perception, immunosuppression, and cardiac effects (Lefman and Prittie 2019). In certain cases, such as with brachycephalic pets, an exuberant stress response can even trigger a life-threatening upper airway crisis. Furthermore, from a practical standpoint, anxiety and stress in patients make commonplace procedures such as diagnostic imaging and phlebotomy more challenging and inefficient. While inappropriate or inadequate sedation can cause deleterious effects, appropriate sedation can improve patient outcomes.

RISK ASSESSMENT IN THE EMERGENT PATIENT

While there are multiple benefits to appropriate sedation and analgesia as discussed above, the use of pain medication and especially chemical restraint is not without some degree of risk. While mortality associated with anesthesia and sedation is generally low at less than 1%, the risk factors for death include the majority of the emergency patient population: the old and sick! (Shoop-Worral et al. 2022). Therefore, appropriate patient evaluation and risk factor identification are critical in reducing the likelihood of an adverse outcome.

Questions to consider when performing this assessment are as follows:

1. How old is the patient? Older patients will metabolize drugs slower than younger ones and therefore require lower doses (Baetge and Matthews 2012, Matthews et al. 2017).
2. Does my patient have any preexisting comorbidities in addition to the condition they are currently presenting with? Certain diseases (especially cardiac) can be destabilized with inappropriate drug selection, which we'll discuss in more detail below. In *elective procedures*, certain lab values such as albumin, PCV, and WBC have been shown to predict the risk of perianesthetic mortality (Salt et al. 2025).
3. Have I assessed for signs of underlying cardiovascular instability? If there are warning signs such as an abnormal heart rate, low blood pressure, or poor mucous membrane color, those should be addressed concurrently to any analgesics and certainly prior to sedation.
4. Have I assessed for signs and causes of respiratory instability such as pulmonary edema, pleural effusion, or pneumothorax? All of these should be addressed concurrently to analgesics and prior to sedation. However, the exceptions to this are if sedation is required to achieve stability (i.e. with an upper airway obstruction) or to facilitate a stabilizing procedure such as thoracocentesis.

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5. Finally, is the diagnostic yield from performing a procedure or test worth the level of risk from sedation? This can be a tough question to answer, especially for the newer clinician who is less comfortable with empiric treatment. Always consider if and how your plan will change based on immediate (versus delayed, post stabilization) diagnostic results.

STRATEGIES TO MITIGATE RISK

Multiple references exist that outline disease-specific sedation/anesthesia protocols, and so I will not cover them here, though I will provide you with my own approach later on. However, there are some important "first principles" to review that can reduce the risk of sedating the general emergency patient.

1. The sicker the patient, the lower your initial medication dose should be (with the possible exception of upper airway obstructions). You can always give additional medication if the first dose is inadequate.
2. Use reversible agents or short acting agents whenever possible in case they provoke an undesirable effect.
3. Always resuscitate prior to sedating unless sedation is required to stabilize your patient.
4. Always monitor to see if additional resuscitation (fluids, vasopressors, intubation) is required after the administration of analgesic or sedative agents.
5. Understand what the respiratory and cardiovascular effects of the drugs you're giving, so that you can monitor for them.

ASSESSING THE CARDIOVASCULAR AND PULMONARY EFFECTS OF SEDATIVE MEDICATIONS

What makes sedative medications unsafe? In relatively simple terms, they alter both cardiovascular (heart rate/rhythm, baroreceptor reflexes, cardiac contractility) and respiratory (respiratory rate, tidal volume) function (Haskins 2006). Inappropriate patient and drug selection can rapidly trigger cardiac and respiratory arrest. Understanding the impact these drugs have on these vital body systems (Table 5.1) can help you apply them in the appropriate situation.

While Table 5.1 is included to help you understand some of the effects these medications can have, there are a couple of important notes to make. The first is to evaluate the doses used in these studies. Several of them are much higher than what you would use clinically. Furthermore, many of the studies cited here used these medications as a sole agent, which, as we'll see below, is not reflective of clinical practice. Finally, one important thing to note about ketamine is that at high doses (and in sick patients) it can act as a direct myocardial depressant in some patients (Kawakubo et al. 1999, Duque et al. 2005). For doses typically used in the emergency setting (i.e. roughly 1–2 mg/kg), that effect is unlikely, though you should still use caution in cats with hypertrophic cardiomyopathy (HCM).

SOME WORDS ON DEXMEDETOMIDINE

You'll note I did not include dexmedetomidine on Table 5.1. There is perhaps no sedation drug that is more misunderstood in veterinary medicine, and so I felt it important to discuss separately. The advantages of dexmedetomidine are that it is an excellent sedative, provides some analgesia, is fast acting, and is reversible (Di Franco et al. 2023). However, due to several idiosyncrasies around its cardiovascular effects, its use in patients deemed "sick" remains very limited in veterinary medicine. Sedatives or high doses of dexmedetomidine will cause increases in blood pressure with

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Table 5.1 Selected analgesic and sedative doses along with expected cardiovascular and respiratory effects. Note that the doses in the first column are from the studies cited and do NOT reflect my recommended doses.

Drug/dose	Heart rate	Blood pressure	Cardiac output	Respiratory rate	Tidal volume	Source
Butorphanol (0.2 mg/kg IV)	Minimal effect	Minimal effect	Minimal effect	Minimal effect	Minimal effect	Jacobson (1994)
Hydromorphone (0.2 mg/kg IM)	Decreased	Minimal effect	Not measured	Minimal effect	Not measured	Smith (2001)
Fentanyl (15 ug/kg IV)	Decreased	Minimal effect	Minimal effect	Decreased	Decreased	Grimm et al. (2005)
Methadone. (0.5 mg/kg IV)	Decreased	Increased	Decreased	Minimal effect	Minimal effect	Maiante (2009)
Ketamine (10 mg/kg IV)	Increased	Increased	Increased	Mildly decreased	Not measured	Haskins et al. (1985)
Acepromazine (0.01 mg/kg IV)	Minimal effect	Decrease	Small decrease	Minimal effect	Not measured	Rangel et al. (2020)
Alfaxalone (4 mg/kg IM)	Increased	Decreased	No change	Decreased	Not measured	Cruz-Benedetti et al. (2018)
Propofol (4 mg/kg IV)	No change	Decreased	No change	Decreased	Not measured	Henao-Guerrero and Riccó (2014)

secondary decreases in heart rate and cardiac output due to a concurrent increase in afterload (Kellihan et al. 2015). This also leads to a reflex bradycardia, which can be disconcerting in critically ill patients. However, use of lower doses (roughly 0.5–1 mcg/kg) may mitigate many of these deleterious changes (Pascoe 2015). Furthermore, for patients that are "sick," they likely do not need a normal "sedative" dose to achieve your desired effect. Finally, for cats with HCM, the use of α2 agonists may be helpful in reducing left ventricular outflow obstruction (Lamont et al. 2002) and better maintenance of blood pressure despite a decrease in cardiac output (Keating et al. 2024). Again, the doses used in those studies were much higher (roughly 8–10 mcg/kg) than what would be recommended in a sick patient.

WHAT IS THE GOAL OF MY SEDATION PROTOCOL?

Our discussion above has justifiably centered on the cardiovascular and respiratory effects of sedative medications because a safe sedation should be your number one priority. However, to this point, we have not discussed ensuring you achieve your overall sedation goal, which is allowing you to perform whatever diagnostic or therapeutic procedure you need to accomplish.

There are two main (non-safety) factors to consider when creating a sedation protocol. The first is what procedure you are trying to complete and how stimulating/painful it is. The second is how agitated or stressed the patient is. This next section will review how I "build" a sedation protocol based on those two factors and what the two "base" protocols I commonly use are, and more importantly, how I modify them according to use, along with their procedural indications and potential modifications.

1. Opioids alone
   The first drug I will typically consider is an opioid because as we saw above, it is the most cardiovascularly sparing and least likely to cause a paradoxical excitatory effect in dogs. While hydromorphone is an excellent analgesic, it has minimal sedation properties on its own (Hofmeister et al. 2010), and so I will always use it with most patients with a secondary agent (see below). Methadone similarly has minimal sedation properties (Monteiro et al. 2008), and so I will tend to pair it with a second agent, especially in brachycephalics, since

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it is less likely to induce vomiting (Kerr and Swanton 2023). Therefore, butorphanol is the only opioid agent I will typically reach for as a sole agent sedative. The typical patient population I reach for butorphanol monotherapy in is older and/or more compliant patients who I just need to "take the edge off" to facilitate a relatively non-stimulating procedure such as intravenous catheter placement or radiographs. The dose I pick (0.2–0.4 mg/kg) is dependent on the route of administration as well as a guesstimate based of age and compliance. Low dose (0.1 mg/kg) butorphanol given intravenously is unlikely to be sedating in the vast majority of dogs (Girard et al. 2010). Its sedative effect is generally dose-dependent with higher doses (~0.2–0.4 mg/kg) capable of producing more pronounced sedation (Gomes et al. 2018, Springfield 2022). One caveat on the studies cited is that they tend to always evaluate middle-aged, healthy pets, and so in all likelihood, the degree of sedation will be higher at comparable doses in the older/sicker population we encounter.

2. The second agent: acepromazine, dexmedetomidine, midazolam
Frequently, butorphanol monotherapy provides inadequate sedation, and so a second agent has to be employed. As a part of my personal practice, I do not employ acepromazine due to my concern over the potential for hypotension and lack of a reversal agent. That being said, it can be a reasonable sedative and in combination with butorphanol will produce a more profound sedating effect than either agent independently (Dantino et al. 2022). The decision of dexmedetomidine versus midazolam as a second agent relates to my assessment of the patient's age, underlying cardiac disease, and shock status (see above). For canine patients (especially older ones) with advanced cardiac disease or some residual shock indicators post resuscitation, I will tend to use midazolam (on average about 0.1–0.2 mg/kg IV) and will reach for a third agent (see below) if additional sedation is needed. For younger patients with no underlying cardiac disease, dexmedetomidine is clearly the superior choice. For older patients with no cardiac disease or shock indicators, I will tend to use lower doses (less than or equal to 5 mcg/kg) of dexmedetomidine. Other factors that will guide my dexmedetomidine dose are the route of administration and the duration of sedation required. Lower doses of dexmedetomidine are required when given intravenously than intramuscularly (Granholm et al. 2007). The duration of sedation with dexmedetomidine is also dose dependent (Pypendop and Ilkiw 2014). My typical intravenous doses will be 1–5 mcg/kg with the following determining factors: age, degree of agitation, degree of stimulation associated with the procedure, and desired duration of sedation. Typical intramuscular doses are 5–10 mcg/kg with similar dose selection criteria as above. Interestingly, intramuscular injection may blunt some of the cardiovascular effects of dexmedetomidine due to increased time of absorption (Pan et al. 2021).

3. The third agent: alfaxalone, ketamine, propofol
I will typically reach for a third agent in the following scenarios: patient is fractious enough where two agents will be insufficient, the duration of the procedure is longer than my two-agent sedation will allow, or the procedure is stimulating enough to require additional sedation. For the fractious dog, I will typically add on ketamine (1–2 mg/kg IM), while for the fractious cat, I will typically add on alfaxalone (1–2 mg/kg IM). I try not to add high-dose ketamine in cats because of the risk of deterioration in cats with occult HCM (Adami and Monticelli 2020). For non-compliant patients with intravenous access, I tend to use propofol the majority of the time because it is titratable and reasonably sparing of the cardiovascular system. I will similarly use it in procedures such as laceration repairs that might take longer than a simple diagnostic procedure, such as radiographs. However, despite its advantages of rapid onset/offset, it is also important not to use it as a crutch in avoiding other agents, as large volumes of propofol can cause hypotension and respiratory depression, especially in sicker patients. Low-dose ketamine can also be a helpful adjunct in these scenarios as well, especially in procedures that will cause significant stimulation or discomfort.

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Table 5.2 The author's sedation approach to different clinical scenarios.

Patient characteristics/ clinical scenario	First-line protocol	Adjuncts/modifications
Anxious healthy dog	Butorphanol 0.2–0.4 mg/kg, dexmedetomidine 3–7 mcg/kg (IV or IM). Substitute hydromorphone (0.1–0.2 mg/kg) if painful	Propofol 1–4 mg/kg (if IV access), otherwise ketamine 1–2 mg/kg IM
Aggressive healthy dog for any procedure	Butorphanol 0.3–0.4 mg/kg, dexmedetomidine 7–10 mcg/kg, ketamine 2 mg/kg all given IM	Augment with propofol as needed once IV access obtained. Substitute hydromorphone for painful procedures
Dog with mild heart disease	Butorphanol 0.3–0.4 mg/kg, dexmedetomidine 1 mcg/kg IV +/– midazolam 0.2 mg/kg IM or alfaxalone 1 mg/kg IM if unable to get IV	Low dose propofol (1–2 mg/kg IV) or alfaxalone (1 mg/kg) for lengthier procedures or additional sedation. Can substitute in hydromorphone for painful procedures
Dog with moderate to severe heart disease	Butorphanol 0.2–0.4 mg/kg, midazolam 0.2 mg/kg IV/IM	Add in alfaxalone 1 mg/kg IM if unable to get IV, then titrate either propofol or alfaxalone. Can substitute in hydromorphone for painful procedures
Healthy, anxious cat	Butorphanol 0.2–0.4 mg/kg; dexmedetomidine 3–7 mcg/kg (IV or IM). Substitute hydromorphone (0.1–0.2 mg/kg) if painful	Propofol 1–4 mg/kg (if IV access), otherwise ketamine 1–2 mg/kg IM
Healthy, aggressive cat	Butorphanol 0.3–0.4 mg/kg, dexmedetomidine 7–10 mcg/kg IM, add in ketamine 1–2 mg/kg IM in severely fractious cats	If insufficient, give alfaxalone 1 mg/kg IM
Anxious/aggressive cat with suspected advanced cardiac disease	Butorphanol 0.3 mg/kg, midazolam 0.2 mg/kg, alfaxalone 1 m/kg IM	Can usually facilitate IV catheter placement or thoracocentesis

While I've put my own personal protocols in Table 5.2, I think it's important to acknowledge that the majority of protocols out there are based on expert opinion. The best evidence-based protocol in dogs I was able to find is a combination of butorphanol (0.3 mg/kg), dexmedetomidine (5 mcg/kg), and ketamine (1 mg/kg) IM for healthy dogs (Imboden et al. 2023). There are multiple described protocols in cats (Simon and Steagall 2020, Bagardi et al. 2023) that I think are reasonable as well.

One final note on cats in general: Feline procedural sedation is commonplace as they can be occasionally more difficult to handle in the clinical setting. Beyond the general principles outlined above, there was an excellent review of feline specific protocols that does an excellent job summarizing the pros and cons of different medications and dosing protocols (Simon and Steagall 2020).

WHAT IF MY INITIAL PLAN IS INADEQUATE?

Frequently, one's initial medication plan may not be enough to achieve the desired or required level of sedation. In these situations, ask the following questions:

- Can I get an IV catheter to give additional medications?
- How close am I to the "maximum" recommended doses recommended for intramuscular administration?
- Is there another drug class I haven't given?

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Evaluating route of administration, drug class, and dose will allow you develop a "second tier" sedation plan. If I have an IV catheter (or my initial plan is enough to allow placement), I will typically either give additional (albeit lower) doses of the same medications used in my initial plan or titrate propofol. For animals where I cannot get IV access, I will augment my protocol with additional intramuscular doses (up to recommended maximum) and consider using either ketamine (2–3 mg/kg) or alfaxalone (1–2 mg/kg) depending on patient size and level of aggression.

SEDATING THE PET WITH HEART DISEASE

As we discussed in Chapter 4, there are many ways to assess the severity of a patient's cardiac disease. When considering sedation options, it is also important to consider what the patients' underlying cardiac disease is so that you know what drugs to avoid. In dogs, the most common cardiac disease is degenerative mitral valve disease (DMVD) (though always assess risk factors for dilated cardiomyopathy [DCM] in larger breed dogs), while in cats, the most common cardiac disease is HCM (Payne et al. 2015). For dogs with DMVD with evidence of ACVIM Consensus Stage B2 cardiac changes as assessed either through thoracic radiographs or point of care ultrasound, avoid using dexmedetomidine as it can increase myocardial work, increase the regurgitant fraction, and decrease cardiac output (Crosland et al. 2021). The use of low dose (i.e. 1 mcg/kg) dexmedetomidine is likely okay in patients with stage B1 though that has not been evaluated. While the newer medication medetomidine/vatinoxan is safe in dogs with B1 DMVD (Välimäki et al. 2024), it may not be universally accessible. In the rare DCM dog, the use of opioids, benzodiazepines, and propofol is likely a safe approach (Yankina et al. 2024). For cats with HCM, the only drug to avoid is ketamine, with the use of opioids, benzodiazepines, alfaxalone, and low dose dexmedetomidine all as reasonable options (Keating et al. 2024). My personal combination for the fractious cat in heart failure is butorphanol 0.3 mg/kg, midazolam 0.2 mg/kg, and alfaxalone 1 mg/kg intramuscular, which is frequently successful at facilitating catheter placement and thoracocentesis in the majority of cats.

CONCLUSIONS

As you can see, there is both an art and a science to sedation in the emergency room. When considering sedation options, always consider the risks/benefits of sedating (or not sedating) as well as your patient's cardiovascular status along with your goals for your protocol. Using multiple medications will allow you to minimize the dose of each individual drug and hopefully minimize each drug's potential adverse effects. Sicker or older patients will typically require lower doses of medication to achieve a sedative effect. Mastering these basic principles will allow you to reasonably approach the sedation of any patient, and over time, you will develop your own "go to" protocols for certain scenarios.

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Use of Antibiotics in the Emergent Patient

CHAPTER 6

CRITICAL QUESTIONS

1. Does this patient actually require antibiotics?
2. What is the location and etiology for the presumptive infection?
3. Does this patient have risk factors for antibiotic resistance or a multi-drug resistant infection?

ASSESSING THE NEED FOR ANTIBIOTICS

Antibiotics are probably one of the most used and abused medications, especially in the emergency setting. The decision as to whether or not to start antibiotics (and which drugs to select) is one that the emergency clinician faces on a daily basis.

In an ideal world, antibiotics would only be used in the presence of a documented bacterial infection, and the agent used would selectively target the causative organism. That ideal world doesn't exist. Verifying the presence of bacterial infection requires culture of a pathogenic organism, which is impractical in the emergency setting. Directly visualizing bacteria in a body fluid (blood, urine, cavitary effusion) is more expeditious than culture, but the comfort level for emergency personnel in utilizing these tests can be variable (Pavlides et al. 2020), and indeed it has limited sensitivity in the hands of clinical pathologists (Allen and Evans 2022, Medardo et al. 2024). Therefore, we are oftentimes left treating based on both the suspicion of infection and a hypothetical bacterial species of unknown susceptibility.

That being said, there are multiple diagnostic tools available to guide decision-making as to the initiation and selection of antimicrobial therapy. The first are the "soft" findings that may or may not indicate the presence of bacterial infection. These are things like fever, physical exam evidence of inflammation (redness/swelling), suggestive laboratory findings such as hematuria, low or high white blood cell counts. While the presence of these factors is consistent with an infection, they are not pathognomonic, and their absence does not rule out infection, nor does their presence rule one in. The second are "harder" findings that are more suggestive of bacterial infection, such as imaging findings of pneumonia, pyuria/bacteriuria on a urinalysis, or a positive serology result. And finally, as discussed above, the definitive diagnosis for the presence of bacteria is both direct visualization of an organism on cytology (which can also be helpful in differentiating a commensal bacterial population through the presence of an inflammatory reaction) and culture, though at times these samples may be challenging to obtain and submit.

The decision to start antibiotics is multifactorial and requires the clinician to combine their historical, physical exam, imaging, and initial laboratory findings to reach a presumptive diagnosis that reasonably includes a bacterial component. However, equally important to the decision to initiate antibiotics is the follow-up to make sure they are actually improving the patient's clinical signs.

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PRINCIPLES TO SELECT THE RIGHT EMPIRIC ANTIBIOTIC

The emergency clinician will almost never have access to culture data to guide antibiotic selection at the time of presentation. Therefore, by default, we are left prescribing antibiotics empirically, without knowing whether or not they are appropriate. However, by taking a disease- and anatomy-oriented approach (along with a risk assessment for drug resistance, we'll talk about later), it can become much more straightforward to make these empiric medication decisions.

Hopefully, based on our assessment from above and the testing discussed in previous chapters, we're at least able to locate an area of the body for the suspected infection. Common sites for infection seen through the emergency department are gastrointestinal, upper respiratory/oral cavity, urogenital, lower respiratory, hepatobiliary, skin/soft tissue, and vector-borne diseases (Robbins et al. 2020). Localizing the area of infection (in combination with the suspected underlying disease process) then allows us to generate a list of likely bacteria and, by extension, rational antibiotics. As we'll see below, understanding what and where an infection is also allows you to access a multitude of resources that can help guide initial antibiotic selection.

AN ANATOMY- AND DISEASE-ORIENTED APPROACH TO ANTIBIOTIC SELECTION

The goal of this section is to start anatomically from the nose and work caudally to review common sites and causes of bacterial infection, along with current antibiotic recommendations to treat those infections.

UPPER RESPIRATORY TRACT/INTRACRANIAL DISEASE

Common bacterial infections of the upper respiratory tract and mouth include feline upper respiratory infections (URIs), canine infectious respiratory disease complex (CIRDC), and tooth root/retrobulbar abscesses. We'll also cover cervical abscesses here, as they are a fairly common presenting complaint.

While the majority of feline URIs are viral, the presence of (muco)purulent discharge may suggest co-infection with certain *Staphylococcus, Streptococcus, Mycoplasma*, and *Chlamydia* species. Current recommendations for antimicrobial therapy in these cats are either amoxicillin or doxycycline (Lappin et al. 2017). Similarly, in dogs with CIRDC, the majority are viral, though co-infection with *Bordetella, Streptococcus,* and *Mycoplasma* species is possible. Doxycycline can be considered in patients with fever/lethargy but no evidence of pneumonia. (Lappin et al. 2017).

Suborbital swellings in the presence of dental disease are usually indicative of an apical tooth root abscess. While surgical extraction is the definitive therapy for these abscesses, that is rarely practical in the emergency setting, and antibiotic therapy is frequently employed as a temporizing measure. While the most common antibiotics used in the dental setting are clindamycin, amoxicillin, and amoxicillin-clavulanate (Weese et al. 2023); the one study evaluating culture data in tooth root abscesses indicated a high level of resistance to these medications (Chawnan et al. 2021). Given the high incidence of anaerobic and gram-negative species in that study, either clindamycin or amoxicillin-clavulanate would likely be reasonable empiric options.

Retrobulbar abscesses present a unique challenge as it is frequently impractical to drain them (requiring a prolonged course of antibiotics), and obtaining a culture sample can be challenging. Available culture data indicate a high prevalence of multi-microbial infections with a mixture of aerobes and anaerobes. Current antibiotic recommendations based on those data would be either amoxicillin-clavulanate or a combination of clindamycin and enrofloxacin. (Fischer et al. 2018, Pumphrey and Wayne 2022, Herrmann et al. 2021)

Cervical abscesses (classically characterized by a peracute onset of swelling in a dog known for stick chewing as shown in Figure 6.1) are a common emergency room presentation.

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FIGURE 6.1 Gross and CT images of typical cervical abscess. *Source*: Walker et al. (2023)/Springer Nature/CC BY 4.0/CC0 1.0.

While much is not known about the ideal diagnostic imaging and treatment approaches, culture data indicates common organisms include *Streptococcus, Bacteroides,* and *Pasteurella*. (Walker et al. 2023, Welch et al. 2024). Given the presence of both aerobic and anaerobic bacteria, amoxicillin-clavulanate was the most commonly selected antibiotic in both studies, and resistance to that agent was rare.

THORACIC CAVITY

Moving backward, the next anatomic area to discuss is the thoracic cavity. Since endocarditis is uncommon (and even more rare to diagnose on an emergency basis), we won't discuss it here. The two most likely conditions you'll encounter that might require antibiotics are pneumonia and pyothorax. Pneumonia can be differentiated into community-acquired (the majority of which are secondary to *Bordetella spp.*) and aspiration, which are most due to gram-negative enteric bacteria, with *E. coli* as the most common (Proulx et al. 2024). Current recommendations for community-acquired pneumonia depend on illness severity with less sick patients receiving doxycycline and sicker patients receiving enrofloxacin and a potentiated penicillin (Lappin et al. 2017). The latter recommendation is similar to that made for patients with aspiration pneumonia. Pyothorax can be slightly more complicated, as traditionally, medical management for cats has been recommended, while surgery is typically indicated for dogs. However, from a bacteriological standpoint, it is not uncommon for patients to have polymicrobial infections that can involve aerobic and anaerobic bacteria (Johnson et al. 2023). Current recommendations involve the use of a potentiated penicillin along with a fluoroquinolone though which agent to use (i.e. enrofloxacin versus marbofloxacin) is debatable (Sim et al. 2021, Johnson et al. 2023, Boothe et al. 2010). In my personal anecdotal experience, the majority of etiologic agents cultured have been susceptible to a potentiated penicillin such as amoxicillin-clavulanate.

HEPATOBILIARY SYSTEM

The next system anatomically is the hepatobiliary system, where commonly encountered bacterial diseases include feline cholangiohepatitis and gallbladder mucoceles. We won't delve into the diagnosis and surgical management for these diseases but rather focus on the etiologic agents and recommended treatments for these disease processes. For cats with bacterial cholangiohepatitis, the most common organisms were aerobes or facultative anaerobes such as *E. coli* and *Enterococcus* (Center et al. 2022, Schlachet et al. 2025). Recommended antibiotics still include the combined use of a potentiated penicillin, fluoroquinolone, and metronidazole (Center et al. 2022, Jaffey 2022), with MDR

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organisms more common in patients with previous antibiotic history (Schlachet et al. 2025). While surgical treatment for gallbladder mucoceles in dogs is generally associated with improved prognosis (Parkanzky et al. 2019), medical management can be useful both as adjunctive or primary therapy in cases of non-ruptured mucoceles. Interestingly, the incidence of positive culture in gallbladder mucoceles is highly variable ranging from 3 to 66%! Similar to cats, the most common isolates tend to include *E. coli* and *Enterococcus*, therefore a similar antibiotic approach is prudent, given that the majority of larger studies did not report susceptibility data (Galley et al. 2022, Jaffey 2022).

GASTROINTESTINAL DISEASE

Commonly encountered bacteria-mediated diseases in the gastrointestinal tract include septic peritonitis (due to intestinal rupture), secondary bacterial infections due to canine parvoviral enteritis, and controversially, acute hemorrhagic diarrhea syndrome (AHDS). While surgical intervention is required for definitive source control in septic peritonitis, early antibiotic administration (currently within 1–3 hours in human medicine) has been demonstrated to influence mortality in sepsis (Sterling et al. 2015). Unsurprisingly, the most common organisms implicated include enteric bacteria such as *E. coli* and *Enterococcus* (Marshall et al. 2019, Dickinson et al. 2015). Typically employed antibiotic choices would include ampicillin/sulbactam and enrofloxacin, though a high-generation cephalosporin such as ceftazidime may also be appropriate (Kalafut et al. 2018). Interestingly, adequacy of antibiotic selection has not been shown to influence mortality in this disease, likely due to the need for surgical intervention (Marshall et al. 2019, Dickinson et al. 2015, Kalafut et al. 2018). While canine parvoviral enteritis is not directly treatable with antibiotics, they can play a role in minimizing the risk for secondary infection as these patients are frequently immunocompromised/immune-naive, especially in leukopenic dogs (Sykes 2010, Mazzaferro 2020). Furthermore, co-infection with a variety of Clostridial and Salmonella species along with other enteric bacteria has been reported in dogs with diarrhea (Tupler et al. 2012, Botha et al. 2018, Silva et al. 2017)

Despite the generally accepted hypothesis that AHDS is at least partially due to Clostridial species (Busch and Unterer 2022), the use of antibiotics such as amoxicillin-clavulanate and metronidazole is controversial, and most consensus statements recommend against routine use except for severe disease (Jessen et al. 2024). Several studies have shown that in the absence of sepsis indicators, these medications provide no therapeutic benefit (Unterer et al. 2011, Reisinger et al. 2024, Ortiz et al. 2018, Dupont et al. 2021). We'll discuss the debate more in the chapter on gastrointestinal disease.

UROGENITAL

Common urogenital infections include urinary tract infections (far and away most common), pyelonephritis, and pyometra. Thankfully, there are relatively current guidelines for managing these cases (Weese et al. 2019) that we'll summarize here. For the uncomplicated urinary tract infection (UTI), which is the most common emergency room presentation, recommended antibiotics would include amoxicillin (+/− clavulanate) and trimethoprim sulfa. Current recommendations for pyelonephritis would include either a third-generation cephalosporin or fluoroquinolone, keeping in mind that culture results should be compared to serum breakpoints NOT urine, even though any culture submitted is urine. Finally, while pyometra (whether open or closed) is almost always managed surgically, adjunctive antibiotics are generally used in the perioperative period, though as we'll discuss in Chapter 23, postoperative use is controversial. The vast majority of pyometras are secondary to *E. coli* with less commonly seen pathogens including *Staphylococcus* and *Streptococcus* organisms (Hagman and Greko 2005). The majority of *E. coli* have been shown to be sensitive to ampicillin though some studies indicate enrofloxacin (Coggan et al. 2008) may be superior. However, and similar to with septic peritonitis, surgery rather than appropriate antibiotic selection is likely the driver of survival.

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INTEGUMENTARY/OTHER

Bacterial infections affecting the skin range from a simple pyoderma, to abscesses, to life-threatening necrotizing soft tissue infections (NSTIs). Thankfully, the most common presentation is a simple pyoderma secondary to *Staphylococcus pseudintermedius* (though due to increasing antimicrobial resistance, many "simple" pyodermas may actually be more complicated). Current guidelines recommend a combination of topical therapy, such as shampoos, with systemic clindamycin or a first-generation cephalosporin (Hillier et al. 2014) as initial treatments. For soft tissue abscesses, there is an important species difference between dogs and cats. For dogs, the majority of abscesses are usually due to internal or external penetrating foreign bodies (the majority of dog bite wounds tend not to form abscesses if treated appropriately). These cases are likely similar to the cervical abscesses discussed above. Cat bite abscesses, however, are different due to their more virulent oropharyngeal flora with a high prevalence of anaerobic bacteria (Love et al. 2000). Antibiotics that have been shown to have reasonable activity (in addition to surgical drainage) include amoxicillin (+/− clavulanate) and cefovecin (Torres-Henderson et al. 2014). While thankfully rare, severe soft tissue infections (encompassing NSTIs and necrotizing fasciitis) are a true emergency as they can trigger systemic complications and can require extensive surgical debridement. Given the complexities and nuance in surgical management for these patients, we're going to focus on antibiotic therapy, but it is critical that you recognize these potentially more complicated infections, as shown in Figures 6.2 and 6.3.

The majority of these infections are caused by *Streptococcus* species, and current recommendations would likely be a combination of ampicillin/sulbactam and clindamycin, with enrofloxacin being contraindicated in these cases (Buriko et al. 2008, Quilling et al. 2022).



FIGURE 6.2 Necrotizing cellulitis secondary to cat scratch in a dog. Note the darker, devitalized tissue around the wound edge indicative of nonviable tissue that extend to the right (cranial). This patient required extensive surgical debridement and skin grafting. *Source*: Banovic et al. (2013) with permission of John Wiley & Sons.



FIGURE 6.3 Severe cellulitis that triggered a septic response and necessitated open wound management. Note the marked lines in the image on the right used to track disease progression. *Source*: Worthing et al. (2019) with permission of John Wiley & Sons.

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Finally, we'll briefly discuss management of bacteria-mediated vector-borne diseases with a focus on those commonly seen and studied in the United States. These include: *Anaplasma, Babesia, Borrelia, Bartonella, Ehrlichia,* and *Rickettsia* species. Currently, the majority of these diseases are managed through doxycycline.

IDENTIFYING RISK FACTORS FOR MULTIDRUG-RESISTANT INFECTIONS

The rising use of antibiotics has led to a concurrent rise in multidrug-resistant (MDR) infections. Identifying these infections is critical because these are the patients who may not respond to the first-line, empiric antibiotics discussed above. Secondly, this patient is one to highly consider the cost and risk of culturing. Current risk factors for the presence of MDR bacteria in veterinary medicine include recent antibiotic administration (usually within 3 months), ingestion of a raw diet, and recent hospitalization for greater than 3 days (Karalliu et al. 2024, Hernandez et al. 2014), which are similar to risk factors identified in human medicine (González del Castillo et al. 2020). Arguably, these are the patients where the benefit of obtaining a culture sample outweighs the cost and procedural risk (i.e. endotracheal wash, cholecystocentesis, etc.).

Beyond the risk factors for an MDR infection above, other factors that should drive you toward culture should be a deep infection, surgical site infections, hospital-acquired infection, and diseases where prolonged (>1–2 weeks) of antibiotics are required (Allerton and Nuttall 2021).

MONITORING RESPONSE TO THERAPY

For all patients, especially those with more complicated or resistant infections, monitoring for improvement in their condition is crucial. Besides signs of clinical improvement (improved activity level, appetite), a successful treatment should also lead to improvements in vital parameters (especially temperature) and clinical symptoms. Ideally, treatment success would also be evaluated through biomarkers such as C-reactive protein and procalcitonin (Viitanen et al. 2017, Goggs et al. 2022, Fernandes Rodrigues et al. 2022), though these values are frequently unavailable. While the appropriate duration for many diseases is unknown (Keir and Dickinson 2015, Robbins et al. 2020), there is evidence that shorter courses are appropriate in pneumonia (Vientós-Plotts et al. 2021).

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Decision-making for Hospital Admission

CHAPTER 7

CRITICAL QUESTIONS

1. Do I know what disease process is occurring (and do I expect it to progress)?
2. Is there a diagnostic test or treatment required that can only be done in the hospital?
3. Will the patient tolerate hospitalization?
4. What are the owners' goals of care?

INTRODUCTION

While for some patients, making the recommendation to admit them to the hospital is quite clear, such as the patient with polytrauma, there are many patients for whom it is unclear whether or not they require inpatient care. There is virtually no literature in veterinary medicine that has examined either criteria or decision-making tools that guide inpatient versus outpatient care. Therefore, unfortunately, a good portion of this chapter will represent my opinion and thought processes. In human medicine, the majority of clinical decision-making guidelines are disease-specific (i.e. for a patient with pneumonia, these factors dictate their risk of deteriorating on an outpatient basis) even though their accuracy is sometimes questionable (Soleimanpour and Bann 2022). In human medicine, a review study identified patient (i.e. preference, living situation), clinician (risk tolerance, knowledge level), caseload/staffing, and financial (insurance) factors that typically guide disposition decision-making among others, as shown in Figure 7.1 (Rutkowski et al. 2023, Capan et al. 2018).

As we consider our critical questions from above, what is fascinating is the similarity and overlap between human and veterinary medicine in the qualitative factors that guide the admission decision, where the benefit of hospitalization is unclear. In examining a population of patients "without a defined medical acuity" (Trinh et al. 2021), researchers identified both social and medical factors that guided the admission decision, such as need for specialist workup, inability to access primary care, and outpatient treatment failure.

DISEASE PROCESS FACTORS

Clearly, the largest factor that can and should impact the admission decision is the actual diagnosis. Understanding what disease process is occurring is powerful because it allows you to confidently initiate treatment that (in theory) should improve the patient's condition, as opposed to empiric therapy, where there is more uncertainty. Making a diagnosis also allows a better prediction as to whether or not that disease will progress. For example, a patient vomiting due to mild pancreatitis can reasonably be expected to improve, provided you rehydrate them and stop the vomiting. Conversely, a patient with immune-mediated hemolytic anemia (IMHA) who requires a transfusion can reasonably be expected to decrease their PCV again, potentially to a dangerous level. Finally, knowing the diagnosis can help you educate the owner as to the expected clinical course and monitoring criteria to return to the hospital, thus decreasing the risk of an adverse outcome at home.

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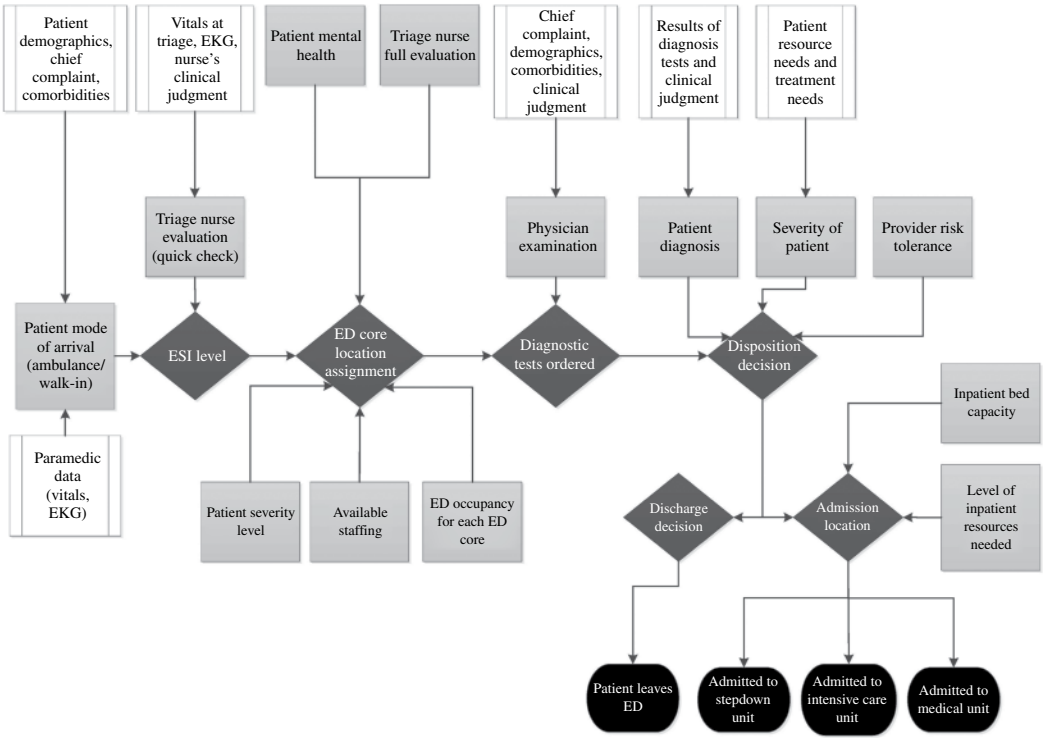


FIGURE 7.1 Flowchart from human medicine about the factors that go into the admit/discharge assessment, including decision points (dark grey diamonds), decision elements (light grey boxes), data points (white boxes), and final decision points (black circles). ESI stands for Emergency Severity Index. *Source*: Capan et al./(2018) with permission of Elsevier.

DIAGNOSTIC AND TREATMENT FACTORS

Excluding patient monitoring such as vital parameters and laboratory, there are several treatments that can only realistically be performed in a hospital. These include continuous fluid therapy/blood product administration, provision of injectable medications, pain management (insufficiently controlled with oral medication), and oxygen therapy. If based on disease process, patient assessment, or potential for disease progression, you feel they require or would benefit from any of those interventions, then hospital admission should be strongly considered.

Unfortunately, there are also times when more stable patients will end up being admitted for diagnostic testing, usually due to either outpatient specialist or imaging availability (i.e. a stable dog with epistaxis requiring a CT scan and rhinoscopy). When evaluating these patients for admission, consider the duration and progression of clinical signs. A patient with acute (but stable) clinical signs may be more suited to outpatient management than a patient with either progressive or chronic signs, as that patient's disease is likely more aggressive or advanced.

PATIENT AND CLIENT FACTORS

There can be patient characteristics that can act both in favor and against inpatient care. Patients unable or unwilling to take oral medications may need to be hospitalized for injectable medications. Age can be an important factor in guiding the decision to admit. Both older and younger patients have less physiologic reserve, and in the case of older patients, usually have more comorbidities. This puts these patient populations at higher risk for failing outpatient management. For patients

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Table 7.1 Some risk factors that should push you toward hospitalization in borderline cases.

Disease factors	Patient factors	Owner factors
Poorly controlled/unknown disease	Age (pediatric or geriatric)	Inability to medicate
Oxygen dependence	High comorbidity load	Inability to access short-term follow-up
Dehydration requiring IV fluid therapy	Inability to take oral medications	—
Requires specialist consultation or procedure	—	—
Pain not controlled with oral medications	—	—

who are anxious or aggressive, hospitalization may actually be counterproductive, and some patients will recover better at home in their normal environment as opposed to the stress of being in the hospital. Weighing these patient factors versus the benefits of hospitalization is an important conversation to have with the pet parents. Finally, you also have to take into consideration the risk of hospital-acquired infections, especially in immunocompromised patients.

Ultimately, it is the client who has to approve hospitalization, both from financial and consent to care standpoints. Beyond the finances, owner considerations may include improved recovery at home due to a pet's stress/anxiety in hospital (especially if older with chronic/terminal illnesses), ability to monitor or medicate, and their willingness to tolerate cleaning up urine, feces, or vomit! Also, consider the follow-up care available to the patient and owner. If they are unable to access quality follow-up on an outpatient basis, that may sway you toward hospital admission. Finally, from a medicolegal standpoint, in borderline cases, always ensure that you and the owner understand and are comfortable that hospital discharge can have adverse outcomes and may make future treatment more challenging and impossible. Documenting shared decision-making and recommendations is critical to protect yourself.

CONCLUSIONS

The admit/discharge decision is a challenging one that encompasses disease/treat, patient, and owner factors. I've summarized some of the risk factors that should sway you toward hospitalization (see Table 7.1).

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Clinical Reasoning for Common Diseases and Presentations

PART II

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Approach to the "Down Dog"

CHAPTER 8

CRITICAL QUESTIONS

1. Is this patient in shock or systemically ill?
2. Does this patient have a neurologic disease?
3. Could this be a drug or toxin ingestion?
4. Are there risk factors for orthopedic injuries?

INTRODUCTION

The "gurney" pet or one that can't walk is a reasonably common presentation and an even more common source of anxiety for the junior doctor. These patients can include those unable to walk due to collapse from shock/systemic disease, severe ataxia or paresis/plegia due to neurologic disease, drug/toxin ingestion, and orthopedic disease. Having a systematic approach to thinking about these patients will not only reduce your stress but also help build pattern recognition to make your assessments more efficient. Integrating vital parameters, physical exam, and point of care diagnostics (Table 8.1) can be helpful in appropriately categorizing these patients. You may find it helpful to start at the bottom of the table to identify some initial point of care diagnostic tests to consider with these patients as well as their association with certain disease conditions. And while this is not to say that you necessarily always have to place an IV catheter and perform point of care labs/ultrasound, but in the case of a down dog, no one should necessarily look askance at you for doing so.

Table 8.1 Potential abnormalities and diseases to consider in pets presenting "down."

	Shock/systemic illness	Neurologic disease	Drug/toxin ingestion	Orthopedic
Diseases to consider	Hemoabdomen, GDV, pericardial effusion, sepsis, hypoglycemia	Vestibular disease, acute spinal cord injury, lower motor neuron disease	Marijuana or opioid toxicosis	Immune-mediated polyarthropathy, hypertrophic osteodystrophy, panosteitis
Vital parameters	Usually high heart rate (except in cats). Temperature can be elevated, low blood pressure	Usually normal, except with increased intracranial pressure	Variable, TPR can be high, low, or normal	Can have fever, otherwise generally normal
Physical exam findings	Pale mucous membranes, poor pulse quality	Nystagmus/head tilt, decreased proprioceptive responses, flaccid muscle tone	Dull/obtunded mentation, otherwise generally normal	Joint/bone pain
Point of care Table 8.1 lab work	High lactate, low PCV	Usually normal	Usually normal	Usually normal
Point of care ultrasound	Can have cavitary effusions	Normal	Normal	Normal

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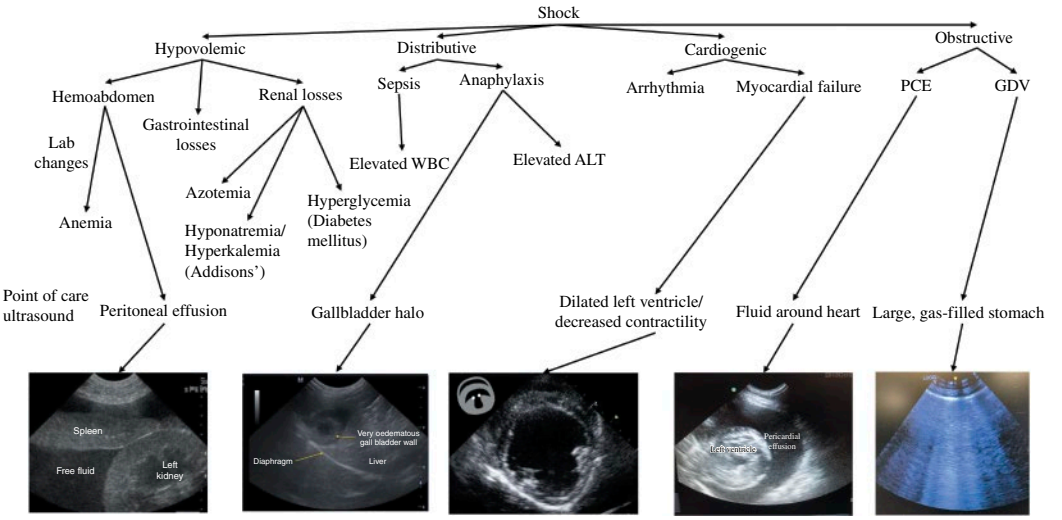


FIGURE 8.1 Common shock etiologies with common associated diagnostic findings. Courtesy of Calgary VPOCUS training academy.

SHOCK ASSESSMENT

Shock is a common cause of difficulty/inability to walk. It is also the most important etiology to intervene on emergently. In the triage exam chapter, we discussed strategies for shock identification using vital parameters, physical exam, and point of care diagnostics. Briefly, a high heart rate, pale mucous membranes, and poor pulse quality, combined with other parameters such as a low PCV, high lactate, low blood pressure, and evidence of cavitary effusions, would be suggestive of shock. I would also add here that fever can be another very common cause for unwillingness to walk (think of how you feel when you're feverish with the flu!). Finally, always assess for hypoglycemia in at-risk patients such as diabetics or toy breed puppies, as this is a quickly and easily reversible condition.

Once you've categorized your down patient into the "shock" category, your initial stabilization for virtually all (noncardiac) patients will be to give some form of a fluid bolus while you further investigate WHY they are in shock using point of care ultrasound (POCUS) and more substantial lab work (Figure 8.1). The goal of POCUS is to evaluate for any cavitary effusions or cardiac sources for shock that can guide initial stabilizing measures. Again, it may be helpful to start at the bottom of the figure with your diagnostic results and then move upward as you are attempting to subclassify the exact cause for their shock. We'll discuss the diagnostic and therapeutic approaches to several of the diseases in this figure in later chapters.

NEUROLOGIC ASSESSMENT

Diseases affecting the brain or spinal cord can be frequent causes for the down dog. As we saw in Table 8.1, there are certain exam findings that can be highly suggestive of spinal cord injury, vestibular syndrome, or a lower motor neuron disease such as myasthenia gravis. Signalment can also be helpful in subclassifying the etiology (i.e. the older labrador retriever with idiopathic vestibular disease versus the younger dachshund with intervertebral disc disease). We'll elaborate further on several of the more common neurologic disorders later on in Chapter 16 when we discuss neurologic emergencies.

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DRUG/TOXIN ASSESSMENT

These can occasionally be challenging to identify as they are frequently diagnoses of exclusion or clinical gestalt as one of the most commonly ingested drugs is marijuana, which is difficult to test for on a point of care basis (Fitzgerald et al. 2021). However, that doesn't mean there aren't clues you can use to increase your level of suspicion for drug ingestion. The majority of drug ingestions occur in younger dogs (Janczyk et al. 2004, Howard-Azzeh et al. 2020, Thomas et al. 2014) and tend to have a peracute onset of signs. The majority of the "depressant" drugs that may cause a down dog are usually either opioid (derivatives) and marijuana, though other drugs can cause similar signs (Oster et al. 2023, Binagia et al. 2024). However, their effects on a patient's vital parameters can be varied, making changes such as hypothermia, bradypnea, and bradycardia suggestive, but not definitive, signs of toxicosis. Point of care lab work will typically be nonspecific, and while urine drug testing is helpful for opioids, it is limited in its ability to reliably detect marijuana (Teitler 2009). Therefore, accurately classifying these "down" patients requires a combination of signalment, history, and physical exam with clinical intuition and a judicious use of urine testing. We'll discuss management for the intoxicated patient in Chapter 20.

ORTHOPEDIC ASSESSMENT

In the absence of trauma, there are only a few orthopedic diseases that would realistically be expected to cause an abrupt inability to walk. These are usually diseases that are affecting multiple joints or bones as a single limb disease such as a soft tissue injury may cause lameness, but generally not an inability to walk. I've listed some of the more common diseases you might encounter as well as some pattern recognition clues that may lead you to consider these diagnoses in Table 8.2.

INITIAL STABILIZING TREATMENT

While clearly there are differing treatment approaches for the varying etiologies of the "down dog," there are times when you have to institute treatment prior to a definitive diagnosis. Furthermore, as we discussed in the imaging chapter, some diagnostic tests (especially radiographs) can be stressful on the patient and require sedation. Correcting shock and treating any cause for dyspnea (as we'll discuss in a later chapter) will reduce the risk of an adverse event during your diagnostic workup. Some disease-specific treatments are summarized in Table 8.3

Table 8.2 Orthopedic (joint/bone) diseases that may cause a pet to become unable to walk as well as some risk factors and clinical clues that may increase your index of suspicion for these conditions.

Disease condition	Risk factors/clinical clues	Reference
Immune-mediated polyarthropathy (IMPA)	Fever, young to middle age, medium/large breed dog, multiple joint pain/effusion	Johnson and Mackin (2012)
Hypertrophic osteodystrophy (HOD)	Large/giant breed puppies, fever, radial/ulnar swelling	Selman and Millard (2022)
Panosteitis	Large/giant breed puppies, diaphyseal pain,	Kieves (2021)
Bilateral cranial cruciate rupture	Overweight, larger breed dog, history of previous rupture	Grierson et al. (2011)

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Table 8.3 Some initial stabilization measures to consider before diagnostic testing such as radiographs, full abdominal ultrasound, or echocardiography.

Disease process	Pre-diagnostic stabilization measures
GDV (see also Chapter 12)	IV fluid bolus, analgesia, trocharization +/– antiarrhythmics
Hemoabdomen (see also Chapter 11)	IV fluid bolus +/– blood transfusion
Pericardial effusion (see also Chapter 22)	Pericardiocentesis
Sepsis	IV fluids, antibiotics, vasopressors, +/– dextrose
Congestive heart failure (see also Chapter 22)	Oxygen, diuretics
Anaphylaxis	IV fluids, antihistamines, corticosteroids
Intracranial disease (see also Chapters 10 and 16)	Hyperosmolar therapy, antiepileptics

A BRIEF NOTE ON CATS

Even though this chapter has been mainly focused on dogs, I think it is important to briefly review some more "cat-specific" diagnoses. Probably the two can't miss diagnoses are urethral obstruction and feline arterial thromboembolism (FATE), both of which we'll talk in detail about in Chapters 13 and 22, respectively. Many cats with significant azotemia and hyperkalemia will also present laterally recumbent (see Chapter 18). One orthopedic disease seen primarily in young overweight male cats is slipped capital physes (McNicholas et al. 2002).

CONCLUSIONS

While the "down dog" can be nerve-wracking for a junior clinician, if you go through a checklist of assessing for shock, neurologic, and toxic etiologies, you will be less likely to overlook a "can't miss" diagnosis. For pets who you have ruled out those causes and who have certain other risk factors, consider the potential for a disease that can affect multiple joints or bones.

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Diabetic Emergencies

CHAPTER 9

CRITICAL QUESTIONS

1. Is it a stress hyperglycemia or true diabetes?
2. What insulin to start in a healthy diabetic?
3. Are they ketotic? Are they acidemic?
4. Do they have any concurrent disease processes?
5. When should I start insulin, and how do I monitor it?
6. Which insulin protocol should I use for a diabetic ketosis/diabetic ketoacidosis?
7. How do I evaluate a cat on SGLT-2 inhibitors?
8. Is this patient hyperosmolar/hyperglycemic, and how does my management differ?
9. What provoked the hypoglycemic incident, and when do I restart insulin?

INTRODUCTION

While diabetes mellitus is by definition a chronic disease, there are multiple ways a pet with diabetes can present to the emergency room. We'll consider our critical questions in the context of these five main presentations: the healthy/newly diagnosed diabetic, the sick nonketotic diabetic, the diabetic ketosis/ketoacidosis (DK/DKA), the hyperosmolar/hyperglycemic syndrome (HHS), and the hypoglycemic diabetic. Finally, you'll note at the end of certain sections there will be a portion labeled "client communication pearls." This is geared at providing you with some important talking points when discussing these diseases with an owner to make sure everyone has the appropriate knowledge and expectations regarding diagnosis, treatment, and prognosis.

THE "HEALTHY" DIABETIC

While infrequent, you will occasionally diagnose a diabetic animal who is otherwise healthy except for its polyuria/polydipsia, weight loss, and polyphagia. This can be a time-consuming challenge because it requires you to begin treatment of a chronic disease and communicate the complexities of managing what can be, admittedly, a difficult disease.

The first step is to confirm a diagnosis of diabetes mellitus, which requires the documentation of a persistent hyperglycemia and glucosuria along with appropriate concurrent clinical signs such as weight loss, polyuria/polydipsia, and polyphagia, among others. Unfortunately, the cortisol increase that accompanies a stress response can trigger an elevation in glucose, also known as stress hyperglycemia. Since roughly 40% of dogs and 60% of cats presenting to an emergency room may have a stress hyperglycemia (Hagley et al. 2020, Ray et al. 2009) with sicker cats having a higher risk for elevated glucose (Frezoulis et al. 2022), it is important to document the presence of other clinical signs prior making a definitive diagnosis and starting insulin therapy. For patients that you are on the fence about, you can consider having the pet rechecked with the family veterinarian for a glucose level or a fructosamine level (Crenshaw et al. 1996).

Once you feel comfortable with your diagnosis of diabetes mellitus, it is time to consider initiating insulin treatment. I think it is important to note that the most current consensus

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management guidelines are from 2018 and do NOT reflect the potential role for sodium glucose transporter-2 inhibitors (SGLT-2) in cats, which has been shown to be noninferior to insulin in certain feline diabetics (Behrend et al. 2024, Niessen et al. 2024). Current patient selection recommendations for these medications are healthy new diabetics with no ketosis, underlying renal dysfunction, or concurrent endocrinopathies (Cook and Behrend 2024), and their use has been recently reviewed in Vieira et al. (2025) and Kraemer and Ramsey (2025). Until there is consensus as to the appropriate indications for these medications, it is reasonable to start parenteral insulin. For dogs, current first-line insulin recommendations (Behrend et al. 2018) would be U-40 pork lente (i.e. Vetsulin), while for cats, it would either be glargine (i.e. Lantus) or prozinc (i.e. PZI). The insulin dose should be reevaluated 1–2 weeks after starting. You should also provide information about insulin storage and handling. In the longer term, diet change to a high protein/low carbohydrate food in cats is beneficial (Bennett et al. 2006).

CLIENT COMMUNICATION PEARLS

There are several key points to discuss with the owner of a newly diagnosed diabetic because it is a chronic disease requiring lifelong management and monitoring. It is estimated that roughly 10% of pets diagnosed with diabetes will be euthanized at the time of diagnosis, with an additional 10% within a year of diagnosis (Niessen et al. 2017). Ensuring that an owner has the ability to give insulin and will comply with recommended monitoring are almost certainly the most important prognostic factors governing a patient's survival. While inevitably there will be some discrepancies depending on the study you read, some of the important details and numbers I try to cover are as follows:

- Management requires twice daily insulin administration that can be logistically challenging, especially for boarding/vacation and diet modification that can also be costly (Niessen et al. 2010, Niessen et al. 2012).
- At-home glucose monitoring (either urine or blood) can be performed and may lead to better outcomes. Some clinicians will utilize continuous/interstitial glucose monitors as an easier way to monitor diabetic control (Del Baldo et al. 2020, Del Baldo et al. 2021, Re et al. 2023).
- Pets who have better adherence to recommended follow-up and treatment recommendations will have better short- and long-term outcomes.
- Cats can achieve diabetic remission while dogs cannot. Roughly 25–50% of cats can achieve remission, especially those with intensive glucose control/monitoring and appropriate diet (Gostelow et al. 2014). Unfortunately, roughly 30% of those cats will relapse and require insulin therapy (Gottlieb et al. 2015).
- Median survival time in cats is roughly 18–24 months (Callegari et al. 2013, Rothlin-Zachrisson et al. 2023), though you should always keep in mind age at time of diagnosis and comorbidities.
- Median survival time in dogs is slightly lower at 16–20 months with older dogs (over 10 years) and higher glucose levels doing worse (Heeley et al. 2020).
- Risk of urinary tract infection (UTI) (Yoon et al. 2020), pancreatitis (Davison 2015), and ocular disease, especially cataracts (Miller and Brines 2018).
- Can have complications such as ketosis, ketoacidosis, and hypoglycemia that can require hospitalization.

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DIAGNOSING KETOSIS

Knowing whether or not ketones are present is crucial to the management of a sick diabetic. Ketone bodies form due to an illness-driven surge of hormones such as cortisol, catecholamines, thyroid hormone, and growth hormone, combined with an inadequate level of insulin as shown in Figure 9.1 as the body searches for energy sources.

Since the body's mechanism for breaking down ketones is insulin-mediated, diabetic patients with ketosis require much higher doses of insulin compared to nonketotic patients. Diabetic patients WITHOUT ketosis likely do not require anything more than their normal insulin dose in hospital. For these patients, they may just need rehydration, gastrointestinal medications +/− antibiotics to get them feeling well enough to eat and restart their normal insulin protocol.

Ketones can be measured both in the blood and urine, with pros and cons to each approach. The use of a urine dipstick (either on blood or urine) is an available and straightforward test for detecting acetoacetate, which is one of the major ketones the body produces. In cats, using a dipstick with plasma is considered superior to urine (Zeugswetter and Pagitz 2009). Unfortunately, despite its ease of use, there are two significant drawbacks to dipstick testing. The first is that it is semiquantitative, relying on a color change to produce an interpretable result (Figure 9.2), limiting its utility to serially track changes in ketone levels with treatment (Chong and Reinecke 2016).

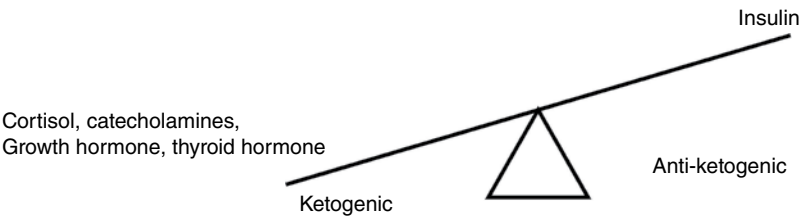


FIGURE 9.1 Increased "stress" hormones in the presence of inadequate insulin trigger ketogenesis. Figure courtesy of the author.



FIGURE 9.2 Semiquantitative analysis of urine/blood ketones using a urine dipstick. *Source*: Chong et al. (2016)/with permission of Elsevier.

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The second is that it doesn't measure 3-hydroxybutyrate, which is the first ketone produced in DKA, which limits its sensitivity (Di Tommaso et al. 2009). Using plasma rather than urine will improve the sensitivity of this test (Di Tommaso et al. 2009, Stojanovic and Ihle 2011, Zeugswetter and Rebuzzi 2012).

To offset some of the drawbacks of a urine dipstick, there are point of care ketometers that will measure 3-hydroxybutyrate. In addition to allowing serial quantitative measurements, these devices are also more sensitive and specific for diagnosing ketosis compared to dipsticks (Di Tommaso et al. 2009, Zeugswetter and Pagitz 2009). Current levels considered diagnostic for ketonemia are greater than or equal to 4.3 mmol/l in dogs (Di Tommaso et al. 2009) and 2.4 mmol/l in cats (Weingart et al. 2012).

DIFFERENTIATING DK AND DKA

Having diagnosed the sick diabetic with ketosis, the next important differentiation to make is whether or not they have acidemia (i.e. DKA) or not (i.e. DK), as patients with DKA have a worse prognosis than those with DK (Weingart et al. 2012). Diagnosing a metabolic acidemia requires the use of a venous blood gas that demonstrates an abnormal base excess (or low bicarbonate level if you subscribe to "traditional" acid–base theory). However, I think it is important to note the limitations of traditional acid–base theory in underdiagnosing DKA in many patients (Cuddy et al. 2025). The presence of a normal pH does NOT rule out DKA because there can be compensatory changes and/or concurrent acid–base disorders that may make the pH inappropriately normal (Duarte et al. 2012, Cuddy et al. 2025).

ASSESSING FOR COMORBID CONDITIONS

There is usually an inciting disease that triggers the hormonal milieu that leads to DK/DKA. It's important to assess for these concurrent diseases to make sure that you are treating both the "primary" disease as well as the DK/DKA. Common comorbidities in dogs include pancreatitis, UTI, and hyperadrenocorticism (Hume et al. 2006), while hepatic lipidosis and pancreatitis were most commonly seen in cats (Bruskiewicz et al. 1997). Therefore, in these cases it is reasonable to consider testing including abdominal ultrasound and urine culture in addition to the standard laboratory workup.

PROGNOSTIC FACTORS AND CLIENT COMMUNICATION

Many pets, up to 65% of dogs in one study (Hume et al. 2006), presenting with DK/DKA are newly diagnosed diabetics, and so it is important to go through the same discussion about long-term diabetic management outlined above. However, this patient population is also sicker and requires significant time and financial commitment, so it is also important to go through survival and prognostic factors as well.

- Survival to discharge in dogs is roughly 70% requiring on average 6 days of hospitalization. Negative prognostic factors included higher APPLE score, higher ketone level, ionized/total hypocalcemia, anemia, and worse acidemia (Hume et al. 2006, Edwards and Hess 2025).
- Survival to discharge in cats is roughly 50% with a similar hospitalization time to dogs. Negative prognostic factors included: higher blood glucose, azotemia, hyperbilirubinemia, and worse APPLE/APPLEfast score, which is an illness severity scale (Ad and Hess 2024, Cooper et al. 2015).
- Recurrence rate for DK/DKA is around 18–42% cats (Ad and Hess 2024, Cooper et al. 2015) while in dogs it is likely a bit lower at around 17% (Hume et al. 2006).

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TREATMENT DECISIONS AND MONITORING FOR DKA/DK

There are three main components for managing pets with diabetic keto(acido)sis: fluid therapy, insulin therapy, and adjunctive/patient-specific therapy.

There are few subjects that spark more debate in emergency and critical care medicine than fluid selection. Traditionally, 0.9% saline has been recommended (especially in humans) due to concerns about osmotic shifts and cerebral edema as blood glucose levels normalize. On the other hand, saline is also an acidifying fluid and has been postulated to increase the risk of renal injury. There are no veterinary studies that have directly compared fluid selection and outcome in DKA (Marella 2022) though expert opinion recommends a balanced crystalloid in most cases (Thomovsky 2017). In all likelihood, it is not the choice of fluid, but rather close patient monitoring of electrolytes and glucose that dictates outcome. My own personal fluid selection is Lactated Ringers' solution for these patients, though I think Plasmalyte-A and Normosol-R are also reasonable. Even in human medicine, there is a high likelihood they will transition away from saline, as in some cases it has led to faster resolution of the DKA state (Catahay et al. 2022). There is an ongoing clinical trial (Ramanan et al. 2025) designed to answer this question.

Beyond fluid choice, it is important to promptly begin correcting the common electrolyte deficiencies (hypokalemia, hypophosphatemia, hypomagnesemia) seen with DKA patients, as the addition of insulin therapy will make these worse, potentially to critical levels. Protocols for electrolyte correction in dogs are shown in Table 9.1 while a feline protocol is shown in Table 9.2. Again, what is likely more important than your initial electrolyte supplementation strategy is monitoring for changes and appropriately responding. Generally, until levels are stabilized, you should be monitoring electrolytes every 4–6 hours and a magnesium/phosphate (if not included on that panel) every 24 hours. Other monitoring considerations are reevaluating

Table 9.1 Initial electrolyte correction protocols for canine patients with diabetic ketoacidosis.

Blood glucose concentration (mg/dl)	Fluid composition[1]	Rate of administration (ml/h)
>250	0.9% NaCl	10
200–250	0.45% NaCl + 2.5% dextrose	7
150–199	0.45% NaCl + 2.5% dextrose	5
100–149	0.45% NaCl + 5% dextrose	5
<100	0.45% NaCl + 5% dextrose	Stop insulin infusion
Serum potassium concentration (mmol/l)	**Potassium (mEq/l, KCl) added to 250 ml bag of 0.9% NaCl[1]**	—
<2.0	20	—
2.0–2.5	15	—
2.6–2.9	10	—
3.0–3.5	7	—
3.6–5.0	5	—
Serum phosphate concentration	**Supplementation using potassium (4.4 mEq/ml) phosphate (3 mmol/ml)[2]**	—
<2.8 mg/dl	0.03 mmol/kg/h	—
Ionized magnesium concentration	**Supplementation rate**	—
<0.25 mmol/l	0.01 mEq/kg/h	—

1 Ensure not to exceed 0.5 mEq/kg/hr with the initial fluid rate.

2 Subtract any potassium contribution from overall potassium supplementation.

Source: Table courtesy of the author with data adapted from Sears et al. (2012) and Macintire (1993).

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Table 9.2 Initial electrolyte correction protocols for feline patients with diabetic ketoacidosis.

Blood glucose concentration (mg/dl)	Fluid composition[1]	Rate of administration (ml/h)
>250	0.9% NaCl	10
200–250	0.9% NaCl + 2.5% dextrose	7
150–199	0.9% NaCl + 2.5% dextrose	5
100–149	0.9% NaCl + 5% dextrose	5
<100	0.9% NaCl + 5% dextrose	Stop insulin infusion
Serum potassium concentration (mmol/l)	**Potassium (mEq/l, KCl) added to 250 ml bag of 0.9% NaCl[1]**	—
<2.0	80	—
2.0–2.4	60	—
2.5–2.9	40	—
3.0–3.4	30	—
3.5–5.0	20	—
Serum phosphate concentration	**Supplementation using potassium (4.4 mEq/ml) phosphate (3 mmol/ml)[2]**	—
2.0–2.9 mg/dl	0.06 mmol/kg/h	—
<2.0 mg/dl	0.12 mmol/kg/h	—
Serum magnesium concentration	**Supplementation using magnesium sulfate (500 mg/ml)**	—
1.6–1.9 mmol/l	0.5 mEq/kg/d	—
<1.6 mmol/ml	1 mEq/kg/d	—

1 Ensure not to exceed 0.5 mEq/kg/hr with initial fluid rate.
2 Subtract any potassium contribution from overall potassium supplementation.
Source: Table courtesy of the author with data adapted from Anderson et al. (2019).

acid–base status using a venous blood gas (usually concurrent with your electrolyte checks) and evaluating serial ketone levels.

INSULIN THERAPY FOR DK/DKA

The questions to consider with insulin therapy are timing, what kind of insulin to use, and which route of administration. Traditionally, delayed insulin therapy after 4–6 hours of rehydration has been recommended for two reasons. The first is that fluid therapy alone will decrease the blood glucose, which may increase the risks of excessive osmotic fluid shifts with early insulin therapy. The second is that early insulin therapy may exacerbate any preexisting electrolyte and acid–base abnormalities that haven't been corrected via fluid therapy (Gal and Odunayo 2023). While my own clinical practice is to initiate insulin therapy after 4–6 hours of fluid therapy, the early administration of insulin (less than 6 hours) has been shown to lead to faster resolution of ketosis with no increase in complications (DiFazio and Fletcher 2016) and may be reasonable in certain patient populations.

The first insulin protocols developed for management of DKA involved the use of short-acting "regular" insulin (Chastain and Nichols 1981, Macintire 1993) given either intramuscularly or intravenously as shown in Table 9.3, which remained the standard for some time.

Interestingly, comparison of a "fixed rate" approach designed to increase the amount of insulin administered versus the sliding scale insulin protocol above showed no difference in outcome (Gant et al. 2024). However, in more recent years, protocols have been developed using more

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Table 9.3 Sliding scale insulin continuous rate infusion for diabetic ketoacidosis in dogs and cats.

Blood glucose concentration (mg/dl)	Fluid composition[1]	Rate of administration (ml/h)
>250	0.9% NaCl	10
200–250	0.9% NaCl + 2.5% dextrose	7
150–199	0.9% NaCl + 2.5% dextrose	5
100–149	0.9% NaCl + 5% dextrose	5
<100	0.9% NaCl + 5% dextrose	Stop insulin infusion

1 Add 2.2 U/kg regular insulin to 250 ml of 0.9% NaCl and run first 50 ml through the administration set.
Source: Hoehne (2023)/with permission of Elsevier.

potent/rapid-acting forms of regular insulin known as "lispro" and "aspart" insulin (Walsh et al. 2016). When compared head-to-head with regular insulin, these protocols have been shown to have faster glycemic control (Anderson et al. 2019) and faster resolution of ketoacidosis (Sears et al. 2012) in dogs, with no difference in cats (Malerba et al. 2019). If your hospital has access to these insulins, then they are reasonable, but I have not personally used either. In cats, there are also protocols involving the use of longer acting glargine that have been shown to lead to faster improvements in biochemical parameters and short hospitalizations (Zeugswetter et al. 2021, Gallagher et al. 2015) compared to a continuous rate infusion of regular insulin. In cats who aren't responding appropriately, I have used a combination of an insulin CRI and intermittent long acting glargine insulin.

Finally, there is the question of intramuscular versus intravenous insulin administration. Given the variability of intramuscular absorption of regular insulin, I have always shied away from intramuscular protocols. However, with the development of newer insulin analogs (i.e. lispro), intramuscular administration may lead to similar outcomes as with intravenous regular insulin infusions (Malerba et al. 2020a).

Insulin therapy should be titrated based on blood glucose levels, typically about every 2 hours. While the use of a continuous glucose monitor (i.e. Freestyle Libre) has been evaluated, the diagnostic accuracy has been insufficient to justify clinical use (Malerba et al. 2020b, Eiermann et al. 2024)

TREATMENT SUMMARY FOR MANAGEMENT OF DK/DKA

I'll provide you with a summary of my diagnostic (Figure 9.3) and DKA treatment (Figure 9.4) protocols for the initial management of sick diabetic animals with DK/DKA, acknowledging there are several areas where alternatives exist.

EVALUATING AND TREATING THE SICK DIABETIC CAT ON AN SGLT-2 INHIBITOR

The challenge in evaluating cats on SGLT-2 inhibitors is that they may not have the classic hyperglycemia despite being affected with ketoacidosis, or what is known as "euglycemic diabetic ketoacidosis." Therefore, any "sick" diabetic cat on one of these medications should automatically have a ketone level checked and a venous blood gas as discussed above. Furthermore, in these cats with ketosis/ketoacidosis, they STILL need insulin despite a normal glucose level, and so you will have to provide parenteral dextrose as part of your treatment protocol.

DIAGNOSING AND TREATING HHS

It is always important to consider this diagnosis in the sick diabetic and know how to differentiate it from DKA because the treatment and prognosis will vary tremendously. The pathophysiology of

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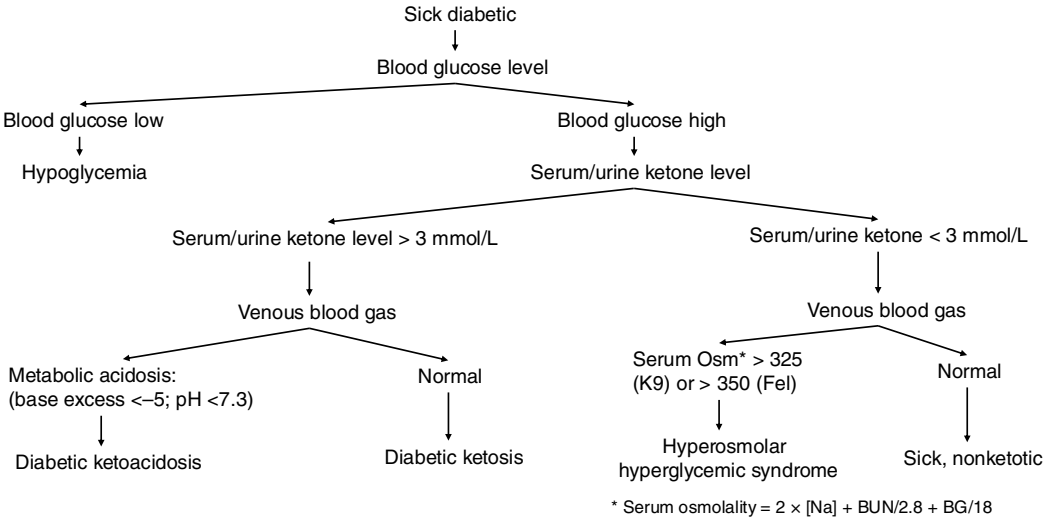


FIGURE 9.3 Diagnostic flowchart for common "sick" diabetic presentations. Figure courtesy of the author.

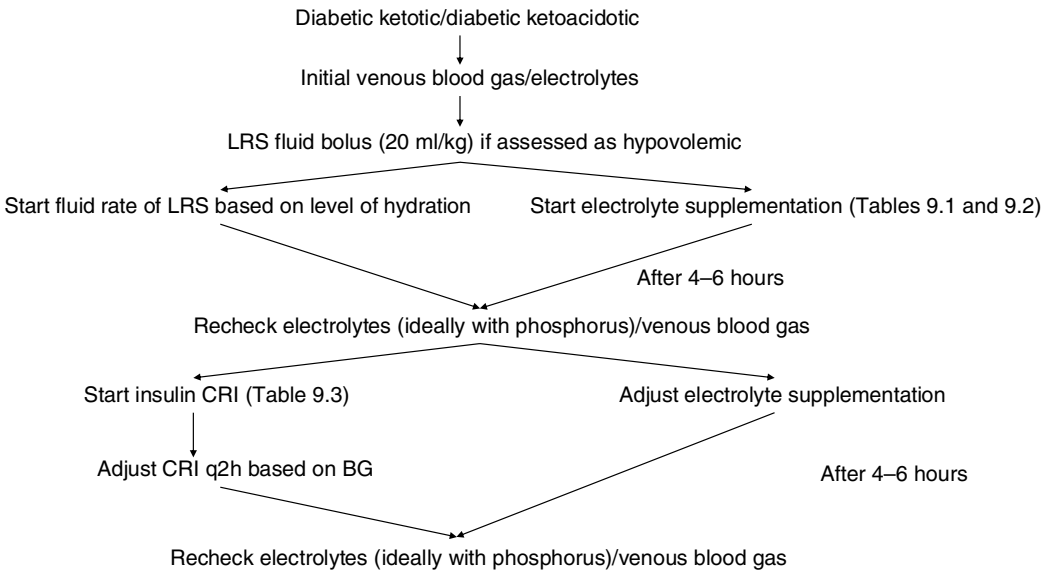


FIGURE 9.4 The author's initial DKA/DKA treatment and monitoring protocol. Figure courtesy of the author.

HHS is also different from DKA in that there is usually sufficient insulin/insulin responsiveness to prevent ketogenesis, but ongoing renal fluid losses due to hyperglycemia combine with inadequate intake to trigger dehydration and subsequent decreased sodium/glucose excretion, leading to hyperosmolarity (Figure 9.5).

The warning signs to consider HHS on your normal lab work are an excessively high glucose level (commonly too high to read on a glucometer) and azotemia (Koenig et al. 2004). If these are present, then you should also calculate a serum osmolality based on their glucose, BUN, and sodium values (see Figure 9.3 for equation). Classically, the definition of HHS in veterinary

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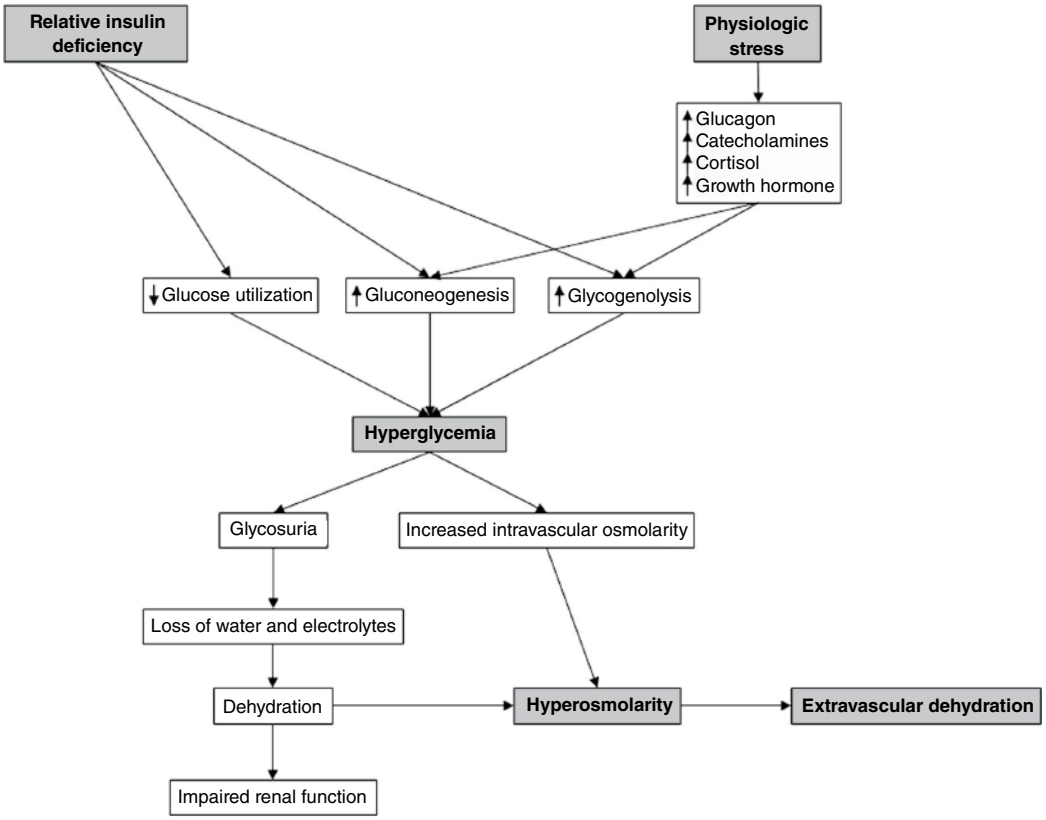


FIGURE 9.5 Pathophysiology for hyperosmolar/hyperglycemic syndrome (HHS). *Source*: Zeitler et al. (2011)/with permission of Elsevier.

medicine has been based on an absence of ketones combined with a glucose value >600 mg/dl and a serum osmolality higher than 325 mOsm in dogs and 350 mOsm in cats (Koenig 2023). However, this is not in line with current human definitions that allow small levels of urine ketones but the absence of "significant" ketonemia, which is usually defined as greater than or equal to 3 mmol/l or 2+ ketonuria (Pasquel and Umpierrez 2014, Mustafa et al. 2023). In my opinion, a patient who meets the other diagnostic criteria for HHS should be treated as such (at least initially), regardless of ketone level. Similar to DKA, it is important to assess for concurrent disease conditions, especially in cats, as they frequently will have clinically significant comorbidities such as congestive heart failure/cardiac disease, neoplasia, and kidney disease (Koenig et al. 2004).

The biggest difference between treating a patient with HHS and DKA is the emphasis on fluid and electrolyte, rather than glucose or ketone management. The concern with these patients is that too rapid a decrease in osmolality may trigger cerebral edema and neurologic complications. Since the largest contributor to osmolality in this patient population is sodium (Dugger et al. 2014), the treatment focus is more on appropriate fluid therapy and electrolyte monitoring rather than insulin therapy and glucose monitoring. You should assume all patients with HHS are severely dehydrated. In human pediatric patients, they estimate these patients are roughly 12–15% dehydrated (Zeitler et al. 2011), and 86% of dogs with HHS were assessed as moderately to severely dehydrated (Trotman et al. 2013). Fluid therapy should attempt to replace this over roughly 24–48 hours with serial glucose, electrolyte, and osmolality monitoring to ensure the **corrected sodium level** is not decreasing by more than 0.5 mEq/l/hr. If the amount of fluid required to correct that degree of dehydration will lower the sodium too quickly (you can calculate this using the modified

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Adrogue-Madias equation), then consider using a fluid with a higher sodium concentration, such as 0.9% saline.

Since fluid therapy and the correction of dehydration will inherently reduce the glucose level, and there are only minimal ketones present, insulin therapy can and should be delayed. Furthermore, when you start insulin, these patients do not require the same large doses of insulin as in DKA because you don't have reverse ketogenesis. There are no current veterinary protocols for the management of HHS, with all expert opinion recommendations extrapolated from human guidelines. These recommendations emphasize fluid resuscitation/electrolyte management with low-dose insulin therapy once the glucose level has stopped dropping by >50 mg/dl/hr (Koenig 2023). For a sample treatment protocol from human medicine, there is an excellent figure in Mustafa et al. (2023) with their treatment goals in Long et al. (2021).

While thankfully rare, HHS patients have a poor prognosis with a 66% mortality rate in cats (Koenig et al. 2004) and 38% in dogs (Trotman et al. 2013).

THE HYPOGLYCEMIC DIABETIC PATIENT

Hypoglycemia is seen more commonly in (obese) cats compared to dogs who have been receiving insulin for less than a year (Viebrock and Dennis 2018, Whitley et al. 1997). Common causes for hypoglycemia in the diabetic patient include owner error (overdose), vomiting after eating/insulin administration, lack of appetite, diabetic remission (cats only), and potentially excessive exercise (Viebrock and Dennis 2018, Whitley et al. 1997). Therefore, important historical questions include:

1. When was the last change to the pet's insulin dose? When was the last blood glucose curve or fructosamine level?
2. Are the owners appropriately administering insulin correctly (correct syringe, insulin type, dose)?
3. How has the pet been eating, and are any gastrointestinal signs present?

Thankfully, management of these patients is generally straightforward in terms of a dextrose bolus followed by continuous rate infusion. However, in extremis, you may need to consider glucagon in the persistently hypoglycemic pet (Park et al. 2023). Current recommendations to restart insulin include waiting at least 12 hours and until the glucose is greater than 250 mg/dl, patient is eating, and to decrease the dose by 25–50% in dogs and by 0.5–1 U in cats depending on the severity of the hypoglycemia (Behrend et al. 2018). Please note that the last recommendation is only for patients who were NOT inadvertently overdosed on insulin.

CONCLUSIONS

Diabetic emergencies can be complicated and challenging due to the multitude of ways they can present. However, if you are able to accurately categorize their illness (i.e. healthy, sick/nonketotic, DK/DKA, HHS, hypoglycemic), then you can at least start a reasonable diagnostic and treatment protocol.

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Trauma

CHAPTER 10

PART I: BLUNT TRAUMA

CRITICAL QUESTIONS

1. Is my patient stable or unstable?
2. Do they have evidence of traumatic brain injury, and should I consider CT?
3. Is there craniomaxillofacial trauma?
4. Do they have clinically significant chest trauma (pneumothorax, pulmonary contusions, rib fractures)?
5. Do they have peritoneal effusion, and what is it?
6. Are there any surgical fractures present?
7. Is there concern for a vertebral fracture?
8. What soft tissue injuries are present?

INTRODUCTION

The "hit by car" or blunt trauma patient causes a fair amount of anxiety in the newer clinician. In this chapter, we are going to review an anatomic systems-based approach to the traumatized patient with an emphasis on initial stabilization and diagnostics rather than definitive repair. The goal is to provide you with a framework and checklist for evaluating these patients using our "critical questions" to minimize the risk of missing an important injury. At the end of each section, we'll review the initial treatment and monitoring plan for that body system, along with some prognostic and client communication notes. The second portion of the chapter will go through an approach to penetrating trauma, mostly in the context of dog bite wounds.

ASSESSING CLINICAL STABILITY

The initial assessment of the trauma patient involves several components of the triage physical exam (Chapter 1) and doesn't really involve any diagnostic tests except for perhaps PCV/TS/BG/Lactate and POCUS. Briefly, it is important to assess for evidence of shock and any difficulty breathing. If clinical evidence of shock is present (pale mucous membranes, high heart rate, poor pulse quality, etc.) then initial stabilization with an IV fluid bolus is recommended. For pets with difficulty breathing, beyond providing oxygen, you should also consider whether or not to empirically perform thoracocentesis. In patients with severe dyspnea, I will always do this because the benefit of prompt evacuation for a pneumothorax greatly outweighs the small risk of iatrogenic lung injury. Finally, while you should ideally visualize a patient's motor function prior to giving pain medications (usually opioids), that can sometimes be challenging, and in certain situations it is reasonable to give a shorter acting medication (i.e. fentanyl) and then worry about neurologic status later. I've summarized the evaluation of shock and initial stabilization in Figure 10.1.

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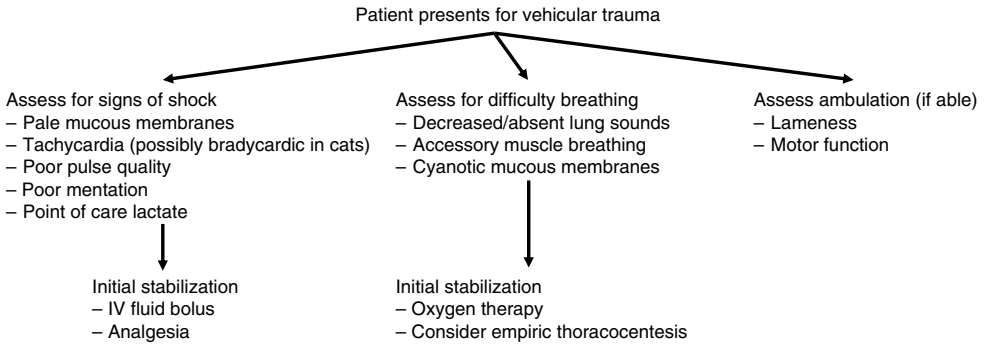


FIGURE 10.1 Initial evaluation (first 2 minutes) and stabilization options for the HBC patient. Figure courtesy of the author.

TRAUMATIC BRAIN INJURY

CRITICAL QUESTIONS

1. Is TBI present and how severe?
2. Would they benefit from a CT scan?
3. Should I give hyperosmolar fluids?
4. Should I start seizure prophylaxis?

With any traumatic injury, there are three main factors to consider. First, is an injury even present? Second, how bad is that injury? And third, what treatment options do I have to manage this injury?

Unfortunately, we have relatively crude mechanisms when assessing for the presence of traumatic brain injury (TBI). We can't ask our patients if they remember the accident or if they lost consciousness, which are helpful indicators in human medicine. While the modified Glasgow coma score (Table 10.1) is helpful in providing some signs to look for, the difference in scores between patients likely to survive and those at risk of dying is actually quite small at about 3–4 (Cameron et al. 2022). Similarly, while the animal trauma triage score provides some symptoms of neurologic dysfunction (Table 10.2), its prognostic utility for TBI (Sharma and Holowaychuk 2015) is mostly in its ability to account for other injuries in the polytrauma patient.

Furthermore, while the Cushings' reflex is one of the hallmark signs of severe TBI and is highly specific for brain herniation (Her et al. 2022), it is a later-stage disease indicator and its absence does not rule out a clinically significant brain injury. To summarize (in my opinion), existing scores (mGCS and ATT) are helpful at identifying patients with moderate to severe TBI that might benefit from more aggressive intervention, but are also more likely to die.

Ideally, advanced imaging would play a role in categorizing the severity of TBI. These modalities (especially CT) are capable of identifying additional injuries, such as depressed skull fractures or intracranial hematomas that might benefit from surgical intervention. In one study, 89% of patients evaluated with CT for head trauma had visible imaging changes (Chai et al. 2017) though the prognostic and treatment value of those changes has been debated (Wyatt et al. 2021, Rapoport et al. 2020). However, as patients with neurologic deficits are more likely to have depressed skull fractures (Amengual-Batle et al. 2020), any patient with a moderate TBI (mGCS 9-12) should be considered a candidate for CT if available. There is little to no role for emergent (or even necessarily delayed) MRI in head trauma patients. While it is good at identifying injuries (Yanai et al. 2015), its prognostic/therapeutic role is likely only in identifying patients at risk for posttraumatic seizures (Beltran et al. 2014).

While advanced imaging is very helpful in identifying patients who might benefit from neurosurgical intervention (i.e. decompressive craniectomy), the reality is that the vast majority of

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Table 10.1 The modified Glasgow coma score.

	Score
Motor activity	
Normal gait, normal spinal reflexes	6
Hemiparesis, tetraparesis, or decerebrate activity	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity with opisthotonus	2
Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1
Brain stem reflexes	
Normal pupillary light reflexes and oculocephalic reflexes	6
Slow pupillary light reflexes and normal to reduced oculocephalic reflexes	5
Bilateral unresponsive miosis with normal to reduced oculocephalic reflexes	4
Pinpoint pupils with reduced to absent oculocephalic reflexes	3
Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	2
Bilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	1
Level of consciousness	
Occasional periods of alertness and responsive to environment	6
Depression or delirium, capable of responding but response may be inappropriate	5
Semicomatose, responsive to visual stimuli	4
Semicomatose, responsive to auditory stimuli	3
Semicomatose, responsive only to repeated noxious stimuli	2
Comatose, unresponsive to repeated noxious stimuli	1

Source: Platt et al. (2001) with permission of John Wiley & Sons.

these patients neither require nor receive any intervention to that extreme. Therefore, when considering treatment options, we first have to consider the "bad things" that can happen with head trauma: decreased cerebral perfusion/increased intracranial pressure (ICP) and seizures.

As with any organ, the brain requires adequate blood flow (15% of cardiac output) to function, and without that perfusion, its tissue begins to die. Cerebral perfusion pressure (and by extension cerebral blood flow [CBF]) is determined by the opposing forces of mean arterial pressure and intracranial pressure. Intracranial pressure is conceptualized using the Monroe-Kellie doctrine (Figure 10.2), evaluating brain, blood, and cerebrospinal fluid volumes along with any trauma-induced swelling/hemorrhage.

Therefore, treatment goals are to maintain adequate mean arterial pressure through a combination of intravenous fluid therapy/vasopressors and minimizing intracranial pressure spikes via hyperosmolar therapy, as shown in Figure 10.3.

Current studies do not indicate a superiority of mannitol or hypertonic saline (HTS) for reducing intracranial pressure (Pigott and Rudloff 2021), though the effects of HTS may last longer than mannitol (Ballocco et al. 2019), and my own practice is to use HTS because it can also help simultaneously treat hypovolemia. Hyperosmolar therapy can be given up to every 6 hours as long as their osmolality and hydration are adequate, acknowledging that with time, the body will adjust and form idiogenic osmoles that may become problematic when withdrawing therapy (Wart et al. 2024). Other mediators of CBF include ventilation and oxygenation through changes in the cerebral vasomotor tone (Figure 10.4).

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Table 10.2 Animal trauma triage score. Both Tables 10.1 and 10.2 are used with permission under STM guidelines.

Grade	Perfusion	Cardiac	Respiratory	Eye/muscle/integument	Skeletal	Neurological
0	mm pink and moist CRT ~ 2 seconds Rectal temp 37.8 °C (100 °F) Femoral pulses strong or bounding	HR: Dog: 60–140 Cat: 120–200 Normal sinus rhythm	Regular resp rate with no stridor No abdominal component to resp	Abrasion, laceration: none or partial thickness Eye: no fluorescein uptake	Weight bearing in three or four limbs, no palpable fracture or joint laxity	Central: conscious, alert →sl dull; interest in surroundings Periph: normal spinal reflexes; purposeful movement and nociception in all limbs
1	mm hyperemic or pale pink; mm tacky CRT 0–2 seconds Rectal temp 37.8 °C (100 °F) Femoral pulses fair	HR: Dog: 141–180 Cat: 201–260 Normal sinus rhythm or VPCs <20/minute	Mildly incr resp rate and effort ± some abdominal component Mildly incr upper airway sounds	Abrasion, laceration: full thickness, no deep tissue involvement Eye: corneal laceration/ulcer, not perforated	Closed appendicular/rib fx or any mandibular fx Single joint laxity/luxation incl. sacroiliac joint Pelvic fx with unilateral intact lilium-acetab Single limb open/closed fx at or below carpus/tarsus	Central: conscious but dull, depressed, withdrawn Periph: abnormal spinal reflexes with purposeful movement and nociception intact in all four limbs
2	mm v pale pink and v tacky CRT 2–3 seconds Rectal temp <37.8 °C (100 °F) Detectable but poor femoral pulses	HR: Dog: >180 Cat: >260 Consistent arrhythmia	Mod incr resp effort with abdmon component, elbow abduction Moderately incr upper airway sounds	Abrasion, laceration: full thickness, deep tissue involvement, and arteries, nerves, muscles intact Eye: corneal perforation, punctured globe, or proptosis	Multiple grade 1 conditions (see above) Single long bone open fx above carpus/tarsus with cortical bone preserved Non-mandibular skull fx	Central unconscious but responds to noxious stimuli Periph: absent purposeful movement with intact nociception in two or more limbs or nociception absent only in one limb; decr anal and/or tail tone
3	mm gray, blue, or white CRT >3 seconds Rectal temp <37.8 °C (100 °F) Femoral pulse not detected	HR: Dog: <60 Cat: <120 Erratic arrhythmia	marked resp effort or gasping/agonal resp or irregularly timed effort Little or no detectable air passage	Penetration to thoracic/abd cavity Abrasion, laceration: full thickness, deep tissue involvement, and artery, nerve, or muscle compromised	Vertebral body fracture/luxation except coccygeal Multiple long bone open fx above carpus/tarsus Single long bone open fx above tarsus/carpus with loss of cortical bone	Central: nonresponsive to all stimuli; refractory seizures Periph: absent nociception in two or more limbs; absent tail or perianal nociception

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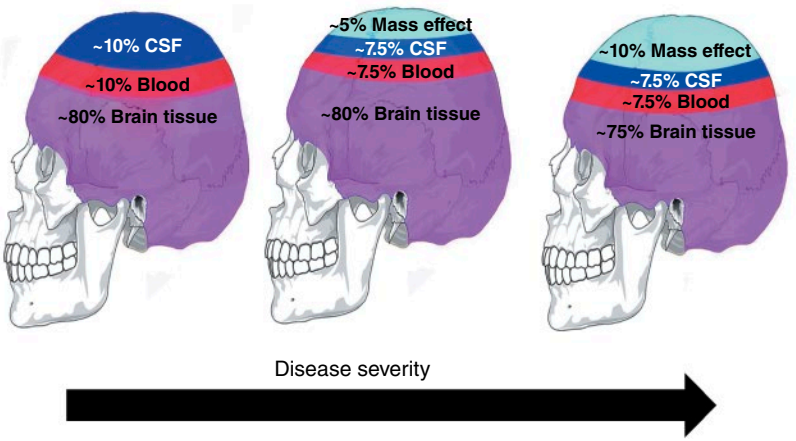


FIGURE 10.2 Monro-Kellie doctrine for increasingly severe brain injury. *Source*: Kalisvaart et al. (2020)/ Springer Nature/CC BY 4.0.

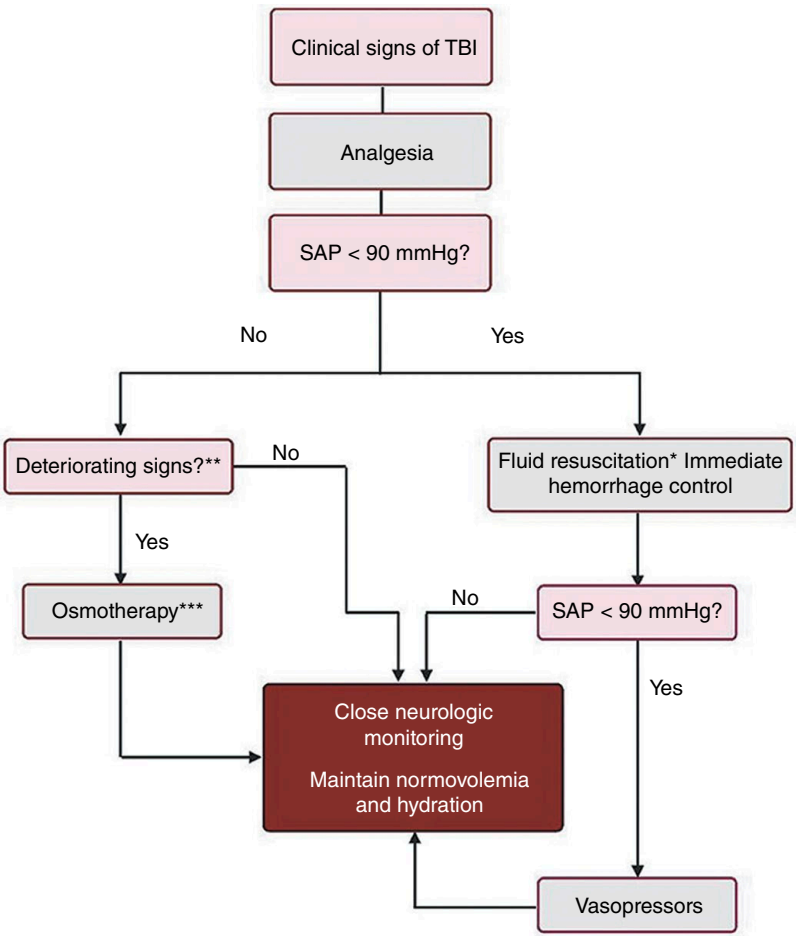


FIGURE 10.3 Treatment flowchart for patients with TBI. The author typically starts with 20 ml/kg of isotonic Crystalloid (*), Deteriorating signs characterized by worsened mGCS (**), Osmotherapy could consist of 1 g/kg mannitol IV up to three doses q60–90 minutes OR 3–4 ml/kg 7% hypertonic saline (***). *Source*: Pigott and Rudloff (2021)/Frontiers/CC BY 4.0.

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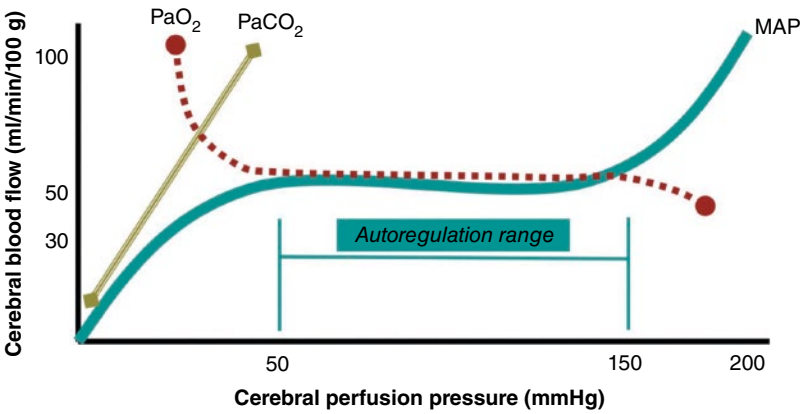


FIGURE 10.4 Cerebral blood flow (CBF) is dependent on ventilation ($PaCO_2$) and oxygenation (PaO_2) along with blood pressure (MAP). *Source*: Nikrouz et al. (2024) with permission of Cambridge University Press.

Current recommendations (though not evidence based) involve maintaining a $SpO_2 > 94\%$ and $PaCO_2$ between 35–40 mmHg (Wart et al. 2024) and limiting pressure on the jugular vein to allow appropriate venous drainage. Final recommendations to minimize ICP increase include judicious sedation/analgesia and in extreme circumstances, induced hypothermia (Hayes 2009).

Dogs with head trauma are at increased risk (roughly 3.5% in hospital, 6.8% long term) for developing seizures (Friedenberg et al. 2012), and there is a probable correlation between injury severity and risk of seizures (Steinmetz et al. 2013). While clearly if a patient HAS a seizure in-hospital, then antiepileptics should be initiated but there are no current recommendations or consensus for prophylactic anticonvulsants, and roughly 50% of surveyed practitioners indicated they routinely use them (Evans and Fernandez 2019). Even in human medicine, current recommendations for these medications are mixed (Thompson et al. 2015) though levetiracetam use is the most recommended agent in patients with severe TBI (Frontera et al. 2024). Unfortunately, dogs developing seizures within 7 days of injury had a worse prognosis (Steinmetz et al. 2013), and so that may be a reason to err on the side of medicating patients at higher risk (i.e. moderate to severe TBI).

SUMMARY OF DIAGNOSTIC/TREATMENT RECOMMENDATIONS AND CLIENT PROGNOSIS COMMUNICATION

- Scoring systems like the animal trauma triage score and modified Glasgow Coma Scale are helpful in identifying patients with moderate to severe TBI and have some prognostic value.
- Patients with moderate to severe TBI can be considered for a CT scan to evaluate for skull fractures and/or hemorrhage that may benefit from neurosurgical intervention.
- Patients with moderate TBI or worsening clinical signs should receive hyperosmolar therapy. HTS likely has more theoretical benefits than mannitol, but this has not been proven in veterinary medicine.
- Other recommendations include maintaining normoxemia, mild hypocapnia, normothermia, and appropriate sedation/analgesia to reduce ICP spikes and secondary brain injury.
- Antiepileptic medications are indicated for any patient who develops seizures and may be considered prophylactically in patients with moderate to severe TBI. Levetiracetam is likely the preferred agent in these cases.

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- Overall mortality for TBI patients is 15% in dogs and roughly 26–35% in cats (Cameron et al. 2022, Mann et al. 2022). Negative prognostic indicators include seizure development, worse mGCS (<8) and ATT (>9) (Platt et al. 2001, Sharma and Holowaychuk 2015), and certain CT parameters such as bleeding, specific skull fractures among others (Chai et al. 2017).

MAXILLOFACIAL TRAUMA

The challenge with maxillofacial trauma is that the majority of injuries involve multiple fractures and are best evaluated either on sedated exam or with CT, which is challenging in the new trauma patient (De Paolo et al. 2020a, b). Therefore, the diagnostic yield/benefit for evaluating these areas should be weighed against the risk of sedation and general anesthesia. We'll cover some of the most common skull fractures (Figure 10.5) and other injuries, including traumatic proptosis, to review some of the basics in their diagnosis and management.

The easiest method to visually diagnose maxillofacial injuries are usually based on visible malocclusion and include symphyseal fracture (Figure 10.6a), mandibular fracture (Figure 10.6b), and temporomandibular joint luxation/fracture (Figure 10.6c).

Since these injuries by themselves are rarely life-threatening, the goal of the emergency clinician for these injuries is rarely to provide definitive treatment but rather to catalogue them and communicate with the pet family about potential need for future CT and surgical correction (wire, plate, dental bonding, etc.). Given the complexity of the anatomy in this area, decisions around repair versus conservative management are outside the scope of this text (for a reasonable overview, you can consult Arzi 2023, Snyder and Lothamer 2022, or Glyde and Lidbetter 2003). In the emergent setting, appropriate analgesia +/− stabilization with a tape muzzle are reasonable approaches.

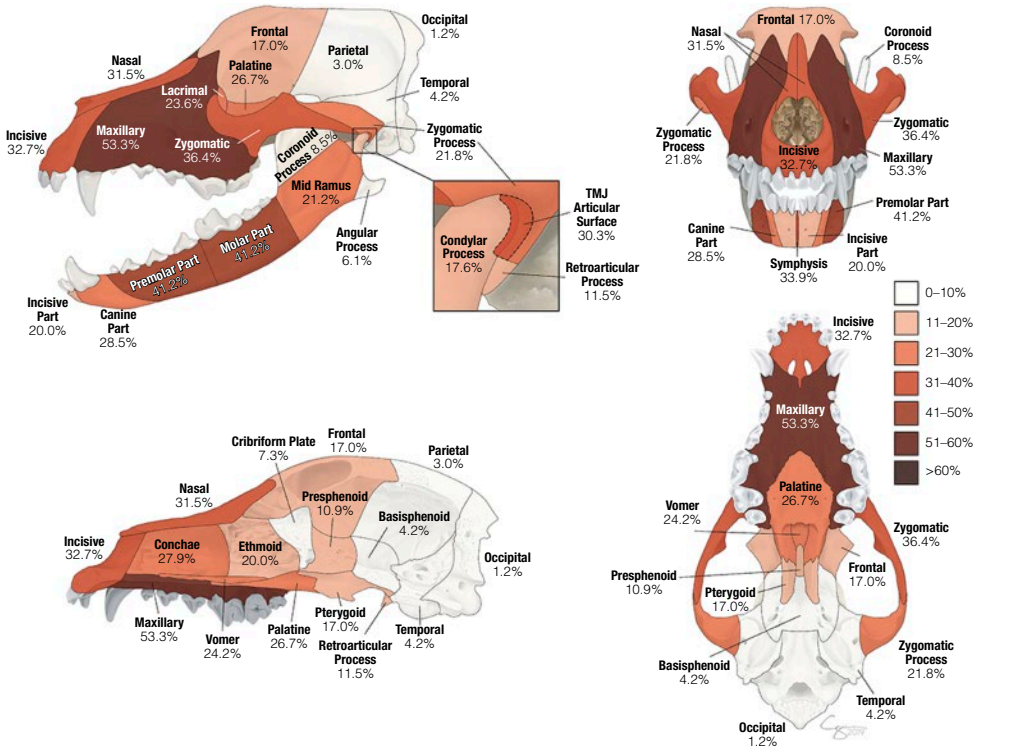


FIGURE 10.5 Commonly fractured skull bones due to blunt trauma. *Source*: De Paolo et al. (2020a)/Frontiers/CC BY 4.0.

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(a) (b) (c)

FIGURE 10.6 (a) Symphyseal fracture. *Source*: Zacher et al. (2013) with permission of Elsevier; (b) mandibular fracture. *Source*: Castejón-González et al. (2022) with permission of SAGE Publications; (c) temporomandibular luxation. *Source*: Jung et al. (2023)/Frontiers Media S.A./CC BY 4.0. Used with permission under STM (a/b) and Creative Commons License.

While traumatic proptosis is always a very "visible" injury, up to 17% of patients with proptosis will have concurrent other systemic injuries that may be more imminently life-threatening (Whinery et al. 2024). In the short term, simple lubrication and provision of an Elizabethan collar to prevent further self-trauma are most important. Evaluating the direct and indirect pupillary light reflex can be important in determining the potential for future vision in the eye (Pe'er et al. 2020). Once the pet is systemically stable enough for general anesthesia, decisions can be made about enucleation versus temporary tarsorrhaphy.

THORACIC TRAUMA

CRITICAL QUESTIONS

1. What imaging should I use to assess the thoracic cavity?
2. When should I perform thoracocentesis and/or put in a chest tube?
3. When should I employ high flow nasal oxygen?
4. Should I ever consider surgery for thoracic trauma?

INTRODUCTION

As we discussed under the head trauma section, the crucial areas for assessing a trauma patient are identifying the presence of an injury, evaluating its severity, and instituting appropriate therapy. For the thoracic cavity, the injuries we are most commonly concerned about would be pneumothorax, pulmonary contusions/hemothorax, rib fractures, and less commonly, diaphragmatic hernia. Identifying both the presence and severity of these injuries requires some form of diagnostic imaging. Combined with your clinical assessment of the patient's dyspnea, this will guide treatment interventions such as thoracocentesis, chest tube placement, oxygen supplementation, and in rare cases, surgery.

DIAGNOSING THORACIC TRAUMA

The three imaging modalities available for the thorax are radiographs, ultrasound, and CT. They all have their advantages and disadvantages in terms of their accessibility, cost/risk, and diagnostic benefit. The benefit of thoracic radiographs is that they provide a fairly good global view of the three main injury patterns (pneumothorax, rib fractures, and pulmonary contusions) while

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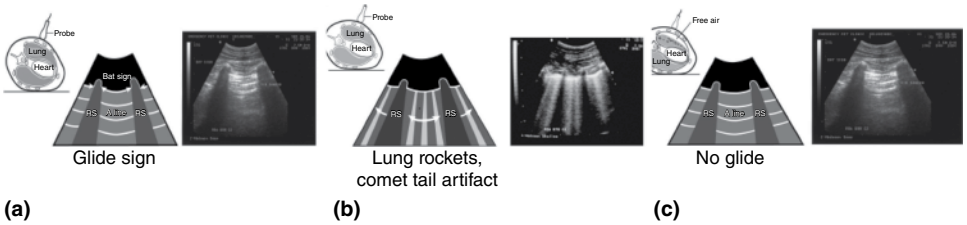


FIGURE 10.7 (a) Lung ultrasound images from normal lung, (b) pulmonary contusions, and (c) pneumothorax. *Source*: Lisciandro et al. (2008) with permission of John Wiley & Sons.

simultaneously allowing you to assess their severity. Disadvantages include the potential need for sedation and possible destabilization of the patient in the radiology suite (or during transport). Surprisingly, roughly 49% of dogs and 63% of cats will show some form of injury even in seemingly "stable" blunt trauma patients (Sigrist et al. 2004), so it is important to have a high index of suspicion for these injuries.

Ultrasound is attractive in that it rarely requires sedation and can be done without transport. Point of care lung ultrasound is capable of detecting pulmonary contusions, pneumothorax, hemothorax, and rib fractures as shown in Figure 10.7.

The disadvantage with ultrasound is its reliance on operator skill/interpretation with large differences in the sensitivity and specificity for its detection of pneumothorax depending on clinician experience (Lisciandro et al. 2008). Overall depending on the study cited, ultrasound is quite good at detecting pulmonary contusions, fairly good at detecting pleural effusion, and questionable for reliably detecting pneumothorax and rib fractures (Walters et al. 2018, Armenise et al. 2019, Lisciandro et al. 2008). In cats, ultrasound findings became more reliable the "sicker" they were (Vidal et al. 2024), which is the patient population we would be most interested in because they would benefit the most from therapeutic intervention.

Finally, thoracic CT has the distinct advantage of the highest "diagnosis" rate compared to radiographs and ultrasound (Walters et al. 2018, Dancer et al. 2019, Dicker et al. 2020). CT is also the most sensitive imaging modality for detecting thoracic vertebral fractures, though we'll discuss those in a later section. Its main disadvantages are need for reasonable sedation, logistics (operator availability, readout turnaround time), and cost.

And so, how do I approach imaging in these patients? After my triage exam as part of my secondary survey, I employ ultrasound to evaluate for the presence/absence of pulmonary contusions along with a rough assessment of their severity (i.e. uni/bilateral, isolated versus diffuse). For moderate to severely dyspneic patients, I will usually perform empiric thoracocentesis rather than imaging for the detection of a pneumothorax in an attempt to stabilize them for radiographs to evaluate for rib fractures or the odd diaphragmatic hernia patient. I do not employ emergent thoracic CT unless it is part of another study evaluating the spine.

MANAGING THORACIC TRAUMA

For patients with a pneumothorax, the decision to be made is whether or not to evacuate that air from the pleural space. While needle thoracocentesis is generally associated with low morbidity/mortality, the benefit of performing that procedure in a eupneic animal is likely minimal. Conversely, as I said above, for severely dyspneic patients, I will just empirically perform thoracocentesis because that is the single most important procedure I can do to stabilize that animal. There are no published criteria to guide your decision-making as to when to (or not to!) do this procedure. Similarly, there are no published guidelines to guide decision-making around chest tube placement, with some expert opinion around a "three strike" rule in traumatic pneumothorax

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(Rozanski 2010), which I think is fair for the majority of cases. Most dogs with a pneumothorax do not require chest tube placement, and my own anecdotal experience is that risk factors for placement are larger dogs (who are associated with large volume pneumothoraxes) and dogs with a very short (<1 hour) time interval in between initial thoracocenteses. While chest tube placement may seem daunting to the newer clinician, the small-bore wire-guided tubes are more than sufficient to manage almost all pneumothoraxes and have a reasonably low major complication rate (Boullhesen-Williams et al. 2022).

For patients with pulmonary contusions, oxygen therapy can help improve their tachypnea/dyspnea, but also remember the importance of adequate analgesia and sedation to reduce a patient's overall work of breathing. Unsurprisingly, animals with more severe injuries on initial chest X-ray were more likely to require oxygen and for longer periods of time (Powell et al. 1999). However, it is also important to have a fallback plan if simple oxygen supplementation is inadequate. High flow nasal oxygen is a reasonable choice (Keir et al. 2016), understanding that the higher flow can have consequences in terms of a pneumothorax and head trauma (Whitney and Keir 2023). Mechanical ventilation is always the therapy of last resort, understanding the high risk for either developing or worsening a pneumothorax (Campbell and King 2000). For patients requiring high flow oxygen therapy or mechanical ventilation, I would strongly consider having a chest tube(s) in place, especially in those with preexisting pneumothoraxes.

The majority of rib fractures are managed conservatively and no differently than the remainder of thoracic trauma. While occasionally surgical fixation can be required in the case of a flail chest, we'll discuss that further in the penetrating trauma chapter as bite wounds are a more common cause of this physiology (Olsen et al. 2002). Interestingly, the number and location of rib fractures (unlike in humans) do not have significant prognostic value, though the presence of concurrent injuries does (McCarthy et al. 2018). Surgical management for rib fractures (or any other blunt thoracic trauma) should be restricted to patients who have failed more conventional treatment and is thankfully uncommon (Simpson et al. 2009). The one exception to this would be a diaphragmatic hernia, but for the emergency clinician, current recommendations are not to pursue emergent surgery as there has been no survival benefit in faster surgery. Furthermore, patients with worse postoperative outcomes have all had additional injuries that would benefit from preoperative stabilization (Robertson 2021, Legallet et al. 2017, Gibson et al. 2005). For patients with a diaphragmatic hernia, additional treatments beyond your basic stabilization and thoracic trauma management might include keeping them at an incline to hopefully reduce the mass of herniated organs.

My personal flowchart for the initial workup and management of the most common thoracic injuries is shown in Figure 10.8. One important note is that while visualizing pulmonary contusions on ultrasound (B-lines) makes significant pneumothorax unlikely since a pneumothorax prevents visualization of the pulmonary parenchyma (Husain et al. 2012), the presence of a pneumothorax on ultrasound does not rule out contusions.

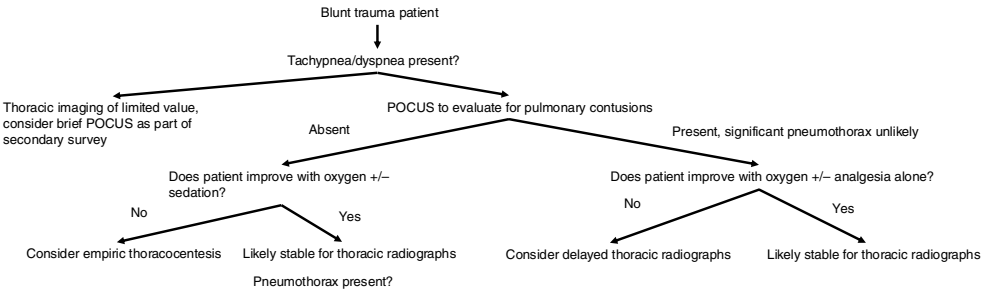


FIGURE 10.8 Initial diagnostic and stabilization approach to thoracic trauma. Courtesy of the author.

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ABDOMINAL TRAUMA

CRITICAL QUESTIONS

1. Is there effusion present (and what is it)?
2. When should I transfuse the traumatic hemoabdomen patient?
3. Is there a role for abdominal counterpressure and surgery?
4. Should I give antifibrinolytics?
5. Does my patient have risk factors for a uroabdomen and is one present?
6. Is there a prepubic tendon rupture?

INTRODUCTION

Between 25 and 45% of dogs presenting with trauma will have some form of cavitary effusion (Lisciandro et al. 2009, Armenise et al. 2019, Boysen et al. 2004). The critical diagnostic questions for these patients that have effusion are what is it and how severe is it. We'll discuss the diagnosis and management of the two most common clinical scenarios, traumatic hemoabdomen and traumatic uroabdomen. Finally, we'll discuss the relatively uncommon scenario of a prepubic tendon rupture.

TRAUMATIC HEMOABDOMEN

The presence of nonclotting hemorrhage (i.e. fluid with a similar or higher packed cell volume to peripheral blood) would be considered diagnostic for a hemoabdomen. In trauma cases, there can be a range in the subjective and quantitative volumes of blood present (Armenise et al. 2019, Lisciandro et al. 2009). However, focusing on the amount of visible effusion on ultrasound should not distract you from considering the patient's cardiovascular stability and shock status, as these factors are more important in guiding treatment than the volume of effusion present. As before, the shock factors to evaluate are mucous membrane color, capillary refill time, heart rate, pulse quality, packed cell volume, total solids, and lactate. These values can be used *in conjunction with* the amount of visible effusion to guide certain resuscitation decisions.

The first treatment decision (beyond the initial stabilization steps discussed at the beginning of the chapter) to consider in the traumatic hemoabdomen patient is whether or not to give a blood transfusion. While absolute recommendations are lacking in veterinary medicine, there are some fairly good guidelines and risk factors that can help make this decision. The first is to consider how much your first crystalloid bolus (and analgesia) helped stabilize the patient's shock status. If those treatments haven't significantly improved their shock indicators (see above), then I would strongly consider a transfusion in patients that I know are bleeding or have bled. There have been several studies evaluating predictors for transfusion in patients with internal hemorrhage and have shown that presence of any abdominal effusion (in cats), as well as a lower total protein and base excess were associated with increased need/risk for transfusion (Buseman et al. 2024, Stillion and Fletcher 2012, Langlois et al. 2025). One interesting (albeit small) study (Delgado et al. 2024) evaluated the use of a composite score called the Trauma Associated Severe Hemorrhage Score (Table 10.3) to determine the risk and need for transfusion.

You should use these predictors and risk factors as one tool in *considering* the need for transfusion, as not all of these patients will require or benefit from transfusion.

The next treatment decision is whether or not to apply abdominal counterpressure (i.e. a belly band) to these patients. While it has been shown to improve survival in experimentally induced hemorrhage (McAnulty and Smith 1986) and there are some theoretical benefits (Herold et al. 2008),

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Table 10.3 Determinants of the trauma-associated hemorrhage scores. Cumulative scores greater than 8 were associated with a higher risk of transfusion.

Variable	Value	Points
Hemoglobin (mg/dl)	<7	8
	<9	6
	<10	4
	<11	3
	<12	2
Base excess (mmol/l)	<−10	4
	<−6	3
	<−2	1
Systolic BP (mmHg)	<100	4
	<120	1
HR [/min]	>120	2
Free intraabdominal fluid		3
Extremities	Pelvic fracture	6
	Femoral fracture	3
Sex	Male	1

BP, blood pressure; HR, heart rate.
Source: Delgado et al. (2024) with permission of John Wiley & Sons.

there have been no trials in clinical patients that have demonstrated improved outcome. I do not routinely use this approach and would only consider it in an unstable patient (despite transfusion) in preparation for surgery. There is a limited role for surgery in the majority of traumatic hemoabdomen patients, with only a minority of cases requiring surgery (Mongil et al. 1995, Simpson et al. 2009).

The last major treatment decision to consider is whether or not to use antifibrinolytics such as aminocaproic acid or tranexamic acid (TXA). Original interest in the use of antifibrinolytics for trauma grew from a study that showed improved survival in hemorrhaging trauma patients with the use of TXA (CRASH 2 Investigators 2011). Without delving too much into the pathophysiology, the goal of these medications is to reduce the pathologic breakdown of clots (hyperfibrinolysis) to promote hemostasis. The challenge is that it is unclear how many veterinary trauma patients have hyperfibrinolysis with some studies saying none (Birkbeck et al. 2024) and some saying 9% (Herrero et al. 2021). Furthermore, identifying patients WITH hyperfibrinolysis requires viscoelastic testing, which is not universally available. The only clinical trial evaluating the use of an antifibrinolytic in patients with diagnosed traumatic hyperfibrinolysis showed no survival or transfusion benefit though the study was not sufficiently powered to detect small differences (Mays et al. 2025). A previous retrospective study had also not shown a transfusion reduction benefit in traumatic hemoabdomen patients who received TXA (Sigrist et al. 2022). And while adverse events associated with these medications are low (Kelley et al. 2022, Davis and Bracker 2016), I generally would only consider them in a patient with severe hemorrhagic shock secondary to trauma and ideally with evidence of hyperfibrinolysis on viscoelastic testing.

UROABDOMEN

While uroabdomen is an uncommon injury affecting roughly 1% of trauma patients (Godart et al. 2025), since it can cause life-threatening hyperkalemia and can require surgical repair, it is not a diagnosis to miss. The first question to consider is does my patient have risk factors for a uroabdomen such as concurrent pelvic fractures (Hoffberg et al. 2016) and pubic hernias (Rein et al. 2024). The presence of these injuries should raise your index of suspicion for a uroabdomen *especially* if there is peritoneal effusion. While there are a multitude of other very *specific* (meaning their presence is reasonably diagnostic) symptoms of uroabdomen, such as hyperkalemia, azotemia, hematuria, lack of urination, and a lack of palpable bladder, the *absence* of these signs does not rule out a potential uroabdomen (Stafford and Bartges 2013). This makes it fairly easy to miss the initial uroabdomen

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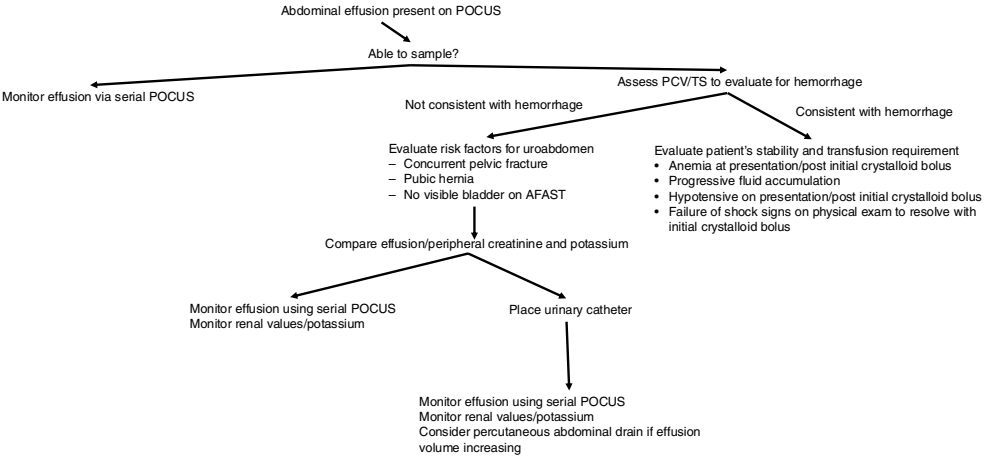


FIGURE 10.9 Initial diagnostic workup and stabilization considerations for patients with abdominal effusion. Courtesy of the author.

diagnosis, which is why it is important to monitor certain parameters such as lab work, urination (especially when on IV fluids), and ultrasonographic effusion level in high-risk patients.

For patients with a high initial index of suspicion for uroabdomen (and enough effusion for abdominocentesis), an elevated effusion potassium and creatinine compared to that of peripheral blood is diagnostic. For dogs, an effusion creatinine more than two times the peripheral and an effusion potassium more than 1.4 times the peripheral is considered diagnostic for a uroabdomen (Schmiedt et al. 2001). In cats, the ratios are 2 and 1.9 times, respectively (Aumann et al. 1998).

Initial management for the uroabdomen patient is reasonably straightforward: you have to divert the urine out of the abdomen. Besides standard stabilization with fluids and pain medications, placement of a urinary catheter will help prevent the majority of ongoing leakage from the bladder or urethra, but for patients with large volumes of effusion (or the rare patient with ureteral/renal damage), I will typically place a small-bore chest tube into the abdomen as a peritoneal drainage device (Crosby et al. 2023). With these treatments, a uroabdomen is NOT a surgical emergency, and so we will not discuss the pros and cons of surgical versus medical management for these cases here. I've provided a summary figure for the initial workup and treatment for traumatic abdominal effusions in Figure 10.9.

PREPUBIC HERNIA

This is an uncommon injury but one that is important again because of the need for potential future surgical intervention. As with a uroabdomen, you should make sure you've evaluated for this injury in patients with significant pelvic fracture as the majority of cases with a prepubic hernia will have concurrent orthopedic injuries (Rein et al. 2024). With this injury, there is avulsion of the prepubic tendon from the pubic bone that can lead to herniation of abdominal contents, including the urinary bladder and intestine (Figure 10.10), often appreciated as ventral abdominal swelling.

While this is an important injury to document as it will impact the need for future treatment, in most cases, there is no emergent (i.e. immediate) treatment required beyond the basic stabilization discussed above.

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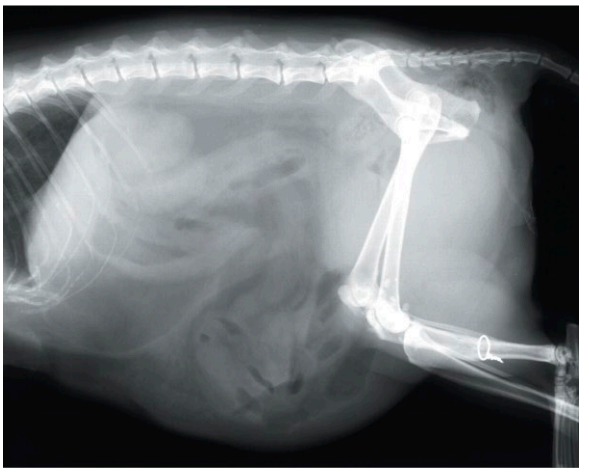


FIGURE 10.10 Lateral radiograph of a blunt trauma victim with a pubic hernia. *Source*: Beittenmiller et al. (2009) with permission of American Animal Hospital Association.

PELVIC FRACTURES

CRITICAL QUESTIONS

1. Is this a surgical fracture?
2. Is there any neurologic compromise?

INTRODUCTION

Pelvic fractures are the most commonly seen fracture pattern in blunt trauma affecting up to 28% of patients (Simpson et al. 2009). Broadly, they can be classified as affecting the ilial wing/body (Figure 10.11a), ischium (Figure 10.11b), pubis (Figure 10.11c), acetabulum (Figure 10.11d), sacro-iliac joint (Figure 10.11e), and sacrococcygeal joint with fractures of multiple bones commonly being present (Messmer and Montavon 2004). One important note is that pelvic fractures can lead to significant hemorrhage, and so consider blood loss in a patient with pelvic fractures if they are or become hemodynamically unstable.

CONCURRENT INJURIES AND NEUROLOGIC COMPROMISE

As we've discussed above, there are several concurrent injuries that can occur in patients with pelvic fractures. However, the location of certain pelvic fractures (especially sacro-iliac luxations and sacral/sacrococcygeal fractures) can lead to damage to the sciatic, pudendal, and coccygeal nerves. The highest risk fractures for neurologic deficits are those affecting the sacro-iliac and sacro-coccygeal joints, due to the close proximity of the sciatic and pudendal nerves, respectively (Lanz 2002). Since injuries to these nerves can cause significant quality of life impairments such as impacting ambulation and fecal/urinary continence, appropriate identification of any neurologic deficits is imperative in guiding surgical and owner decision-making.

Neurologic assessment of this area should focus on ambulation, withdrawal response (assessing the sciatic nerve), anal sphincter tone/perineal reflex (assessing the pudendal nerve), and tail sensory/motor function (Meeson and Corr 2011). The most commonly seen neuropathy associated with pelvic fractures is a sciatic neuropathy (Meeson and Geddes 2017) and is likely more common

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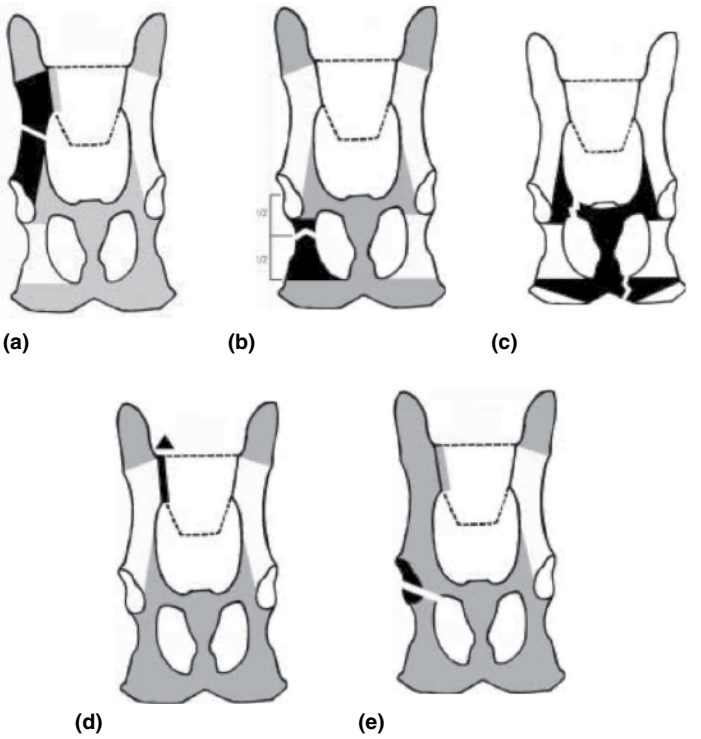


FIGURE 10.11 Configuration of common simple pelvic fractures including (a) ilial wing, (b) ischium, (c) pubis, (d) sacro-iliac joint, and (e) acetabulum. *Source*: Messmer and Montavon (2004) with permission of Georg Thieme Verlag KG.

Table 10.4 Exam findings for most common neurologic injuries secondary to pelvic fractures. Courtesy of the author.

Sciatic nerve injury	Pudendal nerve injury
Spontaneous knuckling	Decreased/absent perineal reflex
Decreased withdrawal reflex	Decreased/absent sphincter tone
Assess deep pain perception	Poor tail carriage
	Assess deep pain perception

in cats. While initial neurologic deficits on presentation are not always permanent, owners should be prepared that they may persist for weeks if not indefinitely. A summary of neurologic exam findings for these fractures is summarized in Table 10.4.

SURGICAL DECISION-MAKING

As with any procedure, the goal of pelvic fracture fixation is to improve function and outcome while minimizing the risk of complications. An in-depth discussion on the pros and cons of surgical versus conservative management of pelvic fractures is outside the scope of this chapter (See Miller 2002 or Grierson 2019 for a reasonable introduction) but is briefly summarized in Table 10.5.

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Table 10.5 Considerations for surgical versus conservative decision-making in pelvic fractures.

Surgical intervention may be required if:	Consider conservative management if:
• The fractures involve the weight-bearing axis (e.g. ilial fractures or sacroiliac luxations)	• The fractures involve non-weight-bearing segments
• The fractures involve the acetabulum	• The fractures are minimally displaced, non-articular fractures that are stable
• Multiple injuries exist (e.g. bilateral fractures, ipsilateral ilial fracture with hip luxation)	• The fractures involve the "pelvic floor" and do not significantly compromise the width of the pelvic canal
• Significant pelvic canal narrowing is seen on a ventrodorsal radiograph (i.e. greater than 50% reduction in pelvic canal width)	
• Refractory or severe pain is evident	
• Sciatic nerve deficits exist that may be associated with a fracture (e.g. of the ilium)	
• Failure of conservative management	

Source: Meeson and Corr (2011) with permission of Sage publication.

VERTEBRAL FRACTURES

CRITICAL QUESTIONS

1. Could/does this patient have a vertebral fracture?
2. Does this vertebral fracture require surgery?
3. What is the prognosis for this vertebral fracture?

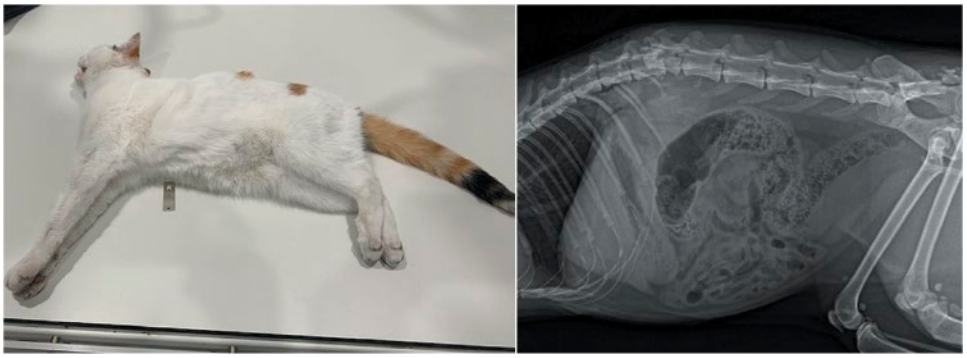
INTRODUCTION

Vertebral fractures in veterinary medicine are thankfully rare, affecting at most 10% of blunt trauma patients (Simpson et al. 2009), but can be devastating injuries when they do occur. Furthermore, their presentation, prognosis, and management are dependent on the location and severity of the injury. The most common locations for fracture were in the thoracolumbar and lumbosacral spine, specifically the T10-L2 and L7-S1 vertebral spaces (Bruce et al. 2008, Bali et al. 2009), which is where we will focus the majority of our attention in terms of diagnosis, treatment, and prognosis.

THORACOLUMBAR JUNCTION FRACTURES

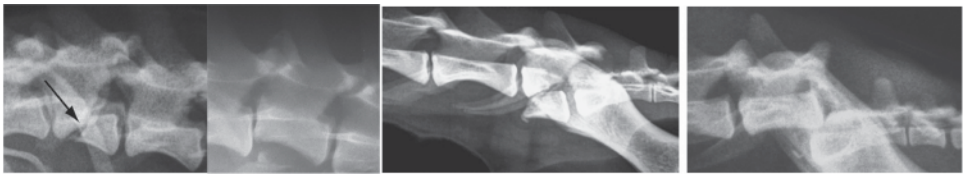
The most important aspect in diagnosing vertebral fractures is to have an appropriate index of suspicion so you can both avoid iatrogenically worsening the injury and order appropriate imaging studies. The challenge in recognizing these cases is that there are usually multiple confounding factors and disease processes, such as shock and pelvic fractures, that can confuse your neurologic exam. There are also phenomena, such as spinal shock, that can lead you to mislocalize a lesion (Jeffery 2010). Unfortunately, the best "warning signs" I can give you to raise your initial suspicion for vertebral trauma are a patient who is unable to walk (especially if they are not in shock or have been appropriately resuscitated), those exhibiting Schiff-Sherrington posture (Figure 10.12), and those with pain on spinal palpation. When present with abnormalities on a brief neurologic exam, these signs should prompt you to consider backboard immobilization and radiographs of the vertebral column.

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(a) (b)

FIGURE 10.12 (a) Schiff-Sherrington posture and (b) due to an L2 luxation. *Source*: Holland (2023) with permission of John Wiley & Sons.



(a) (b)

FIGURE 10.13 Typical fractures and luxations of (a) L1/L2 and (b) L7. *Source*: Jeffery (2010) with permission of Elsevier.

If, based on your exam findings, you have a reasonable index of suspicion for spinal cord trauma, the typical first imaging modality to reach for post stabilization is radiographs due to their relative ease of access and lack of general anesthetic requirement. It is important to acknowledge that while they have a reasonable specificity for vertebral fractures (Figure 10.13), the sensitivity can be limited (Kinns et al. 2006), and so for patients you still have concern for, a CT scan may be required. Furthermore, not all spinal cord trauma is due to a fracture as we'll discuss traumatic disc injuries in a later chapter.

Surgical decision-making is based on whether or not the fracture is considered "unstable" based on what is called the three-compartment theory (Figure 10.14) as stability is required for both bone and nerve healing. Fractures that are unstable (greater than two compartments compromised) would benefit from surgical intervention.

In addition, fractures causing compression of the spinal cord with neurologic deficits would also benefit from surgical reduction and fixation, just as you would consider for a disc injury. Patients with stable fractures, ideally demonstrated via CT (Figure 10.15) and mild neurologic deficits, can likely be managed without surgery (Orgonikova et al. 2021a/b).

Your neurologic exam will dictate the prognosis for patients with vertebral fractures managed surgically and medically. While there is an injury severity scale (Table 10.6) available, traditionally, the largest prognostic factor is whether or not deep pain sensation is intact.

Several studies have demonstrated a grave prognosis for dogs lacking deep pain (Olby et al. 2003, Gallucci et al. 2017) with no dogs requiring the *spontaneous* ability to walk. Interestingly, cats may actually do better than dogs (Grasmueck and Steffen 2004) though in both species, intensive physical therapy is required to have any chance of recovery usually via the spinal walking reflex. That being said, the most recent study evaluating surgical fixation of vertebral fractures showed even

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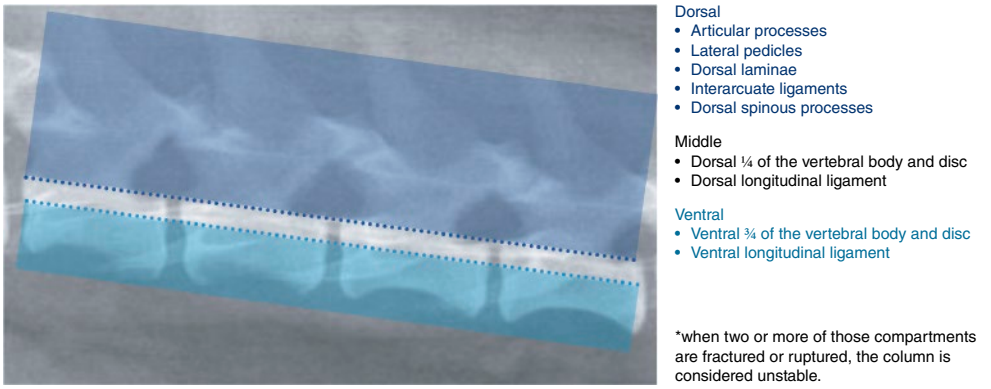


FIGURE 10.14 Three-compartment theory for determining the stability of vertebral fractures. *Source*: Orgonikova et al. (2021) with permission of MA Healthcare Ltd.



FIGURE 10.15 CT image of a stable T13 fracture. *Source*: Gallastegui et al. (2019) with permission of John Wiley & Sons.

Table 10.6 Neurologic injury severity scale.

Grade	Neurological status
0	No spinal hyperesthesia and no neurological deficits
1	Hyperesthesia, no neurological deficits
2	Proprioceptive deficits and/or ataxia
3	Nonambulatory with purposeful movement
4	No purposeful movement with deep pain sensation
5	Loss of deep pain sensation

Source: Ciou et al. (2024) with permission of Georg Thieme Verlag KG.

animals lacking deep pain may still have a chance at neurologic recovery (Ciou et al. 2024). From an emergency perspective, in patients with vertebral fractures, the presence/absence of deep pain should prompt an in-depth conversation with the owners about potential neurologic recovery, need for physical therapy, and regain of function.

LUMBOSACRAL FRACTURES

The majority of management and diagnosis for lumbosacral fractures is similar to those in the thoracolumbar spine. However, some of the findings on your neurologic exam will differ as

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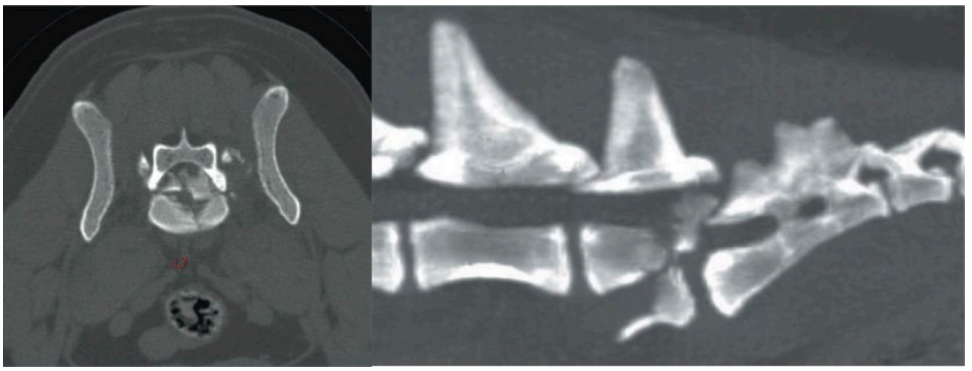


FIGURE 10.16 CT images of an unstable L7 fracture and luxation. *Source*: Shani et al. (2018) with permission of John Wiley & Sons.

damage is more common to the nerves of the cauda equina (sacral plexus and pudendal nerves) than the spinal cord itself (see Table 10.4). Common clinical signs for dogs include severe back pain, fecal/urinary incontinence, and paraparesis (Caterino et al. 2022). However, given its location, as we discussed above, it is crucial to assess for concurrent orthopedic injuries in these patients. Imaging (Figure 10.16) and surgical decision-making are similar to those discussed above.

Overall, the prognosis for reasonable return to function is better than in thoracolumbar injuries, though some dogs can have some residual sciatic nerve dysfunction (Segal et al. 2018, Di Dona et al. 2016) and so a positive withdrawal response may be a positive prognostic indicator as is the ability to flex/extend the tarsus (Dell'Apa et al. 2024).

APPENDICULAR SKELETON FRACTURES AND SOFT TISSUE INJURIES

CRITICAL QUESTIONS

1. Can/should I immobilize the fracture?
2. Is this a surgical fracture?
3. Is the limb salvageable?

This section will be brief because for appendicular skeletal fractures and soft tissue injuries, the role for the emergency clinician is mostly around fracture stabilization and wound lavage rather than definitive repair. However, you should be familiar with some basic concepts of fracture repair to appropriately prepare an owner for the definitive treatment. Furthermore, for patients with fractures below the knee/elbow, you *should* try to immobilize the limb for pain control and to minimize further soft tissue damage. Fractures that may be amenable to external coaptation are simple, incomplete, minimally displaced fractures or those that have an internal strut, such as a single ulnar or fibular fracture (Roush 2005). Most other fractures should be considered for surgical fixation, and it is never wrong to consult a surgeon about what the best approach would be in their hands.

While many blunt trauma patients will have concurrent soft tissue injuries, they can range in severity from relatively minor abrasions/"road rash" up to a complete degloving injury. Initial stabilization for these patients is mostly around clipping, cleaning, and lavaging the wounds to reduce contamination while protecting them with bandages to prevent ongoing damage and contamination as well as hospital-acquired infections. Depending on the degree of soft tissue damage and concurrent orthopedic injury, you should also consider whether or not the limb is

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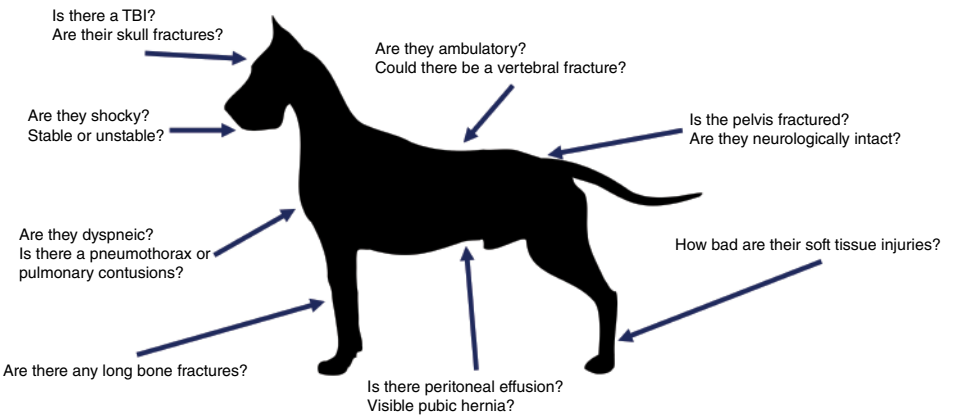


FIGURE 10.17 Initial assessment and injuries to consider for a blunt trauma patient. Courtesy of the author.

salvageable. Factors to consider when contemplating amputation are degree of nervous/vascular injury, presence of a severely comminuted fracture, and owner's finances (Ness 2002) though this is almost never pursued in the first 24 hours post trauma.

BLUNT TRAUMA SUMMARY AND CLIENT COMMUNICATION

Figure 10.17 is aimed at summarizing the system-based approach to the blunt trauma victim we've discussed above.

It's also important to review some prognostic factors that should be communicated to owners. My own client communication in these cases follows the system-based approach outlined above, so I can review the possible injuries a patient can sustain and the recommended diagnostic tests. Depending on the injuries I think are present, I then discuss the initial stabilization measures and treatments for these patients, including fluid therapy, analgesics +/− oxygen with definitive management depending on the injuries cataloged from imaging and physical exam. Overall survival rate for blunt trauma in dogs is 89.5% with negative prognostic indicators including higher shock index, higher ATT, lower mGCS, higher lactate, and lower pH (Kraenzlin et al. 2020, Poirier et al. 2023, Hall et al. 2014, Davros et al. 2023). In cats, survival is slightly worse at around 80% (Gregory et al. 2023) with negative prognostic factors including evidence of multiple organ dysfunction, hypothermia, and higher injury severity scores such as ATT and APPLE (Lapsley et al. 2019, Murgia et al. 2019, Fitzgerald et al. 2022).

PART II: PENETRATING TRAUMA/BITE WOUNDS

CRITICAL QUESTIONS

1. Does this wound penetrate the chest/abdomen?
2. How do I stabilize a penetrating thoracic wound?
3. Is this a "complicated" bite wound?

INTRODUCTION

While the majority of penetrating trauma are bite wounds (Davros et al. 2023, Gregory et al. 2023) that are relatively uncomplicated, this category can also include more complex (though less common) injuries such as gunshot and impalement wounds. Recognizing both perforating and "complicated"

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FIGURE 10.18 Ventrodorsal radiograph of a penetrating chest wound showing subcutaneous emphysema, multiple rib fractures (pseudo flail chest), a pneumothorax, and pulmonary contusions. *Source*: Scheepens et al. (2006)/John Wiley & Sons.

bite wounds is important to guide decision-making about the need for specialist-level surgical management and the potential for wound healing complications (Reviewed in Risselada 2017).

RECOGNIZING THE PENETRATING THORACIC WOUND

Certain injuries, such as impalement, are fairly easy to identify as high risk for penetration of the thoracic cavity. However, penetrating bite wounds can occasionally be difficult to recognize, and so evaluating your patient for risk factors of perforation is critical, and thoracic radiographs are strongly recommended for any patient you have concern for. Risk factors for a penetrating thoracic bite wound in dogs include dyspnea, pleural effusion, small breed (<10 kg) dogs, pneumothorax, rib fracture, and pseudo flail chest as shown in Figure 10.18 (Scheepens et al. 2006, Frykfors von Hekkel and Halfacree 2020).

Risk factors in cats are similar to dogs though most cats with penetration had greater than or equal to three radiographic lesions (Frykfors von Hekkel et al. 2020).

For other mechanisms of injury such as an impalement or gunshot wound, the bias should always be toward proving that thoracotomy is NOT needed rather than that it IS. For impalement injuries, CT can be a valuable tool in guiding both the need for surgery and the procedure that would be required (Matiasovic et al. 2018) but should not be an impediment to emergency surgery if unsafe or unavailable. While there are several descriptive studies of gunshot wounds in veterinary medicine (Capak et al. 2016, Baker et al. 2013, Olsen et al. 2014, Fullington and Otto 1997) there are no current recommendations or risk factors for the need for surgery. In human medicine, current recommendations for urgent thoracotomy involves the presence of significant (and not improving) hemothorax and pneumothorax (Caputo et al. 2024). Given the significant differences in trauma management/monitoring (i.e. most human patients have chest tubes) between human and veterinary medicine, any gunshot wound bad enough to cause a pneumothorax or hemothorax should likely have an exploratory surgery of some sort.

STABILIZING THE PENETRATING THORACIC CHEST WOUND

Besides your typical stabilizing measures of IV fluids, antibiotics, analgesics, and oxygen therapy; the two biggest concerns with a penetrating chest wound are shock due to hemorrhage and respiratory compromise due to a (tension) pneumothorax. We discussed the use of transfusions to manage

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hemorrhagic shock above as well as the use of thoracocentesis for pneumothorax. However, penetrating chest wounds have the potential to cause a tension pneumothorax where air is continuously entering the thoracic cavity via a one-way valve with no ability to exit (Figure 10.19).

In these patients, thoracocentesis may be insufficient, and sometimes you need to intubate and anesthetize the patient to perform a mini or "finger" thoracostomy (Figure 10.20) to change the one-way valve into an open system where air can continuously escape. As we talked about above, patients that require these interventions also require operative management.

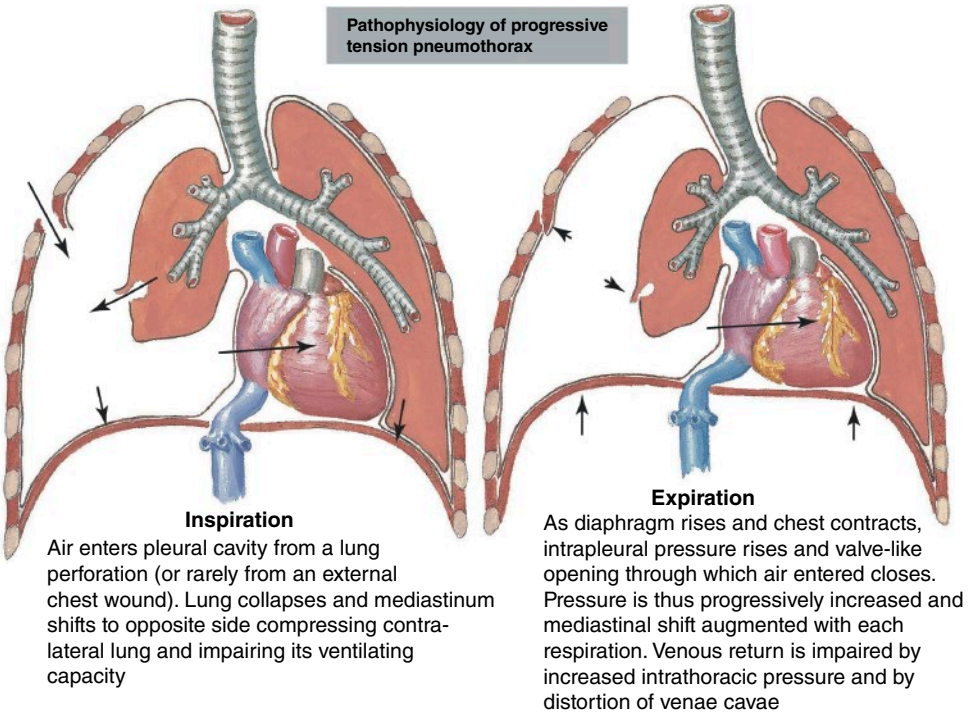


FIGURE 10.19 Pathophysiology of progressive tension pneumothorax. From Netter's images, used with permission.

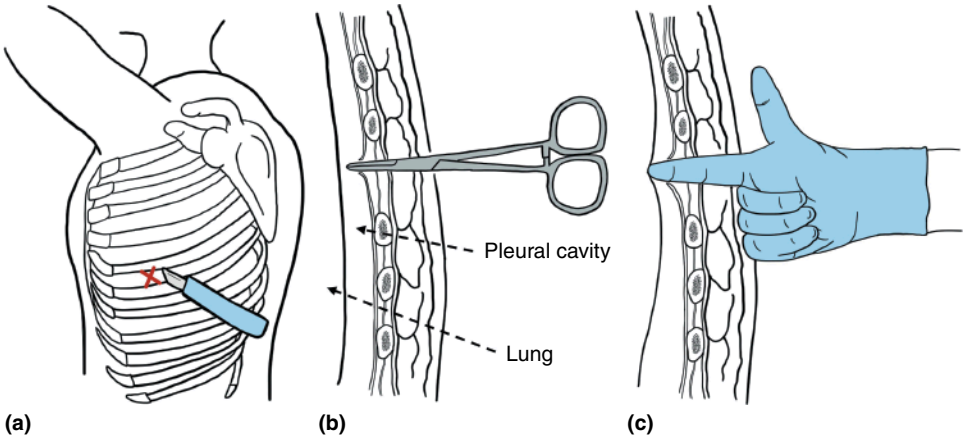


FIGURE 10.20 Finger thoracostomy technique for traumatic cardiac arrest or severe tension pneumothorax. I will usually place a large-bore chest tube under my finger after performing this technique. *Source*: Mohrsen et al. (2021)/Springer Nature/CC BY 4.0.

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IDENTIFYING THE PENETRATING ABDOMINAL WOUND

As with penetrating thoracic wounds, prompt identification of a perforating abdominal wound is important in guiding the need for operative management, as many of these patients will have injuries to the small intestine, kidney, and urinary bladder (Shamir et al. 2002). The only pathognomonic sign (with the exception of direct palpation of a body wall defect or visualization of viscera) on radiographs is the presence of a pneumoperitoneum (Risselada et al. 2008) though I think the presence of peritoneal effusion should also likely warrant surgical exploration in most cases. Furthermore, any impalement or gunshot injury should warrant an exploratory laparotomy, especially if they have peritoneal effusion or a pneumoperitoneum due to an unknown wound tract.

THE COMPLICATED BITE WOUND

While we've discussed penetrating bite wounds above, which would certainly qualify as complicated, there are non-penetrating bite wounds that can also be complicated due to either their location and/or severity. More complicated bite wounds are either those that are having life-threatening systemic effects or those with a high likelihood to heal poorly. Identifying these wounds (especially as a newer doctor) is beneficial both in planning your repair (and the need for help) and setting prognostic expectations.

The "typical" ER bite wound case is one that undergoes a sedated clip, clean, repair procedure and goes home. However, being able to identify the "sicker" population of bite wound pets is crucial in recognizing animals that would benefit from hospitalization for monitoring and care. Unsurprisingly, cats with higher lactate, lower pH, and lower ionized calcium/albumin levels tended to have more significant injuries (Klainbart 2022, Lyons et al. 2019) and older/overweight cats also tended to have a worse prognosis (Tinsley et al. 2023). In dogs, the presence of multiple organ dysfunction, longer anesthesia time, and evidence of systemic inflammation tended to have worse outcomes (Ateca et al. 2014). Therefore, in these patients, more aggressive therapy and hospitalization are likely beneficial.

The second group of patients with more "complicated" bite wounds are those at higher risk of experiencing wound healing complications. Unfortunately, there are not a lot of data identifying risk factors such as location and severity for the failure of bite wound repairs and so we are left with some general principles and anecdote. One risk factor in wound healing is the time to surgical debridement as delay converts what is a contaminated wound to a dirty wound, which are more prone to complications (Pedrina et al. 2022). A second risk factor is the number of wounds present as this portends a larger degree of subcutaneous tissue damage that can be difficult to fully debride, thus increasing the risk of abscess/seroma (Amalsadvala and Swaim 2006). In addition, the larger the number of wounds, the longer a repair can take, which has also been associated with negative outcomes (Ateca et al. 2014). Finally, the presence of infection (either due to a dirty wound or inadequate debridement) will also inhibit wound healing and increase the risk for complications even in the face of appropriate antimicrobial therapy (Demetriou and Stein 2011). My own experience is that patients with the highest risk for wound healing complications are those with multiple wounds over a large surface area where the biting animal has been able to crush/macerate a significant amount of tissue.

While your initial approach to these "high-risk" patients may not differ, it may be worth delaying repair until the tissues have started to declare themselves. Once the tissue has declared itself, it is much easier to accurately debride the affected tissue and hopefully prevent further complications. It is also important to communicate with owners that these patients may require multiple surgeries to repair their wounds.

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Spontaneous Hemoabdomen

CHAPTER 11

CRITICAL QUESTIONS

1. How sick is this hemoabdomen?
2. How do I best resuscitate the patient? When do I give a blood transfusion?
3. What is the role of imaging?
4. Is it okay to wait on surgery? Can they be managed medically?
5. Is it hemangiosarcoma?
6. What is the short- and long-term prognosis for these patients?

INTRODUCTION

In Chapter 10, we discussed management of the traumatic hemoabdomen, which is almost never surgical. In this chapter we will discuss the nontraumatic hemoabdomen, which almost always (with the exception of anticoagulant rodenticide or anaphylaxis) is. We'll go through patient identification and illness severity, followed by decision-making on fluids and transfusions. Then we'll cover the role (if any) for preoperative imaging and the evolving role of surgical timing. Finally, we'll discuss patient outcomes and complications with the goal of providing you with client communication pearls that encompass your discussion with a family about this common disease. For the purposes of this chapter, I will almost exclusively discuss the data associated with ruptured splenic masses as this is the underlying cause in the majority (roughly 75%) of hemoabdomen patients with the assumption that of the patients with malignancy, the majority will have hemangiosarcoma (Aronsohn et al. 2009).

PATIENT ASSESSMENT

You should have an incredibly high index of suspicion for a spontaneous hemoabdomen in any older canine patient with vague signs, *especially* if they have any signs of shock on your triage exam. You would be shocked at how many older patients with seemingly benign presenting complaints such as "lethargy" or "hindend weakness" will end up having a hemoabdomen. For dyspneic or cardiovascularly unstable patients, up to 75% of them will have free fluid somewhere in their body (McMurray et al. 2016), and so I would argue that any vaguely sick older patient should likely have a point of care ultrasound exam performed.

Having established the presence of free fluid in the abdomen, you should verify that it is indeed nonclotting blood via abdominocentesis. That being said, in at-risk patient populations with compatible clinical signs where I can visualize a mass, I will frequently not actually do this test. Once you've confirmed a spontaneous hemoabdomen, it's important to begin assessing disease severity and instituting resuscitation measures. Illness severity in these patients will directly correlate to the degree of shock as assessed through the following measures: mucous membrane color/capillary refill time, heart rate, pulse quality, degree of anemia, degree of hyperlactatemia, and mentation. The more abnormal findings present, the sicker your patient likely is.

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INITIAL STABILIZATION

Resuscitating these patients is conceptually very easy, but in practice, a bit more difficult. The goal is a simple one: correct shock in preparation for surgery. However, the best method to achieve that goal and your ability to accomplish it can be much more variable.

At this point, you've hopefully obtained intravenous access and have the ability to give some form of intravenous fluid. However, the choice of fluid is unfortunately highly controversial. Options for resuscitating these patients include isotonic crystalloid fluids (i.e. normal saline or Lactated Ringer's), hypertonic fluids (hypertonic saline), colloids (i.e. starches), and blood products. Given their expense and availability, I will never give blood products (rightly or wrongly) prior to talking with an owner to verify their intention to proceed with treatment. While for most shocky patients, one would typically give roughly 20 ml/kg of a crystalloid solution, the use of larger volumes of crystalloid (greater than 30 ml/kg in the average-sized human) in human patients with hemorrhagic shock leads to higher mortality (Goldman et al. 2025). Other options include colloids that have been associated with increased renal injury in humans (Hilbert-Carius et al. 2018) and hypertonic saline, which has shown no benefit over crystalloid therapy (Wu et al. 2017), both shown in human clinical trials. Current human recommendations would advocate for initial crystalloid resuscitation if blood is not immediately given (Rossaint et al. 2023). I do think that it's important to acknowledge this is a controversial point in human medicine, and I have provided a simple answer to what is a very complex question. In veterinary medicine, there is only one study looking a resuscitation fluid choice in spontaneous hemoabdomen that showed a 15-minute faster resuscitation with hypertonic saline/colloid compared to crystalloid, though that made no impact on survival (Hammond et al. 2014). The remainder of the veterinary literature is either expert opinion or in experimental models of hemorrhage (Hall and Drobatz 2021, Boyd et al. 2021). My personal practice is to give 20 ml/kg of crystalloid while I go and talk to the owner. If the patient is unstable, I will very quickly try to get permission for a blood transfusion and may consider hypertonic saline.

Beyond the type of fluid to give, the next question is how much to give and what blood pressure to target. The concept of low volume resuscitation and permissive hypotension implies that you don't give as many fluids because you are not targeting a normal blood pressure. While this has been shown to potentially lead to better outcomes in human medicine (Tran et al. 2018), experience in veterinary medicine is again limited to expert opinion (Balakrishnan 2020) and research models. My concern with this approach is that in human medicine, their "door to surgery" time is usually much lower than in veterinary medicine, and leaving a patient hypotensive for too long has the potential to precipitate organ dysfunction.

Next comes the decision about when to give a red blood cell transfusion. We discussed transfusion risk factors in the section on abdominal trauma but to briefly reiterate: dogs with lower total protein, higher lactate, lower PCV, and a worse acidosis are more likely to require a transfusion (Lynch et al. 2015, Buseman et al. 2024). However, I would also add that failure to stabilize (i.e. improved perfusion and shock parameters) with one crystalloid bolus is also a trigger for me to reach for a packed red blood cell transfusion. The rate at which I give the transfusion will vary depending on patient stability: the more unstable, the faster I give it. If I feel like I'm reaching my resuscitation goals, then I will slow down the transfusion speed.

There are also patients who will not stabilize with one transfusion or who are so unstable on presentation that they require a massive transfusion. Massive transfusion in veterinary medicine is traditionally defined as giving 100% of a patient's blood volume in 24 hours, 50% of blood volume in 3–4 hours, or administration of greater than 1.5 ml/kg/min for 20 minutes (Jutkowitz et al. 2002, Boyd and Smart 2023). While survival and prognosis for patients receiving a massive transfusion are not very high at around 30% on average (Lynch et al. 2015, Tucker et al. 2022), it's crucial that you monitor for hypocalcemia in this patient population.

If you have an inadequate supply of packed red blood cells, you may need to consider an autotransfusion where you remove the blood from a patient's abdomen, run it through a filter,

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and inject it into a vein. Traditionally, the use of autotransfusion in spontaneous hemoabdomen was not done due to concern over "seeding" potential tumor cells elsewhere in the body. However, the use of a cell salvage device and leukocyte reduction filter has been shown *in vitro* to remove hemangiosarcoma cells (Ciepluch et al. 2018). I would also argue that we infrequently have a definitive diagnosis of hemangiosarcoma preoperatively, so if there aren't any other options, this is better than nothing. In the largest retrospective studies of patients receiving autologous blood transfusions, there were no clinically significant complications (Higgs et al. 2015, Blunschi et al. 2025) and virtually identical survival when compared to "traditional" transfusion therapy (Comas Collgros et al. 2024).

As we discussed in Chapter 10, antifibrinolytic therapy is frequently considered in the management of these patients. While hyperfibrinolysis is fairly common (40%) in shocky patients with a spontaneous hemoabdomen, given its rapid resolution with surgery (DeStefano et al. 2023) the role for antifibrinolytics such as tranexamic acid is unclear in this patient population. For all patients with hyperfibrinolysis due to hemorrhagic shock, the use of tranexamic acid resolved the hyperfibrinolytic state but conferred no survival or transfusion benefit (Mays et al. 2025). The growing availability of point of care viscoelastic testing may make it easier to identify hyperfibrinolytic patients (Baird et al. 2025), but future trials would be required to document improved outcomes with antifibrinolytic medications.

PROS AND CONS OF IMAGING

Traditionally, we were always taught that all patients with a hemoabdomen should have an abdominal ultrasound and thoracic radiographs to evaluate for evidence of metastatic disease. However, that approach begs two very important questions, especially when abdominal ultrasound is not available 24/7: what is the accuracy of these tests, and what is the prognostic impact of metastatic disease in this patient population?

A landmark study by Cudney et al. showed that there is a discrepancy in up to 54% of dogs when compared abdominal ultrasound findings to those at surgery or necropsy (Cudney et al. 2021). Follow-up studies have shown that the appearance of a splenic mass does not predict malignancy (Millar and Zersen 2021) and that the presence of liver nodules was actually associated with a higher incidence of a benign splenic lesion! (Ramirez et al. 2024). While this is not to say that abdominal ultrasound is a useless test, as these retrospective studies do have some level of selection bias, you should be aware of these limitations when talking to the owner. I will discuss the pros/cons of this test, and if there are similar cavitated lesions in the liver AND spleen with evidence of hemoabdomen, I will not recommend surgery. That being said, there is a percentage of patients (perhaps up to 25%) with multiple lesions where both are benign (Levinson et al. 2009, Leyva et al. 2018) though these studies were not exclusively in patients with a hemoabdomen. Even abdominal CT, which is thought to be more sensitive and specific, is debatable at reliably detecting metastatic lesions with different studies yielding variable results (Fife et al. 2004, Jones et al. 2016, Parry et al. 2023) though the addition of a thoracic CT likely improves overall diagnostic accuracy (Mattolini et al. 2024). Finally, chest X-ray can be a reasonably specific but insensitive test for pulmonary metastasis when compared with histopathology (Holt et al. 1992, Hammer et al. 1993) and the presence of sternal lymphadenopathy does not necessarily indicate malignancy (Kelsey et al. 2022).

The final question with imaging is *does* the presence of metastasis change the prognosis of these animals. If we compare patients with stage III splenic hemangiosarcoma, which is defined as a ruptured primary splenic tumor with evidence of visible metastasis who were treated with *surgery only* compared to those with a ruptured splenic mass with NO visible metastasis (stage II) the survival times were 1.3 months versus 2 months (Marconato et al. 2019, Wendelburg et al. 2015). While this difference is likely statistically significant, I think it is also important to ask an owner

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whether or not that additional 3 weeks would make a difference in terms of their decision to treat. If the answer is no, then imaging may not make a difference in their decision-making.

As we started off with, the decision to do or not to do imaging is mostly based on the question of would it change an owner's decision-making? As we have seen, the prognosis for hemangiosarcoma with and without metastasis (barring chemotherapy) is different but not exponentially so. Available testing including thoracic radiographs, abdominal ultrasound, and CT are all imperfect at detecting metastasis. As we'll talk about later, an owner has to make the surgical decision based on an inadequate amount of data and imaging may or may not change that. Finally, there are also patients who are unstable for abdominal imaging (most can tolerate thoracic radiographs), and if you have to call in a radiologist to perform a scan, that delay in surgery can also be detrimental to patient outcome.

SURGICAL TIMING AND NONSURGICAL MANAGEMENT

As an emergency clinician, it can be very daunting to have to call a surgeon in the middle of the night. While after-hours surgery has been associated with worse outcomes in humans (Stefani et al. 2018), this has not been demonstrated in dogs (McConkey et al. 2021). To date, there has been no study that has directly evaluated immediate versus delayed surgical management in spontaneous hemoabdomen. The closest study examined patients who underwent immediate surgery versus those who attempted medical management (Wright et al. 2021). Of the medically managed patient population, 43% went on to surgical management (though not necessarily due to decompensation) with minimal difference in survival and cardiovascular stability. Your surgery and emergency/critical care teams will have to develop a hospital protocol as to surgical timing in these patients.

One question an owner always asks is what is the prognosis with nonsurgical management. Obviously, this is case dependent because the shocky hemoabdomen will do very poorly with medical management, whereas a more stable one may survive for some time. While there is a general dearth of literature on the subject, of hemoabdomen patients who *didn't* receive surgery, 38% died within 12 hours and an additional 13% likely died shortly thereafter (Wright et al. 2021). There are a large number of unknowns in sending these patients home in terms of whether or not there is an active hemorrhage and when a bleed will restart. Owners should be appropriately counseled about the potential of their pet passing away at home.

IS IT HEMANGIOSARCOMA?

Obviously, the long-term prognosis for a hemoabdomen patient depends on whether or not they have an aggressive neoplasia, such as hemangiosarcoma versus a benign lesion. If an owner knew their patient had hemangiosarcoma, then most would make a different treatment decision, then if they knew the lesion was benign.

As we all know, there is no easy answer to this question. The first question is to understand the overall likelihood of hemangiosarcoma in a dog with a ruptured splenic mass. Overall, the likelihood of a malignancy is roughly 53–73% and the likelihood of hemangiosarcoma being that malignancy is high at 87% in one study (reviewed in Schick and Grimes 2023). While some other studies have indicated a lower incidence of hemangiosarcoma at around 58% (Ziogaite et al. 2024), the cited study included dogs with and without hemoabdomen, which is an important confounding bias. Factors that *may decrease* the likelihood of hemangiosarcoma include a larger mass compared to the spleen itself/total body weight (Mallinckrodt and Gottfried 2011) and a small breed dog or one weighing less than 28 kilograms (Sherwood et al. 2016, O'Byrne and Hosgood 2019).

The holy grail of hemoabdomen management would be a preoperative test that would differentiate between benign and malignant etiologies. While there have been multiple studies trying to elucidate this, they have all come up short. The first predictive score (HeLP) used body weight, total protein, platelet count, and thoracic radiograph results to stratify patients into low (<36%

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chance), medium (36–76% chance), and high (>76% chance of having hemangiosarcoma (Schick et al. 2019). A later study (T-STAT) used eight variables including total protein, ≥ 2 nRBCs/100 WBCs, splenic mass diameter, presence of liver nodules, solitary versus multiple splenic lesions, degree of splenic mass homogeneity, degree of abdominal effusion, and the presence of mesenteric/omental/peritoneal nodules to calculate a likelihood of hemangiosarcoma (Burgess et al. 2021). While both of these scores showed some degree of promise in the initial phase, external validation showed only "acceptable" performance for HeLP and "poor" performance in T-STAT for differentiating benign versus malignant etiologies (Hillier et al. 2024). While advanced testing, such as contrast-enhanced MRI, microRNA, and nucleosomes may allow differentiation in the future (Lux et al. 2024, Meazzi et al. 2024, Ludwig et al. 2025), until it is a point of care, it will not be clinically applicable to the emergency clinician. Therefore, to this point, there is no single diagnostic tool that can definitively differentiate between benign and malignant etiologies.

SHORT-TERM COMPLICATIONS

It's also important not to ignore the potential short-term complications for patients undergoing surgery for a hemoabdomen as the perioperative mortality rate is between 7 and 15% (Lux et al. 2013, Wendelburg et al. 2014). Beyond shock, which we discussed above, cardiac arrhythmias are probably the most common perioperative complication we'll see with these patients, occurring in between 30 and 40% of dogs (Wendleburg et al. 2014, Michael et al. 2023). And while the majority of these patients are fairly easy to control with lidocaine and/or sotalol if persistent, both of the above studies did show a small increase in mortality for patients with arrhythmias. While I tell owners that this is a possibility and that we monitor for it, in my experience, the majority of dogs require neither discharge medication nor long-term management. Anecdotally, the frequency of arrhythmias I have seen has decreased since I became more aggressive with early blood transfusions.

The more challenging (but thankfully less common) complications are thromboembolic events such as a portal vein thrombus that are frequently fatal (Wendelburg et al. 2014). The mechanism for this complication is unknown. In one study, 87% of patients undergoing splenectomy were hypercoagulable (based on viscoelastic testing) in the preoperative phase (McPhetridge et al. 2022) though none of them went on to develop a thrombus. The majority of postoperative patients were also shown to be hypercoagulable and have elevated platelet counts (Phipps et al. 2020), though only one of those patients developed a suspected thrombus. While one group did evaluate the use of clopidogrel in postoperative patients, they saw no difference in outcome despite an improvement in platelet count/viscoelastic parameters in the group who received the drug (Joung et al. 2024). That study was significantly underpowered to detect a survival difference given the rarity of thromboembolism in this patient population. I do not routinely start these patients on anticoagulants because there are no data supporting a survival benefit. I have also had one patient develop a mesenteric thrombosis after stopping their clopidogrel 2 weeks postoperatively. While I warn owners about this complication, I also inform them that it is quite rare.

Unfortunately, there is a population of dogs (potentially between 10 and 20%) with benign splenic lesions that will also pass away prematurely (Patten et al. 2016, Millar et al. 2022). Whether these deaths are due to an inaccurate histopathologic diagnosis, cardiac arrhythmia, or thromboembolism is not entirely clear.

SURGERY ALONE VERSUS CHEMOTHERAPY

With surgery alone for stage II splenic hemangiosarcoma (ruptured tumor with no visible metastasis), the mean survival time is roughly 2 months while for stage III (visible metastasis) is between 1 and 2 months depending on the study (Wendelburg 2014, Marconato et al. 2019).

It would be outside the scope of this chapter to review absolutely every chemotherapy protocol that exists for canine hemangiosarcoma, and the presence/absence of metastatic disease will

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influence prognosis. For stage II (ruptured tumor, no visible metastasis) treated with adjuvant chemotherapy (usually doxorubicin though some will usually involve carboplatin or metronomic chemotherapy protocols) is likely around 5 months (Moore et al. 2017). For stage III (ruptured tumor with metastasis), the survival is likely closer to 4 months (Marconato et al. 2019). Are these differences worth it compared to the potential side effects of chemotherapy? That's honestly not for me to answer but rather is information for an owner to consider.

Ultimately, there may be treatment involving immunotherapy that improves survival further, providing a cost-effective mechanism for enhanced survival (Marconato et al. 2023); these treatments remain in clinical trials.

CLIENT COMMUNICATION PEARLS

It can be daunting to walk into talk to a family whose pet has a hemoabdomen, especially when the patient is sick. Below are the bullet points that I use to describe the diagnostic and therapeutic approach to this disease.

1. Your dog is currently bleeding into its abdomen, which is most commonly associated with rupture of a mass on an organ in the abdomen, most commonly the spleen.
2. The majority of these tumors (likely between 75 and 80%) represent a blood vessel tumor known as hemangiosarcoma.
3. This is a disease that requires surgery to definitively treat, as there is no viable medical management option that leads to a reasonable long-term outcome. If you would not take your pet to surgery, then the remainder of the diagnostic workup and treatment discussion is mostly academic.
4. The goals of imaging (ultrasound and radiographs) are to try and detect metastatic lesions that would preoperatively confirm a diagnosis of cancer. These tests are imperfect and the absence of evidence (of metastasis) is NOT evidence of absence.
5. There is no 100% definitive preoperative test that allows us to distinguish benign versus malignant lesions, only tests that can increase our level of suspicion. Unfortunately, you will have to make a big decision without all of the data that would be helpful to make the most informed/perfect decision.
6. The best-case scenario is that this is a benign lesion and removing it is curative; the worst-case scenario is that this is hemangiosarcoma. For dogs with hemangiosarcoma treated with surgery alone, the median survival time is roughly 1–2 months though the quality of life during that time period is usually good to excellent. Chemotherapy can sometimes extend this time to a median survival time of 4–5 months.
7. I have found that many owners are satisfied with their surgery decision regardless of underlying diagnosis, which correlates with the one survey study performed (Menard et al. 2023). And while junior clinicians are more likely to recommend euthanasia in these patients (Molitoris et al. 2022), in my opinion, recommending euthanasia for all hemoabdomen patients is ultimately harmful.
8. Perioperative risks and complications include need for red blood cell transfusion, cardiac arrhythmias (usually manageable, occasionally require temporary treatment), and rarely, blood clotting complications. While blood clots are frequently fatal, they are also rare. Prophylactic treatment has not been shown to be beneficial.
9. Review the cost of care at your hospital for these tests and treatments.

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Gastric Dilatation and Volvulus

CHAPTER 12

CRITICAL QUESTIONS

1. How stable is this patient?
2. Is lactate important?
3. Is lidocaine helpful?
4. How and when should I decompress?
5. How quickly should surgery happen?
6. What is the postoperative prognosis with GDV?

INTRODUCTION

Similar to spontaneous hemoabdomen, the gastric dilatation and volvulus (GDV) patient can represent another frightening condition for the junior clinician because of its ability to cause rapid cardiovascular instability and death. When the stomach twists on itself, it will cause a shock state through multiple mechanisms and can potentially cause gastric ischemia and necrosis. These patients can also have a concurrent cardiac injury and multiple organ dysfunction as a result of this disease (Sharp and Rozanski 2014) that can similarly be life-threatening.

It's important to always have GDV as a differential diagnosis for any patient who has any of the following clinical signs: nonproductive retching, pacing, restlessness, and abdominal distention, especially in predisposed breeds such as a Great Dane or German Shepherd (Rosselli 2022). Anecdotally, I would also add that standard Poodles are prone to GDV and will look and be deceptively stable. While abdominal radiographs, as shown in Figure 12.1, are diagnostic for this condition, in patients you deem unstable (see below) you should already initiate stabilizing measures prior to obtaining radiographs.

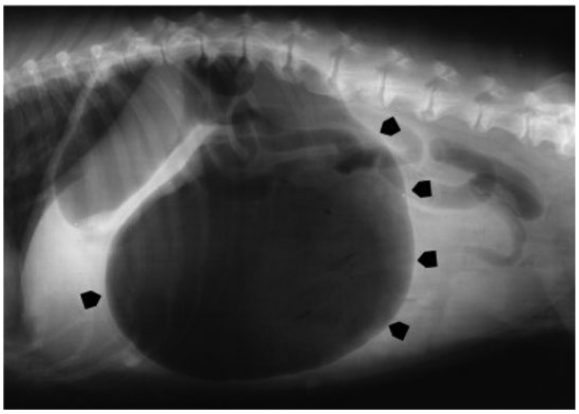


FIGURE 12.1 Right lateral radiograph showing the "boxing glove" or "double bubble" of a GDV. *Source*: Carberry (2022) with permission of Elsevier.

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I have also found that point of care ultrasound can also be helpful in demonstrating a large gas-filled structure on the left side of the abdomen that, combined with a shock state, is highly suspicious (though not diagnostic) for GDV. Abdominal radiographs are reasonably specific (but very insensitive) at predicting gastric necrosis (Fischetti et al. 2004). That being said, the majority of the patients I have treated with gastric necrosis presented unstable and failed to stabilize preoperatively. Concurrent thoracic radiographs can be considered, though the stability of the patient should be weighed against their diagnostic yield, as the majority of changes seen generally don't change treatment approach (Green et al. 2012).

THE 360° GDV

I've included this section here in the beginning because, to be honest, in over 9 years of emergency and critical care practice, I have not identified one of these cases, meaning it is fairly uncommon. However, I do think it's important to always have an appropriate index of suspicion for atypical cases especially when the patient is quite sick or unstable. The limited literature on this condition indicates that it tends to affect older dogs with radiographically greater esophageal dilation and less small intestinal dilation than seen with gastric dilatation without volvulus (Espinosa Mur 2023). The sensitivity, specificity, and agreement between radiologists in diagnosing a 360° GDV **were also poor and the incidence rate in this study was low** (Mur et al. 2025).

ASSESSING CLINICAL STABILITY

GDV has the potential to cause shock through four different mechanisms: obstructive (due to gastric dilation with secondary decreased venous return to the heart), distributive (due to splanchnic pooling of blood), hypovolemia (usually through the gastrointestinal tract), and cardiogenic (if arrhythmias or myocardial dysfunction are present). Therefore, your initial assessment for the stability of this patient is no different than for a patient with any other shock-inducing disease: mucous membrane color/CRT, heart rate, pulse quality, mentation, and lactate.

Cardiac arrhythmias (usually ventricular) can be present in up to 17% of patients presenting with GDV (Aona et al. 2017), and it is not uncommon for them to develop postoperatively. While the prognostic significance of these arrhythmias is unknown (Thomson 2021), it's crucial to screen these patients for ventricular arrhythmias to optimize their preoperative resuscitation for anesthesia. The majority of these arrhythmias will improve/resolve with a lidocaine bolus and continuous rate infusion. Generally, I will start lidocaine if their heart rate is greater than 180 bpm (post fluids and analgesia), R-on-T phenomenon is present, or there are multiform ventricular complexes.

Lactate receives special attention for the GDV patient because of the plethora of studies that have evaluated its prognostic potential and predictive ability for gastric necrosis. Without going into each individual study, I will summarize both mine (and others) conclusions about lactate in these patients. **A low lactate at presentation (<4 mmol/l) is generally a good sign, as is a high lactate that improves (>40% clearance) with stabilization.** A high lactate at presentation or one that doesn't improve with stabilization does not always portend a negative diagnosis (Mooney et al. 2014, Mackenzie 2022). If the lactate fails to normalize or dramatically improve postoperatively, then that would likely be a negative prognostic indicator (Verschoof et al. 2015). In my practice for patients with GDV, a positive trend in lactate is a good thing though a static value would not deter me from recommending surgery.

IMPROVING CLINICAL STABILITY

As with patients with a spontaneous hemoabdomen, the goal of resuscitation is to improve their shock state as much as possible, acknowledging that definitive correction of their obstructive

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shock requires surgery. As we discussed above, the shock state for these patients is multifactorial, therefore, our stabilization approach must be multipronged as well.

As with all (noncardiogenic) etiologies for shock, an intravenous fluid bolus is typically the first step in correcting whatever hypovolemic (+/− distributive) component of shock is present. While your choice of fluid may have some acid/base implications, it has not been shown to affect outcome or prognosis (Rauserova-Lexmaulova et al. 2022), so any isotonic crystalloid is likely a reasonable choice.

Appropriate analgesia is imperative for these patients and can be very helpful in reducing heart rate, which is one of the values we track to indicate improving stability. There have not been any studies evaluating opioid selection in GDV patients, and so likely any pure mu opioid (fentanyl, hydromorphone, or methadone) is likely okay. I will frequently also give maropitant as an antiemetic though admittedly its overall contribution to preoperative stabilization is probably small.

For patients experiencing cardiac arrhythmias, lidocaine is the first-line agent (see above). However, the prophylactic use of lidocaine in GDV patients without cardiac arrhythmias has also been studied. The first study using a bolus of lidocaine at presentation followed by a continuous rate infusion showed reduced risk for cardiac arrhythmias (unsurprising), acute kidney injury, and decreased hospitalization, albeit this was compared to historical controls (Bruchim et al. 2012). However, subsequent studies failed to show improvement in inflammatory and renal biomarkers (Brunner et al. 2024, Lehmann et al. 2023). I do not routinely use lidocaine in these patients unless indicated by cardiac arrhythmias.

GASTRIC DECOMPRESSION

Having (hopefully) addressed or improved the components of hypovolemic, distributive, and cardiogenic shock, gastric decompression is the procedure most likely to improve obstructive shock. There are two widely accepted techniques for this: orogastric intubation and percutaneous trocharization (Figure 12.2).

The single study comparing the two approaches head-to-head showed no difference in success rate and outcome (Goodrich et al. 2013). I prefer trocharization because it is technically easier and doesn't require general anesthesia. I will always use ultrasound to ensure that the spleen is



(a) (b)

FIGURE 12.2 Gastric trocharization can be accomplished using an 18- or 14-gauge IV catheter. (a) Identify an area of tympany and (b) verify the absence of solid organs (i.e. spleen) in your needle tract and place an extension set in water to verify ongoing gas extrusion. *Source*: Tivers et al. (2015) with permission of John Wiley & Sons.

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not present in the field I intend to trocharize to reduce the risk of splenic puncture. You should only see the gas-dilated stomach in your ultrasound field if you are in the correct window. Other techniques such as placement of gastrostomy catheter (Fox-Alvarez et al. 2019) and nasogastric intubation (Lhuillery et al. 2022) are described but should likely only be employed when surgery is being delayed for a significant period of time (see below).

SURGICAL TIMING

A GDV has almost always been described as a "true surgical emergency" because increased time to surgery was intuitively thought to correlate with an increased risk of gastric necrosis and had been demonstrated to reduce mortality (Mackenzie et al. 2010, Song et al. 2020). Since those studies, there have been two further papers that have demonstrated that in a patient population *whose shock state/gastric dilation effectively stabilizes and remains stable*, surgery can be delayed with no significant change in mortality or gastric necrosis (White et al. 2021, Lhuillery et al. 2022). I think this is a conversation to have with your surgery team because the success of a delayed approach is predicated on successful stabilization of shock and gastric dilation through orogastric intubation, trocharization, nasogastric intubation, or a combination of above, and the maintenance of that stable state.

POSTOPERATIVE MANAGEMENT AND PROGNOSIS

As with our preoperative assessment for these patients, postoperative management involves ensuring the resolution of a shock state (physical exam and laboratory/vital parameters), monitoring for arrhythmias, and managing any respiratory (pneumonia), gastrointestinal (ileus), and renal sequelae (Bruchim and Kelmer 2014).

The overall survival rate for postoperative GDV patients is between 77 and 88% depending on the study cited (Rosselli 2022). Negative prognostic indicators for these patients include concurrent splenectomy (may increase mortality up to 15–32%), gastric necrosis/partial gastrectomy (may increase mortality from 10–46%), and gastric perforation/sepsis (Mackenzie et al. 2010, Beck et al. 2006, Brourman et al. 1996). In my own experience, I think the survival is likely a little bit higher, and it is uncommon for a partial gastrectomy to be required. I have not necessarily found splenectomy to be a negative prognostic indicator either.

CONCLUSIONS AND CLIENT COMMUNICATION PEARLS

Your first GDV stabilization can be intimidating, but focusing on the tools you have available and frequent patient assessment will help you get them safely to surgery. I've included my own initial approach in Figure 12.3. What is most important is to constantly reevaluate whether or not your treatments have improved your patient's condition to determine if additional measures are required. Some of my communication points with owners are as follows:

- GDV is a disease where the stomach has rotated on itself and become gas distended, which causes a shock state. Stabilization includes fluid therapy, pain medications, and decompression.
- Surgery is required to derotate the stomach and fix it back in place. Due to this rotation, some dogs need their spleen removed, which may slightly worsen their prognosis. Less commonly, a portion of the stomach may have died and require removal, which is also a negative prognostic indicator.
- These dogs will frequently have ventricular arrhythmias that we monitor for and, if necessary, treat. They tend to be temporary.
- The majority of these dogs will recover well and the prognosis in most is good.

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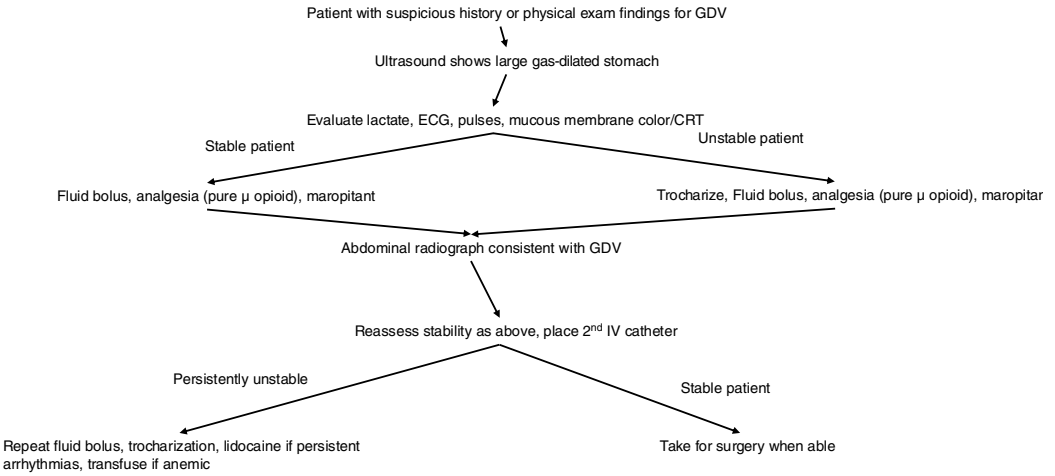


FIGURE 12.3 The author's initial diagnostic/therapeutic approach to the GDV patient. Courtesy of the author.

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The Blocked Cat

CHAPTER 13

CRITICAL QUESTIONS

1. Are they hyperkalemic?
2. Should I perform a decompressive cystocentesis?
3. What is the utility of diagnostics?
4. What if I can't unblock him?
5. Can they be managed as an outpatient?
6. Should I give prazosin?
7. What are the chances he re-blocks?
8. What if the catheter isn't flowing?

INTRODUCTION

Feline urethral obstruction (UO) is a common emergency comprising up to 4% of feline cases presenting to an emergency room with a recent increase during the global COVID-19 pandemic (Finstad et al. 2023). It's crucial to have a high index of suspicion for UO in any male feline patient presenting with vague signs, including abnormal vocalization, vomiting, excessive licking, and abnormal gait in addition to the more traditional signs of stranguria, in/out of litter box, hematuria (Lee and Drobatz 2003).

My diagnostic, treatment, and monitoring protocols are outlined in Figure 13.1, and hopefully, you get more information as to the support behind my approach below!

ASSESSING PATIENT STABILITY AND MANAGING HYPERKALEMIA

The vast majority of instability in feline UO patients is due to hyperkalemia with concurrent severe metabolic acidosis and hypocalcemia that occurs in roughly 40% of patients (Lee and Drobatz 2003). While running point of care lab work should be performed in all UO patients to assess illness severity, those at the highest risk for hyperkalemia include those with low temperatures and heart rates among other physical exam and historical factors (Lee and Drobatz 2006).

For the hyperkalemic cat, traditional management has included the use of calcium gluconate, fluid therapy, and parenteral insulin/dextrose administration (Jones et al. 2022). More severe cases may receive beta-adrenergic agonists and sodium bicarbonate, though these approaches are based only on expert opinion. However, one study in healthy cats *without* UO did show that albuterol will lower blood potassium levels within 3 minutes and may be a good stabilizing measure (Jaffey et al. 2025). A study just published showed that beyond the use of fluids, calcium gluconate, and unblocking, there is no survival or time to resolution of hyperkalemia benefit in using any of those medications (Maciorowski et al. 2025). My current initial stabilization approach for these cats is to give calcium gluconate along with IV fluids, and then expedite unblocking, though in certain cases I have given albuterol.

DECOMPRESSIVE CYSTOCENTESIS

While decompressive cystocentesis in this patient population is relatively low risk with few reported complications (Hall et al. 2015, Gerken et al. 2020), it hasn't been shown to improve the ease of subsequent catheterization as many anecdotally claim (Reineke et al. 2021). It is not a standard part of my practice, but there may be times when you have to consider it because you can't

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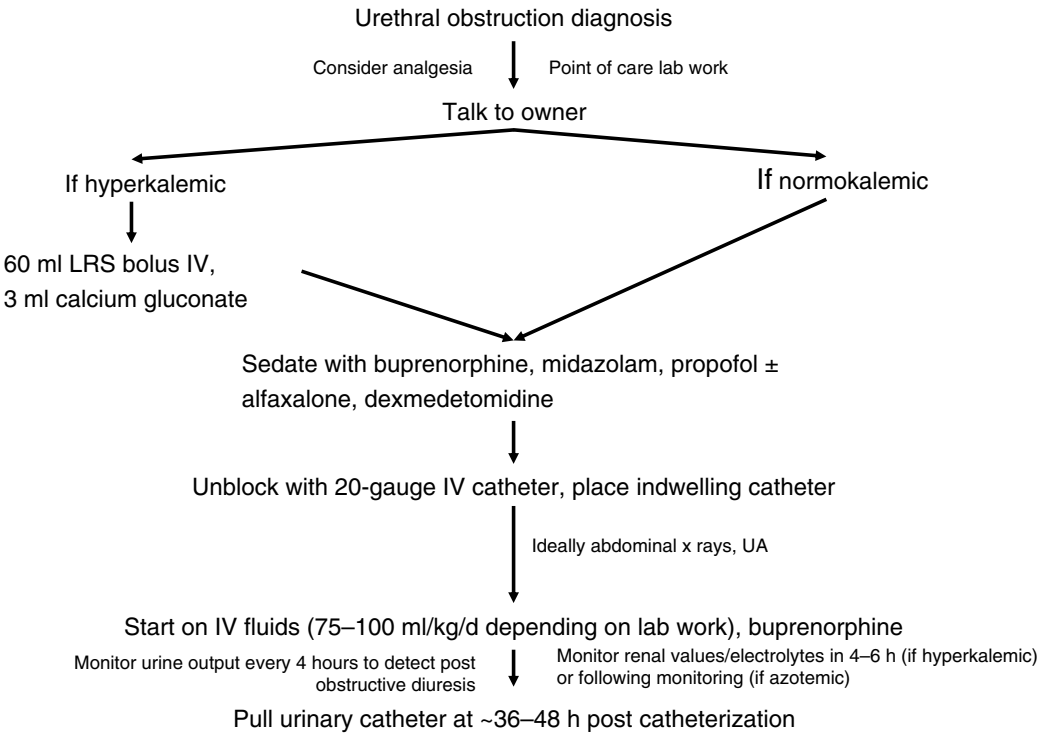


FIGURE 13.1 The author's diagnostic, therapeutic, and monitoring approach to feline urethral obstruction. Courtesy of the author.

unblock them in a timely manner, they require transport, or you are unable to successfully place a urinary catheter. I think this procedure has an appropriate time and place but should not inordinately delay more definitive management. My personal practice is to use the procedure if I expect a lengthy delay prior to urethral catheterization or if the owner elects transfer of care to another facility.

THE USE OF DIAGNOSTIC TESTING

Besides point of care lab work, which I think is nonnegotiable, it is worth discussing the diagnostic value of imaging and urinalysis in these cases, given their financial implications. The goal of abdominal radiographs post catheterization is twofold: to verify appropriate catheter placement and evaluate for the presence of (radio opaque) cystic calculi. While there is no study on the former topic, anecdotally, I have used point of care ultrasound frequently to verify catheter placement with good success. In a study of radiography in UO cats, only 10% of cats had changes (i.e. stones) that would warrant a treatment change (Young et al. 2021). In that same study, more changes (clinically relevant or not) were seen in radiographs taken BEFORE catheterization! Therefore, for owners with financial constraints, it is reasonable to omit radiographs after providing them with informed consent.

The rationale for urinalysis and urine culture in this patient population is to evaluate for biochemical changes in the urine and evidence of infection, even though infection was documented in zero cats in one study (Cooper et al. 2019). While UO cats will frequently have changes such as hematuria, pyuria, and crystalluria (Segev et al. 2011), it is uncommon that

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those results lead to a change in treatment approach. The exception is for cats with a recurrent urethral obstruction (rUO) who have been recently catheterized, who I would expect to be at higher risk for bacterial cystitis.

The purpose of the above discussion is not to say you should *never* do these tests, but as we talked about in Chapter 2, you should be aware of their diagnostic yield. For owners with financial constraints, these are the first things I will eliminate from a cost estimate after informing them of the data we won't get with that approach.

THE CHALLENGING (AND UN-UNBLOCKABLE) CAT

I will omit a step-by-step description of how to unblock a cat, as you can easily find that elsewhere (see additional reading). Briefly, my sedation protocol is a combination of an opioid (usually butorphanol), midazolam, and propofol with the occasional low dose (1 mcg/kg) of dexmedetomidine. For fractious cats we can't place an intravenous catheter in, I will add on alfaxalone as part of an intramuscular protocol. I do not use ketamine in these patients, as anecdotally, I have had two or three cats go into cardiac arrest post administration. That being said, as we discussed in Chapter 5, low dose (i.e. 1–2 mg/kg IM) is likely safe in the majority of patients. The only study examining injectable sedation protocols in an experimental UO population showed faster recovery when propofol was used (compared to ketamine/diazepam) with no difference in biochemical stabilization (Freitas et al. 2012). For "higher risk" patients, you can also refer to Chapter 5 for further information on building a sedation protocol.

For initial unblocking, I will start with a 20-gauge IV catheter (without the stylet) as that allows me to use a Luer lock t-set to facilitate retropulsion. Following successful unblocking, I will generally replace that with either a 3.5 French red rubber catheter or Slippery Sam™ catheter, depending on backorder availability.

For cats you are struggling to unblock, there are a couple of strategies you can employ. The first is to change the catheter you are using. Sometimes changing the catheter type (i.e. from Tomcat to Slippery Sam) is enough to allow you either bypass or retropulse the obstruction. The second is to consider placing the cat under general anesthesia to theoretically reduce urethral sphincter tone. The only study directly comparing procedural sedation and general anesthesia only looked at recurrent obstruction and complications, not ease of catheterization, and found no differences (Perrucci et al. 2023). A decompressive cystocentesis (see above) may also reduce back pressure, though as discussed above, its benefit is questionable. Next is to consider using a sacrococcygeal block (Figure 13.2) to reduce urethral spasm (O'Hearn 2011). While this technique has

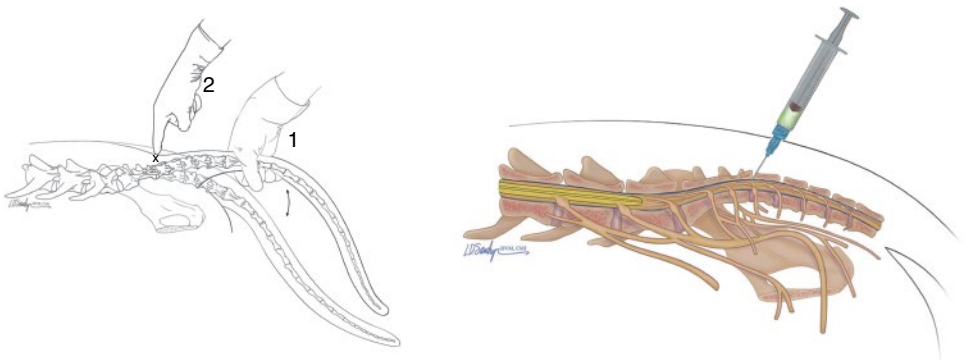


FIGURE 13.2 A sacrococcygeal block can facilitate/ease urinary catheterization. *Source*: Grubb et al. 2020 with permission of American Animal Hospital Association.

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been shown to reduce sedation requirements (Pratt et al. 2020), its utility as a rescue technique has not been evaluated and in the study cited, it was not perceived to make catheterization any easier.

Finally, while intra-urethral atracurium (a paralytic) was shown to facilitate ease and timing of catheterization (Galluzzi et al. 2012), I have never done this and there is a case report of it causing systemic neuromuscular blockade (Casoria et al. 2023), so definitely weigh the risk/benefit ratio!

Approximately 1% of cats are un-unblockable (Beeston et al. 2022) likely due to either a urethral tear or urethral stones. Risk factors for urethral rupture would include a recent obstructive event and a challenging unblock (Manchester et al. 2024). If you are truly unable to catheterize the patient, you have the following options besides euthanasia. The first is an emergent perineal urethrostomy surgery (Corgozinho et al. 2007), though if your patient is hyperkalemic/azotemic, this may be risky from an anesthetic perspective. The second is to manage/stabilize with repeated decompressive cystocentesis and fluids (if azotemic/hyperkalemic) until surgical intervention, understanding that the cumulative risk of bladder trauma will increase the more times you perform that procedure. The third is to attempt anterograde catheter placement over a guidewire under fluoroscopic guidance (Holmes et al. 2012), though few hospitals will feel comfortable with this and there are several downsides. The fourth is to place a pigtail cystostomy catheter in the bladder to allow ongoing drainage (Nurra et al. 2022). Which of these options is the most appropriate will depend on the time of day, situation, and your hospital's logistics/equipment availability.

OUTPATIENT MANAGEMENT: THE UNBLOCK AND GO

Frequently, owners will be unable to afford the cost of inpatient diagnostics and management for UO. At my current hospital, the cost is roughly US$4500. We discussed the pros and cons of diagnostic testing in a "financial" case, and the second option to reduce cost is to reduce hospitalization time, which we'll talk about briefly in a later section. For owners who can only afford outpatient care (unblock and go), success depends on appropriate case selection and an honest conversation about expectations with that family.

The ideal outpatient candidate is a young, stable, nonazotemic, normokalemic cat, as these patients are the least likely to develop a potentially life-threatening post obstructive diuresis (Muller et al. 2022). Owners should also be aware that cats treated as an outpatient have approximately a three times greater likelihood of recurrent obstruction (Seitz et al. 2018) though that was not necessarily linked to an increase in mortality (Beeston et al. 2022). There is also a gray area for the mildly to moderately sick UO (i.e. azotemia, mild hyperkalemia) whose outcome with outpatient care could be okay. My own rough cutoff for outpatient therapy is a creatinine of roughly 4 mg/dl though that is not based on anything beyond my opinion.

LIKELIHOOD OF RECURRENT OBSTRUCTION AND SHOULD I BE GIVING PRAZOSIN

The factors affecting the rate of rUO are likely one of the most highly researched areas in feline emergency medicine. The most commonly cited factors thought to influence the rate of rUO include catheter size, urinary bladder lavage, duration of catheterization, improvement in urine color, and, of course, the administration of prazosin (Cosford and Koo 2020). In this section, I hope to review the available literature and will provide my own clinical practice as well, understanding that there are numerous conflicting studies that recommend different approaches.

We briefly touched on the duration of catheterization above when discussing inpatient versus outpatient management. However, there is also controversy as to how long an indwelling catheter should remain in place with one study saying it isn't important (Hetrick and Davidow 2013) while another indicates an optimal 48-hour catheterization (Eisenberg et al. 2013). My own personal practice is to recommend a 48-hour hospitalization as long as that is affordable to the owner.

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While an initial study seemed to indicate a lower rUO rate with smaller (3.5 fr) catheters (Hetrick and Davidow 2013), its competing study (Eisenberg et al. 2013) did not show any difference. Similarly, no difference was seen depending on the composition of the catheter itself (Davidow 2020). I will place a 3.5 fr red rubber or slippery Sam catheter in the vast majority of cats.

Intuitively, bladder lavage would seem to be a good idea in these cats to remove any blood, crystals, or other debris. Unfortunately, both studies examining this intervention failed to show any benefit (Dorsey et al. 2019, Tsuruta et al. 2022) though there were no significant complications reported. I will have my technician lavage the bladder if I am treating the patient as an outpatient or if the urine is grossly hematuric.

Perhaps one of the most interesting factors that may affect recurrence is the improvement (or lack thereof) in urine color from bloody to clear. Anecdotally, I have noticed that cats in the hospital whose urine quality visibly improves are more likely to promptly urinate when I remove their catheter compared to those who remain hematuric. While abnormal urine color at presentation was associated with more significant metabolic derangements (Brabson et al. 2015), visual improvement at the time of catheter removal *has* also been shown to correlate with rUO risk in the one study that examined it (Seitz et al. 2018).

No medication in UO management has sparked more controversy than prazosin. Since the original study that showed it reduced the risk of rUO compared to phenoxybenzamine (Hetrick and Davidow 2013), there have been three subsequent studies comparing prazosin to no treatment that have failed to find any benefit (Reineke et al. 2017, Hanson et al. 2021, Conway et al. 2022). The final study showed an *increase* in risk of rUO though admittedly, each of these studies has its flaws (Hearne 2023). As there has been no follow-up study showing a *benefit* to the administration of prazosin in this patient population, I do not prescribe it.

So, what is the overall risk for recurrent obstruction (rUO)? The honest answer is that it depends on what study you cite and what time interval you're evaluating, as it will range from 11 to 58%! I've included some of the data from the studies cited in this chapter in Table 13.1 that indicate a roughly 10% short-term and 20% medium-term risk for recurrent obstruction rate. I think these numbers for 24-hour, 7-day, and 30-day rUO are reasonably congruent with my clinical experience plus or minus a couple percentage points.

Table 13.1 Recurrent urethral obstruction (rUO) rates for cats managed on an inpatient basis.

Study	Number of patients	24-hour rUO (%)	7-day rUO (%)	30-day rUO (%)	6-month rUO (%)
Tsuruta et al. (2022)	72	—	6.60	21.80	—
Hanson et al. (2021)	65	—	—	25	—
Reineke et al. (2017)	47	6	—	16	34
Cooper et al. (2019)	34	6	—	—	—
Davidow (2020)	91	9	10	13	—
Pratt et al. (2020)	88	2	7	13	—
Hetrick and Davidow (2013)	192	11	—	24	—
Seitz et al. (2018)	46	—	—	11	—
Dorsey et al. (2019)	137	16	—	—	—
Reineke et al. (2021)	88	9	10	—	—
Perrucci et al. (2023)	200	18	—	—	—
Segev et al. (2011)	82	—	—	—	22
AVERAGE	—	10	8	18	28

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COMPLICATIONS OF INPATIENT MANAGEMENT

These cases are so common that inevitably you will end up troubleshooting the myriad complications they experience, either as a swing/overnight doctor or if you do daytime inpatient work.

The most common issue you'll encounter is your technician notifying you that the catheter isn't flowing or the urine output is low. Your first step should be to check bladder size, if it is big that either indicates the urine collection line/set or your catheter is obstructed. Secondly, I will usually flush the line with saline to see if it flows freely and if I can aspirate the same volume back. Sometimes you need a fair amount of pressure to dislodge an obstruction. You can also do a "bubble test" by injecting saline up the catheter while visualizing the bladder on ultrasound to verify patency and presence in the bladder. If you are unable to verify patency or if the catheter is not visualized in the bladder, you should remove it. If the bladder is small with decreased output, you should also evaluate the hydration status and fluid rate to make sure you are appropriately keeping up.

Another somewhat common complication is fluid or volume overload, where the cat develops either pulmonary edema or pleural effusion secondary to IV fluid administration. Beyond monitoring respiratory rate/effort, the onset of a new murmur or gallop sound is a red flag for impending overload (Ostroski et al. 2017) and should trigger either discontinuation or substantial reduction of IV fluid therapy. Patients at higher risk for developing fluid overload include those with higher potassium levels and azotemia (Duperrier-Simond et al. 2024). My feeling is that these patients receive more fluid under the assumption that the azotemia/hyperkalemia will correct faster, as both of the studies cited above demonstrated higher risk with the administration of a fluid bolus and higher initial fluid rate. While these patients *are* more likely to develop a post obstructive diuresis as we discussed above, there is some thought that our fluid therapy may also *drive* a persistent diuretic state (Fröhlich et al. 2016). My own personal practice in these cats is to start a mid-range fluid therapy (roughly 90 ml/kg/d) and monitor their urine output to make sure that they aren't producing exponentially more urine than that. To my knowledge, no study has shown that a higher fluid rate necessarily hastens the resolution of hyperkalemia.

Anemia due to urinary blood loss can occur in the sicker UO population (Beer and Drobatz 2016). That being said, the incidence of severe anemia is approximately 1% (Solari et al. 2024), and so while it is something to be aware of/monitor for, I have not had to transfuse a UO cat who has not undergone a perineal urethrostomy. However, a complication I *have* experienced (though it is reportedly much less common) is the intra-vesicular blood clot causing an obstruction (Keeshen et al. 2015). While I have tried using tissue plasminogen activator (tPA) similar to Young et al. (2021), I was not as successful as they were. I mention this complication only for you to consider in patients with severe hematuria who either obstruct their urinary catheter or who experience rUO in the hospital.

CONCLUSIONS AND CLIENT COMMUNICATION

Feline UO is a common emergency, and it is critical to always have a high index of suspicion for it in any sick male cat. Even cats that are critically ill can recover provided they receive appropriate treatment. Some communication points I relay to owners are as follows:

- Your cat has a UO, which means they cannot pass urine due to that blockage.
- This can lead to a life-threatening buildup of toxins and potassium in their blood, which can have adverse cardiac effects. While these are reversible with time and treatment, these cats are sicker in the short term.
- The most important treatment is to relieve the obstruction with a urinary catheter. The concern with this procedure is the need for sedation in a sick patient and the potential that we may not be able to successfully pass that catheter.

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- After the catheter is in place, we typically submit a sample of the urine to the lab to evaluate for infection and any potential cause for the obstruction. We also take radiographs to evaluate for any stones that may be present that might require future surgical management.
- Medical management for these cats typically involves maintaining an indwelling urinary catheter for 36–48 hours, as that reduces the short-term risk of a recurrent obstruction (though not to zero). For cats with an uncomplicated clinical course, roughly 20% will experience another obstruction within 4 weeks.
- Post hospitalization management will typically involve dietary change and environmental (i.e. litter box, water consumption) modification. Cats can experience future flareups of FLUTD that may or may not cause an obstruction.
- Since this can be a recurrent condition and some cats require surgery (perineal urethrostomy) to prevent recurrence.

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Approach to Vomiting and Diarrhea

CHAPTER 14

CRITICAL QUESTIONS

1. Is the cause of vomiting primary gastrointestinal versus extra-GI?
2. Is it a foreign body?
3. Do I need diagnostics?
4. Should I give antiemetics without imaging?
5. Can I induce emesis or scope to retrieve the gastric foreign body?
6. Do we still give metronidazole for diarrhea?
7. Do they need to be hospitalized?

INTRODUCTION

Vomiting and diarrhea are some of the most common presenting complaints in veterinary medicine because they are so outwardly visible, and it's inconvenient for owners to be constantly cleaning their house. Unfortunately, the range of underlying diagnoses ranges from benign to life-threatening and appropriate treatment can range from outpatient symptomatic care to emergency surgery! The goal of this chapter is to provide you with some tools in risk stratifying these patients so you can best advise clients as to who really needs a diagnostic workup and who would benefit from hospitalization.

NARROWING THE DIFFERENTIAL DIAGNOSIS

In the broadest terms, vomiting is either due to a disease directly affecting the gastrointestinal (GI) tract (peripheral) or a disease that indirectly causes vomiting through stimulation of the chemoreceptor trigger zone (central) (Elwood et al. 2010). Some common differential diagnoses for each category are listed in Table 14.1. While the use of diagnostic testing, such as imaging and lab work, is helpful in narrowing these down, in many cases you can refine your differential based on age, species, and signalment.

For dogs, the majority (roughly 59%) experience vomiting due to a problem within the abdominal cavity with most of those being a primary GI disease, such as dietary indiscretion (Rosé and Neiger 2013). That being said, as age increases, that study also showed that the likelihood of a systemic (or non-GI disease) increased as well. I was not able to easily identify a study looking at the epidemiology of vomiting in cats, though their differential list can be equally extensive (Batchelor et al. 2013). However, my own clinical impression is that while non-GI causes for vomiting become more common with age, it is still more common for the abdominal cavity to be the source for vomiting in the older feline due to an increased risk of IBD and GI lymphoma.

While there is a large amount of literature examining chronic diarrhea, there is much less looking at the underlying causes for acute large bowel diarrhea, which is what we see most commonly in the emergency room. This is likely because most of the time we do not obtain a definitive diagnosis for the cause of acute diarrhea, as it is either self-limiting or easily responds to medication, making it a difficult research subject. My very broad differential for acute diarrhea

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Table 14.1 Common GI and non-GI etiologies for vomiting in small animals.

Gastrointestinal diseases	Non-gastrointestinal diseases
Gastroenteritis	Peritonitis (septic, biliary, urine)
Gastrointestinal neoplasia	Hepatobiliary (cholangiohepatitis, neoplasia, mucocele)
Gastric/intestinal ulceration	Splenic disease (torsion, neoplasia)
Foreign body obstruction	Pancreatitis
Infectious (parvovirus, parasitic, etc.)	Intra-abdominal neoplasia
Inflammatory bowel disease (IBD)	Metabolic (renal, ketoacidosis, hepatic encephalopathy, Addison's, electrolyte)
Intussusception	Drug-induced
Mesenteric volvulus	Neurologic disease (vestibular, meningoencephalitis, neoplasia, vascular)
Dietary indiscretion/intolerance	—

Table 14.2 Broad differential diagnoses for acute large bowel diarrhea in small animals.

Disease category	Specific examples
Bacterial	Campylobacter, *Escherichia coli*
Viral	Canine parvovirus, feline panleukopenia
Diet related	Dietary indiscretion, food allergy
Parasitic	*Coccidia, Giarida*, hookworms, roundworms, whipworms
Neoplasia	Colonic, cecal
Acute hemorrhagic diarrhea syndrome	—
Systemic disease	Renal, hepatic, Addison's disease
Idiopathic	—

(with and without concurrent vomiting) is viral/bacterial, acute hemorrhagic diarrhea syndrome (AHDS), endoparasitism or giardia, or dietary indiscretion. I've included a more comprehensive list in Table 14.2, though the majority of these cases can be classified under one of the above differential diagnoses. For stable patients with strictly acute diarrhea, I do not tend to run a significant number of diagnostic tests except for perhaps a fecal exam with giardia and a parvoviral enteritis test in at-risk patient (read: young or unvaccinated) populations.

COULD IT BE A FOREIGN BODY?

When I speak to newer emergency veterinarians about the differential diagnoses for acute vomiting, invariably, the first one out of their mouth is foreign body obstruction, regardless of signalment, history, or physical exam. By that logic, every acute vomiting patient should have imaging of some sort, which in many hospitals is abdominal radiographs that are frequently equivocal. While we'll talk about the pros and cons of differing imaging modalities shortly, I think it's important to consider the prevalence and risk factors for foreign body obstruction so that we know who would most benefit from imaging.

Unfortunately, there aren't any real case-control studies that examine risk factors in history and physical exam comparing patients with and without foreign body obstruction. The only two studies I could find identified males, longer duration of clinical signs, and certain dog breeds as having an elevated risk of a foreign body (Luce and Hans 2022, Hayes 2009). My own anecdotal

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experience would indicate that major risk factors include abdominal pain and a previous history of foreign body surgery.

DIAGNOSTIC TESTING FOR THE VOMITING PATIENT

We'll first focus on the utility of various imaging modalities for diagnosing a foreign body obstruction and then evaluate how useful they are in evaluating the general population of vomiting dogs. We'll then briefly discuss the utility of lab work in achieving a diagnosis in the vomiting patient. The final goal is to combine history and physical exam findings to help classify vomiting patients as "sick" or "not sick" with those in the former category being those you should test in even if the results are nondiagnostic. Unfortunately, there are no peer reviewed guidelines I am aware of about which vomiting patients benefit from a diagnostic workup.

IMAGING AND LAB WORK IN THE ACUTELY VOMITING PATIENT

Classic radiographic parameters for a foreign body obstruction include visualization of a foreign body and two distinct populations of bowel with a distinctly dilated orad portion compared the aborad (Finck et al. 2014). While advantageous in terms of accessibility and convenience, its sensitivity and specificity are lacking compared to abdominal ultrasound and computed tomography (CT) (Corrick 2023). Even as tele-ultrasound becomes more and more available, its utility in diagnosing foreign body obstructions is currently still limited (Sombrio et al. 2023). I think further research on the use for point of care ultrasound in diagnosing foreign body obstruction would also be helpful.

There have been two studies evaluating the utility of diagnostic imaging in vomiting dogs. Radiographs either made or assisted in a diagnosis in 12% of cases, while ultrasound was useful in 33% of cases (Rosé and Neiger 2013). Five percent of the dogs in that study had a foreign body obstruction. The second study was relatively similar (8% utility for radiographs, 36% for ultrasound) and showed that ultrasound imaging was more helpful in patients who were not eating and had a longer duration of signs (Holzmann et al. 2023). The take-home message is that in the majority of your acute vomiting patients, be prepared that you likely won't find a treatment-altering diagnosis with imaging, especially using abdominal radiographs.

The same two groups cited above also evaluated the utility of lab work in achieving a diagnosis of vomiting. While lab work was more helpful in achieving a diagnosis (36–40%), that still leaves the majority of patients where it was unhelpful! (Rosé and Neiger 2013, Holzmann et al. 2023).

RISK STRATIFICATION TO GUIDE DIAGNOSTIC TESTING

The goal of this section is not to discourage you from doing tests in vomiting patients but more to show you that in the majority of cases, you will not find anything that changes your treatment approach. While there are no peer reviewed guidelines on who requires imaging and/or lab work, the expert opinion that is available identified the following risk factors to identify the "sicker" vomiting patient including frequent/large-volume emesis, weight loss, chronicity, marked lethargy, abdominal pain, hematemesis, melena, fever, dehydration, shock, and failure to respond to symptomatic management (Elwood et al. 2010, Batchelor et al. 2013).

While I offer and discuss diagnostic testing with the family of each vomiting patient, I explain the rationale for those tests, and we make a joint decision on its pros/cons for that particular animal. In general agreement with the above, risk factors for me that push me to strongly encourage diagnostics include history of foreign body ingestion/obstruction, abdominal pain, and severe dehydration or shock. These are patients that are either "sicker" or I perceive as higher risk for having a surgical lesion that would benefit from a timelier intervention. For imaging, as a primarily a

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"daytime" veterinarian, I am fortunate to have access to ultrasound during the majority of my working hours and so that is my primary imaging modality. However, when I speak with the junior veterinarians who do not always have access to ultrasound, my advice on abdominal radiographs is to only do them if the diagnosis would cause you to send them for emergency surgery after hours. My goal with that advice is to limit the number of equivocal radiographs that then require further imaging while maximizing the diagnostic yield for patients who require emergent surgical intervention, such as a gastric dilatation and volvulus (GDV) or mesenteric volvulus. We'll also discuss the consequences of "missing" a foreign body in the next section.

ANTIEMETICS AND "MISSING" THE FOREIGN BODY OBSTRUCTION

One dogma in emergency medicine is that unless you've ruled out an obstruction, you should not give antiemetics for fear of "masking" the progression of clinical signs due to a foreign body obstruction. However, this statement begs two questions. The first is, does that actually happen, and the second is, does that impact their prognosis? While antiemetic administration did increase the time to foreign body removal by roughly 24 hours, it was not shown to increase their complication rate (Puzio et al. 2023). In fact, it is more the total *duration* of clinical signs that can lead to an increased complication rate and more involved surgical procedures (Maxwell et al. 2021, Schoelkopf et al. 2023). My own personal practice is that I have no problem giving one dose of an antiemetic to any patient without imaging as long as I have discussed the potential for missing an obstruction. However, I am very clear with the owner that if they are still vomiting or not noticeably better (eating) within 24 to at most 48 hours, then they should be reevaluated. Inevitably, you will miss patients with foreign body obstructions, but as long as you are diligent about recommending appropriate follow-up, it is unlikely to be catastrophic.

CAN I AVOID SURGERY?

Besides the obvious financial advantages of avoiding surgery, there could be patient-related factors, such as age and comorbidities, that might make medical management beneficial. There are three ways to avoid abdominal surgery for a foreign body: induce emesis (gastric foreign body only), endoscopic retrieval (usually gastric foreign bodies), and passage through the intestinal tract.

We'll discuss how to induce vomiting in the toxicology chapter and focus more on its use to remove gastric foreign bodies here. For patients with a known gastric foreign body, the success rate for retrieval via emesis is roughly 75–80% in dogs and 50% in cats (Kirchofer et al. 2019, Zersen et al. 2020, Fischer et al. 2023). Ideally, emesis is performed within 2 hours, though it can still be successful later on, provided you know or suspect it is still in the stomach (Kirchofer et al. 2019). There were no reported complications, and even though I warn brachycephalic owners about the potential for aspiration, they have not been demonstrated to be at elevated risk for complications (Harkins et al. 2024).

The next nonsurgical intervention to consider is endoscopy. While most emergency clinicians do not scope themselves, it is important to consider the risk factors and chances for success to appropriately prepare owners for the potential of treatment failure. The overall success rate for endoscopic retrieval of a gastric foreign body is wide, depending on the study cited, anywhere between 56 and 94% (Poggiani et al. 2020, Gianella et al. 2009, Dollo et al. 2020, Barash et al. 2022, Šmit et al. 2018, Di Palma et al. 2022, Maggi et al. 2023). Unfortunately, none of these studies directly evaluated risk factors for procedural failure. My own personal experience is that foreign bodies that are most likely to fail endoscopy are the larger items, smooth/round objects, or those likely to shred (i.e. grass).

The final option for nonsurgical management is using fluid therapy, single-dose antiemetics, and time. The concept of medical management for foreign body obstruction was initially studied

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in needle foreign bodies and had a high success rate of 88% (Crinò et al. 2023). However, a broader study had a lower success rate of 47%, with linear foreign bodies, and those with a longer duration of signs being more likely to fail (Carrillo et al. 2024). In my experience, I will usually give owners a 50/50 shot with medical management for nonlinear foreign bodies. For cats with a linear foreign body lodged under the tongue with financial constraints, a reasonable proportion of these may be able to pass them just by clipping the stricture (Basher and Fowler 1987).

DO WE STILL GIVE METRONIDAZOLE FOR ACUTE DIARRHEA?

Despite a general lack of data on the underlying diagnosis for acute diarrhea, there have been multiple studies evaluating the best treatment protocol, including the controversial use of metronidazole.

Current veterinary literature, with the exception of one study, does not support the use of metronidazole for acute diarrhea (Ellis et al. 2023). While the review I just cited is excellent in terms of its breadth, the definition they use for acute diarrhea: "an episode(s) of diarrhea less than 10–14 days in duration (usually a few days)" is challenging to interpret because it doesn't address the presence/absence of hemorrhagic stool. While several of the studies specifically excluded dogs with evidence of AHDS (which I think is appropriate), I think lumping dogs with hematochezia along with those that don't as one disease group may be inaccurate. That being said, a more recent meta-analysis that did attempt to include disease severity, including hematochezia, still did not show a beneficial effect of metronidazole (Scahill et al. 2024). While my personal practice was to only use metronidazole (along with a probiotic or dewormer, depending on risk factors) in the presence of hematochezia to treat for possible *Giardia*, that position may have to evolve. Diet alone was also shown to be superior to metronidazole in one trial of canine acute diarrhea (Rudinsky et al. 2022), though that study was in all dogs with colitis. I generally only treat for 5 days, which may or may not obviate some concerns raised over disruption to the fecal microbiome. The use of pro/prebiotics is also reasonable based on existing evidence (Schmitz 2024), and I do incorporate them in outpatient management for acute canine and feline diarrhea management.

HOSPITALIZATION DECISIONS FOR THE NONSURGICAL PATIENTS

We previously discussed evaluating for the presence of a surgical lesion, and clearly those patients are ideally hospitalized postoperatively for monitoring and analgesia. However, this leaves us with a patient population who don't require surgery but may still benefit from hospitalization. Unfortunately, as we saw in Chapter 7, there are very few clinical guidelines or studies examining this issue, and so you'll be left with my own opinion and experience for this portion of the chapter. This section is geared toward the vomiting/diarrhea patient where you don't have an underlying diagnosis, i.e. the "nonspecific gastroenteritis," and may not be applicable for patients with a known diagnosis such as Addison's disease or renal insufficiency.

I think there are roughly three groups of factors that guide a hospitalization decision for the nonspecific vomiting and diarrhea patient: owner, patient, and disease. The first are owner factors that may include finances, emotional attachment, and the fact that it is inconvenient to be up all-night walking and cleaning up after their pet! While these are not the end-all be-all, discussing them with an owner can sometimes help joint decision-making for borderline cases.

The second broad category is patient factors that include age, comorbidities, duration of symptoms, and mentation. The very old or young patient does not have as much physiologic reserve or healing capacity, which puts them in a higher risk category for failing outpatient medical management. Similarly, those with other chronic diseases can also be more fragile, even with relatively benign presentations. For these patients, I will tend to err slightly more on the side of hospital admission rather than outpatient therapy. The next is the duration of signs. Patients with

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a longer history of GI signs and inappetence are likely more dehydrated and more malnourished, which both lend themselves to inpatient rather than outpatient management. Finally, a patient's mentation is important. A patient who is lethargic and less interactive in the exam room is likely one who is a bit sicker than they are letting on because their body is more tired than their history and physical exam might suggest. Over time, by interacting with hundreds of patients with the same presenting complaint, you will develop this clinical gestalt to separate out this patient population from the more straightforward outpatient one.

Finally, there are disease-related factors. The success of outpatient (and indeed inpatient) treatment for these animals is mostly predicated on two factors: replenishing fluid stores (rehydration) and reducing/stopping ongoing fluid loss. While conceptually simple, in practice, it can be more complicated, especially for patients with bad AHDS. We discussed fluid therapy in Chapter 4, but I'd like to put it in the context of clinical decision-making for the vomiting/diarrhea pet in the form of a case example. Say you have a 30 kg canine patient with a 24-hour history of vomiting and diarrhea that you assess as being approximately 5% dehydrated. That patient's fluid deficit (fluid deficit = body weight in kilograms × percent dehydration expressed as a decimal point) is roughly 1.5 l, and your goal is to rehydrate them over 12–24 hours (Pardo et al. 2024). With a maximum recommended dose of 30 ml/kg (I think most of us would likely give around 1 l of fluids to this pet), that would still leave 500 ml of that deficit behind. Now that's almost certainly okay for a dog who you expect to rally pretty quickly with minimal ongoing losses and a fast return to oral intake. But for the higher-risk patient we discussed above, where you *can't* rely on that recovery, it may be insufficient. Furthermore, if our patient is now closer to 7% dehydrated, that volume is now over 2 L and is not achievable with a single dose of subcutaneous fluids. These are the patients who would likely benefit from hospitalization to receive continued intravenous (IV) fluid therapy, where it is a lot easier to provide that volume over time (for reference, this would be the equivalent of 83 ml/h or 66 ml/kg/d). Clearly, for patients that you assess as shocky, IV fluid therapy and hospitalization should be recommended.

The second factor affecting the success of an outpatient treatment plan is your ability to reduce or, ideally, stop ongoing fluid losses. For most nonsurgical/nonspecific causes of vomiting, I've found that a single dose of maropitant is almost always effective at reducing or eliminating vomiting shortly after administration. Therefore, our discussion here is going to focus on AHDS, where medications may NOT dramatically reduce fluid loss in a timely manner.

ACUTE HEMORRHAGIC DIARRHEA SYNDROME

AHDS is characterized by the peracute onset of massive hemorrhagic diarrhea that can be associated vomiting and quickly leads to severe dehydration and hypovolemia (Unterer and Busch 2021). Current hypotheses surrounding its etiology mostly involve the role of *Clostridium perfringens* and associated enterotoxins (Busch and Unterer 2022). There is a spectrum of disease with the condition that can be described using the AHDS index (Table 14.3), and while this index is a helpful assessment tool, it does have some limitations.

Several of the categories are subjective, and later studies using it have all been in hospitalized patients, limiting its applicability to the inpatient/outpatient decision-making and antibiotic usage (Reisinger et al. 2024a, b, Dupont et al. 2021). If I were to use this score (I tend to use my own clinical judgment), I would likely recommend hospitalization for those patients with moderate to severe AHDS (score $\geq$ 6). Standard treatment for AHDS includes IV fluid therapy, antiemetics, potentially antacids, and as needed analgesics. The use of antibiotics in this patient population is controversial as it was for the use of metronidazole. While the concept of bacterial translocation has not been proven in AHDS (Unterer et al. 2015), we do know that the intestinal wall is compromised with this disease and does correlate with illness severity (Reisinger et al. 2024b). Indeed, many of the sicker patients who present with this disease have many characteristics of

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Table 14.3 Acute hemorrhagic diarrhea syndrome (AHDS) index.

Parameter	0	1	2	3
Activity	Normal	Mildly reduced	Moderately reduced	Severely reduced
Appetite	Normal	Mildly reduced	Moderately reduced	Severely reduced
Vomiting (times/day)	0	1	2–3	>3
Faecal consistency	Normal	Slightly soft	Very soft	Watery diarrhea
Defecation (times/day)	1	2–3	4–5	>5
Dehydration (%)	0	<5	5–10	>10
Total AHDS score	0–3	4–5	6–8	9 or greater
Clinical significance of disease	Clinical insignificant	Mild AHDS	Moderate AHDS	Severe AHDS

Source: Mortier et al. (2015) with permission of John Wiley & Sons.

sepsis, such as elevated C-reactive protein (CRP), elevated lactate, vital parameter derangements, and WBC abnormalities (Dupont et al. 2021, Mortier et al. 2015, Sänger et al. 2023). And so, while there has not been a demonstrated benefit to antibiotics in *every* AHDS patient (Reisinger et al. 2024a, b, Dupont et al. 2021), in both of these studies, the sicker patients still received antibiotics at clinician discretion. Until there is a randomized control trial showing no benefit to antibiotics in that sicker population, I will continue to give them (usually ampicillin-sulbactam), especially in neutropenic dogs who are at higher risk of a negative outcome (Dolan and Santo-Domingo 2025). This is roughly in line with current consensus guidelines strongly recommend against the use of any antibiotic therapy for all patients without evidence of severe disease or systemic inflammation (Jessen et al. 2024).

CONCLUSIONS AND CLIENT COMMUNICATION PEARLS

For the last bit here, I will give you my general approach to the GI patient from a diagnostic and therapeutic perspective (Figures 14.1 and 14.2) understanding there is no one-size-fits-all approach. I'll also give you how I speak to clients about these approaches to hopefully provide you with a basic conversation outline.

Client communication pearls:

- Vomiting is due to a disease that either directly affects the GI tract or one outside the GI tract that triggers vomiting as "bystander damage."
- Therefore, our diagnostic workup is geared at evaluating both of these broad categories through a combination of imaging (for the GI tract) and lab work (for outside the GI tract).
- However, for certain low-risk or stable patients, it is reasonable to treat symptomatically without diagnostics, understanding that if they fail to improve within 24–36 hours, they should return for a workup.
- While many patients with no evidence of systemic or obstructive disease can be managed symptomatically as outpatients, there are some higher risk patients who need to be hospitalized.
- For patients with non-hemorrhagic diarrhea, as long as they are eating/active, the majority can be managed conservatively without diagnostics using probiotics, diet, and time unless there are risk factors for endoparasitism in which case one can consider a fecal exam/Giardia test to guide deworming or metronidazole.

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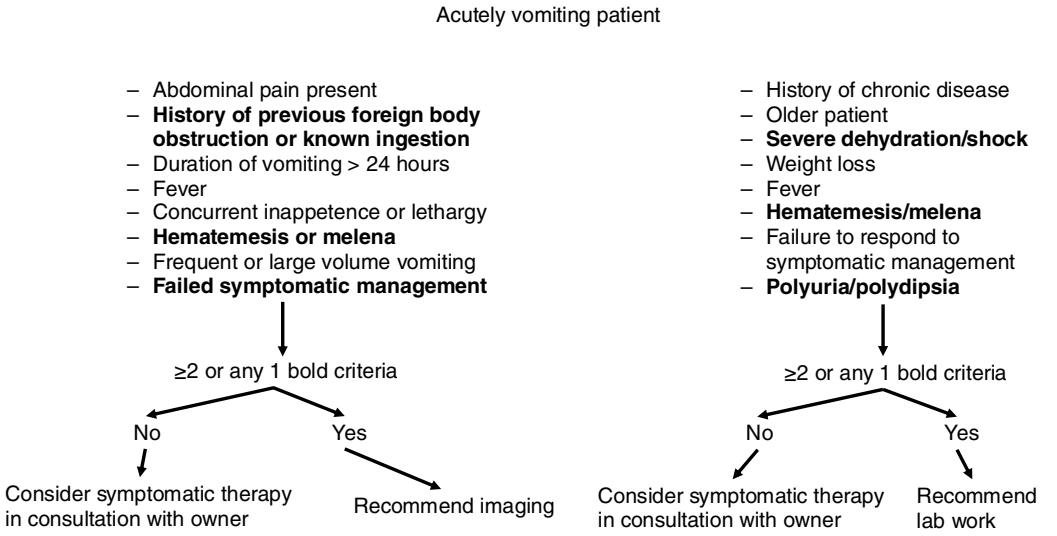


FIGURE 14.1 Potential decision tree for diagnostic testing in the acutely vomiting patient. Courtesy of the author.

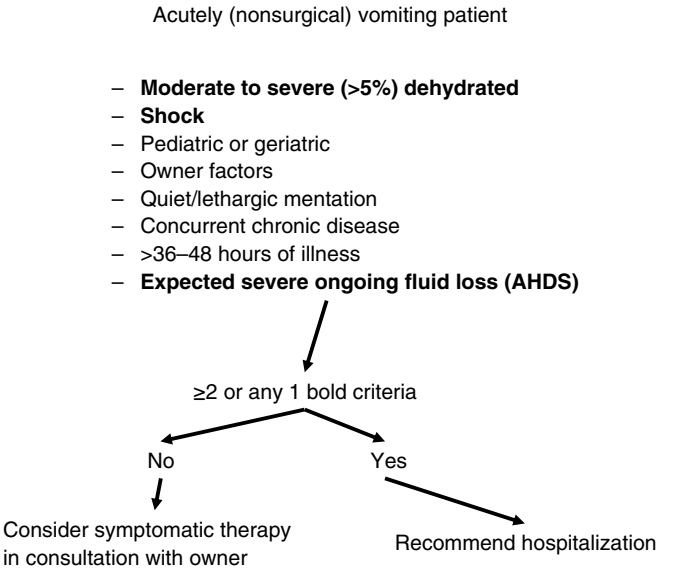


FIGURE 14.2 Potential decision tree for hospitalization of the nonsurgical, nonspecific vomiting patient. Courtesy of the author.

- Patients with hemorrhagic diarrhea who are stable can be trialed on conservative management unless there are risk factors for endoparasitism/giardiasis in which case I may consider empiric metronidazole.
- However, for patients with concern moderate to severe AHDS, their rapid fluid loss generally requires a diagnostic workup (labs +/− imaging) and hospitalization for fluid therapy and injectable medications.

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Dyspnea

CHAPTER 15

CRITICAL QUESTIONS

1. Is this upper airway, lower airway, or pleural space disease?
2. Is this cardiac or noncardiac dyspnea?
3. Do I need a chest X-ray?
4. Should I sedate this patient?
5. Should I intubate this patient?
6. What if I can't extubate an upper airway patient?
7. Is the oxygen cage adequate?

INTRODUCTION

Difficulty breathing is an incredibly common emergency because it is something that is very noticeable to owners. It can also be a frightening presentation for the junior clinician because its severity can range from trivial to peri-arrest and everywhere in between. My goal with this chapter is to provide you with a mental flowchart that you can apply to every dyspneic patient so that within 5 minutes of evaluating them, you can have (ideally) a diagnosis and a stabilization plan in action if required.

LOCALIZING THE DYSPNEIC PATIENT

The first important step in patient assessment is to localize whether this is a problem with the upper respiratory tract, lower respiratory tract, or pleural space, acknowledging that sometimes multiple areas can be affected. Furthermore, you should also be aware of the "non-respiratory lookalikes," such as shock, metabolic acidosis, along with stress/anxiety, and consider them *after* you've evaluated the other areas of concern. In broad terms, for dogs, roughly 33% of dyspneic patients will localize to the upper airway, 45% to the lower airway, and 19% to the pleural space (Fonfara et al. 2011), though this may vary depending on breed and location of practice. In cats, upper airway localization is less common with a plurality of cats having pleural effusion and/or cardiac disease (Dickson et al. 2018, Abboud et al. 2025). Proper localization is critical both from a diagnostic/prognostic standpoint but also because the emergent and definitive treatments vary so significantly.

The first step in dyspnea localization is to simply look and listen (without a stethoscope). If you can hear audible sounds when a patient is breathing (i.e. stertor or stridor), there is very likely an upper airway component with stertor more associated with nasopharyngeal or tracheal disease and stridor more associated with laryngeal dysfunction (Domínguez-Ruiz et al. 2021a). The presence of a paradoxical breathing pattern (Figure 15.1) has been highly associated with pleural space disease (Le Boedec et al. 2012), while inspiratory dyspnea is more associated with disease outside the thoracic cavity (Sigrist et al. 2011).

The next step in your evaluation should be brief thoracic auscultation for a heart murmur and any adventitious lung sounds. While the use of lung sounds can be helpful in localizing certain diseases (i.e. crackles with pulmonary parenchymal disease, wheezes with lower airway), there is not always agreement as to their presence/absence, and they can sometimes be masked by upper airway sounds (Domínguez-Ruiz et al. 2021b). Similarly, while the presence of a heart murmur is

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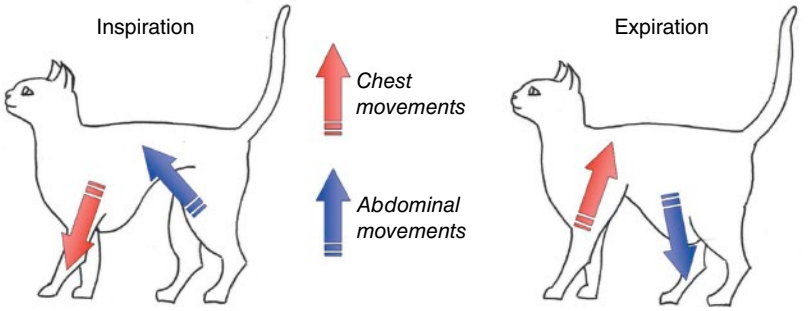


FIGURE 15.1 Paradoxical breathing pattern classically associated with pleural space disease. *Source*: Domínguez-Ruiz et al. 2021 with permission of Elsevier.

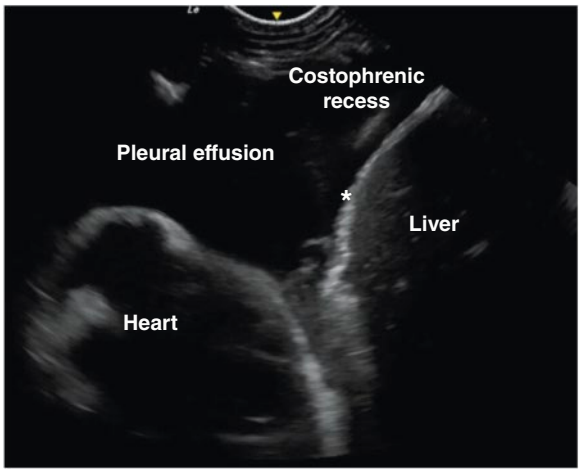


FIGURE 15.2 Pleural effusion outlining the heart and diaphragm (*). *Source*: Swanstein et al. (2024)/SAGE Publications.

suggestive of cardiac disease, it does not guarantee a cardiac source of a patient's dyspnea, and in cats, its absence does not rule out cardiac dyspnea.

We'll discuss the role of thoracic radiographs in the diagnosis of these patients later, but the next tool you should reach for is the ultrasound machine. The three diseases you can easily evaluate with an ultrasound exam are pleural effusion/pleural space disease, pulmonary parenchymal disease, and congestive heart failure (which we'll talk about in the next section). The *absence* of changes consistent with one of those disease processes can also be diagnostically helpful in guiding you either toward a non-respiratory lookalike or asthma in cats.

The first question the ultrasound can answer is whether or not pleural effusion is present (Figure 15.2).

For a truly dyspneic or unstable patient, the vast majority of the time, the effusion (if present) will not be subtle. We'll review the different scanning protocols below, but for the emergent/unstable patient, your initial ultrasound should be to simply just put the probe on the chest and answer the question, "pleural effusion: Yes, or no?" and if the answer is yes, you should proceed directly to thoracocentesis. Even though the sensitivity and specificity for the detection of pleural effusion with ultrasound when compared to CT is moderate (Walters et al. 2018, Cole et al. 2021, Mahfouz 2023), if it is a major contributor to your patient's dyspnea, you'll see it. While point of

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care ultrasound can be useful in detecting a pneumothorax using either "glide sign" or "curtain sign" as seen in Figure 15.3, its accuracy in novice hands is imperfect (Boysen, 2021, Lisciandro et al. 2008, Walters et al. 2018, Cole et al. 2021, Mahfouz 2023, Vidal et al. 2024).

For a demonstration of curtain sign, view the videos from Boysen et al. (2019). I personally do not search for these signs (though it isn't wrong to try!) because in the emergent patient with a reasonable risk for pneumothorax, I will likely empirically tap the chest regardless of ultrasound findings, as discussed in the trauma chapter.

Ultrasound can also be helpful in answering the question: Are the lungs part of the problem? I will frequently use ultrasound to evaluate whether or not a thoracic radiograph is likely going to be helpful in diagnosing the cause of cough or dyspnea. While there are several described lung ultrasound protocols, including the 4-quadrant (Rademacher et al. 2014) and sliding ultrasound techniques (Armenise et al. 2019), probably the most commonly utilized protocol for examining the lungs is VetBlue (Figure 15.4).

The goal of this exam is to comprehensively evaluate the lungs to detect either fluid-filled or consolidated lung (Figure 15.5), known collectively as alveolar-interstitial syndrome or AIS. AIS comprises the following common diseases: cardiogenic pulmonary edema, noncardiogenic pulmonary edema, pneumonia, and neoplasia.

When combined with your physical exam and signalment, the presence and distribution of abnormal lung findings are powerful in providing a provisional diagnosis. A recent review article (Lisciandro and Lisciandro 2021) has some excellent flowcharts on correlating lung ultrasound findings with a clinical diagnosis. However, it is important to recognize that this imaging modality is not perfect (reviewed in Gajewski 2024) with only moderate accuracy for detecting any noncardiac pulmonary changes (Lin et al. 2020, Cole et al. 2021, Łobaczewski et al. 2021) and a sensitivity/specificity of 60/65% for detecting metastatic nodules (Pacholec et al. 2021).

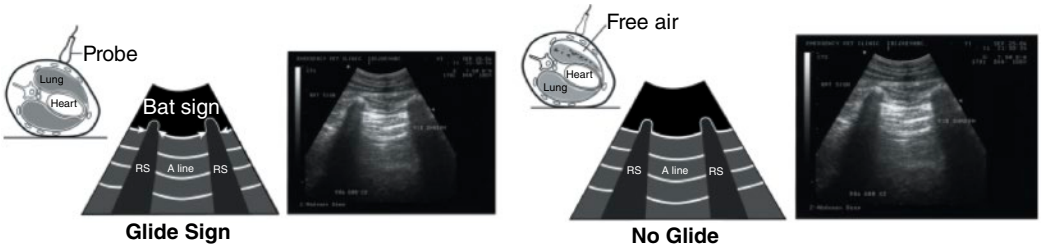


FIGURE 15.3 The absence of glide sign can indicate the presence of a pneumothorax. *Source*: Lisciandro (2011) with permission from John Wiley & Sons.

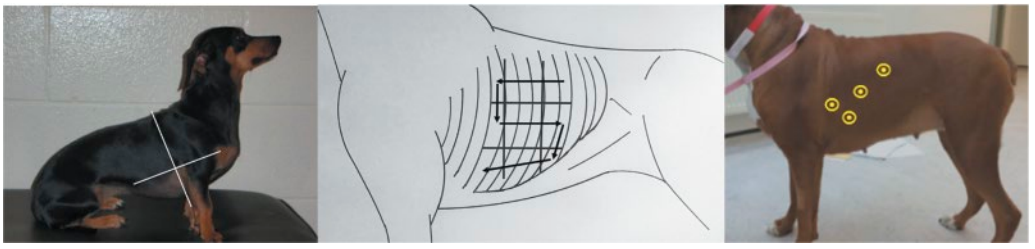


FIGURE 15.4 Three different lung ultrasound protocols: (a) 4-quadrant. *Source*: Rademacher et al. (2014)/John Wiley & Sons; (b) sliding. Armenise et al. (2019); (c) VetBlue. *Source*: Cole et al. (2021)/John Wiley & Sons.

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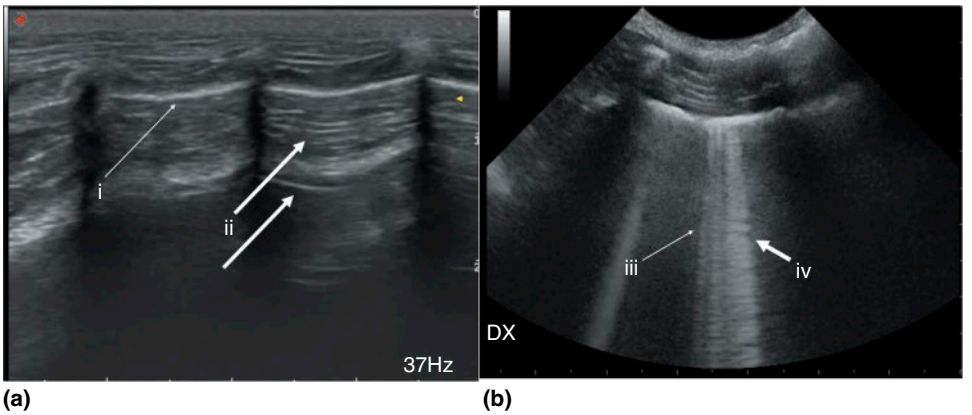


FIGURE 15.5 (a) Normal lung on ultrasound showing (i) pleura and (ii) A-lines; (b) fluid filled lung with (iii) single B-lines and (iv) confluent B-lines. *Source*: Armenise (2025)/MDPI/CC BY 4.0.

Finally, the absence of ultrasonographic lung changes in a dyspneic animal (provided your localization is correct!) can also be helpful. Diseases that could cause dyspnea with minimal to no visible pulmonary parenchymal changes could include feline asthma, pulmonary hypertension, pulmonary thromboembolism, elevated temperature, and shock/metabolic acidosis.

IS THIS LEFT SIDED CONGESTIVE HEART FAILURE?

Prompt identification of left sided congestive heart failure (L-CHF) is critical both from a therapeutic perspective (you can start giving furosemide) and prognostic standpoint (you can immediately talk to the owner about workup, treatment, and prognosis). While we'll discuss the management of congestive heart failure in a later chapter, since almost all L-CHF patients present dyspneic, it seems appropriate to discuss the diagnosis here.

While traditional diagnosis of L-CHF required the demonstration of cardiogenic pulmonary edema on thoracic radiography, there are several downsides to this approach. You frequently can't get radiographs done in a timely manner, there are risks in restraint or sedation to obtain adequate images, and they're expensive. My own opinion, which I hope you'll share at the end of this section, is that in the vast majority of these cases, chest radiographs are unnecessary for diagnosing L-CHF.

As discussed above, the first step in assessing the potential L-CHF patient is evaluating for a heart murmur and any pulmonary crackles. While these signs are suggestive of L-CHF, it's important to acknowledge the limitations of the physical exam. Sometimes it can be difficult to auscultate a heart murmur over airway sounds, and so I will sometimes palpate the apex beat to try and detect a thrill as the majority of dogs (but not cats) in L-CHF will have a substantial heart murmur you may be able to feel. Interestingly, in dogs, the use of the shock index (heart rate/systolic blood pressure) was also helpful in detecting L-CHF with a cutoff of 1.1 yielding a sensitivity of 92% and specificity of 93% in dogs with degenerative mitral valve disease (DMVD) (Matson and Llewellyn 2024). Finally, not all cats in L-CHF will have a heart murmur or gallop sound, so the absence of an audible murmur should NOT rule out L-CHF if there are other factors that might support that diagnosis.

As we discussed above, after your brief physical exam, we move to imaging. Without using chest X-rays, the diagnosis of L-CHF with point of care ultrasound should include the following criteria: evidence of pulmonary edema (or pleural effusion in cats) on lung ultrasound and demonstration of an enlarged left atrial:aortic ratio (La:Ao). For borderline cases or those where you are

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struggling to get an adequate La:Ao, point of care cardiac biomarkers can also help raise or lower your index of suspicion.

Cardiogenic pulmonary edema will appear as "wet lung" or B-lines on lung ultrasound. The presence of greater than or equal to three B-lines at more than two anatomic sites (Figure 15.6) was fairly predictive of L-CHF, especially in cats (Ward et al. 2017). That study did not use or evaluate the La:Ao in diagnosing L-CHF, which as we'll see, does increase the accuracy of your point of care ultrasound diagnosis.

Obtaining an accurate subjective La:Ao is a core skill for the emergency clinician but is one that requires substantial practice and repetition to achieve proficiency. While newer emergency doctors and general practitioners often feel uncomfortable obtaining this view, with repetition and, if necessary, a formal training course, it is a skill you can master (Tse et al. 2013, Darnis et al. 2019, Dickson et al. 2022, McDonald et al. 2023, Valcke et al. 2024). How I teach junior doctors is to first obtain the short axis parasternal view or mushroom view (Figure 15.7a) with the patient standing

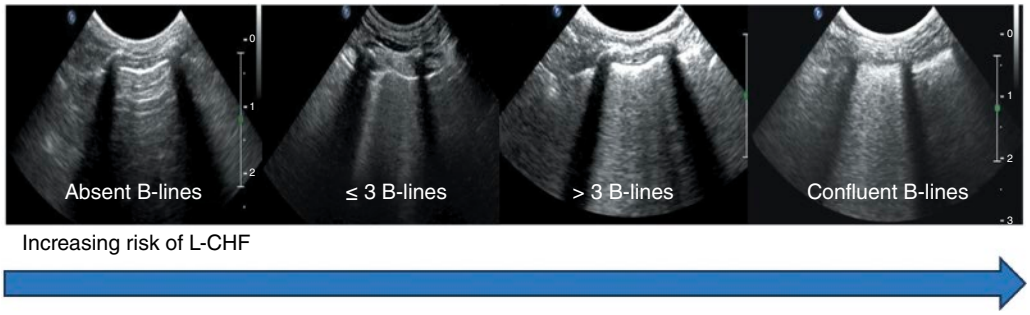


FIGURE 15.6 Increasing number of B-lines on lung ultrasound correlates to an increased risk of L-CHF when found bilaterally. *Source*: Lam et al. (2022)/John Wiley & Sons/CC BY-NC 4.0.

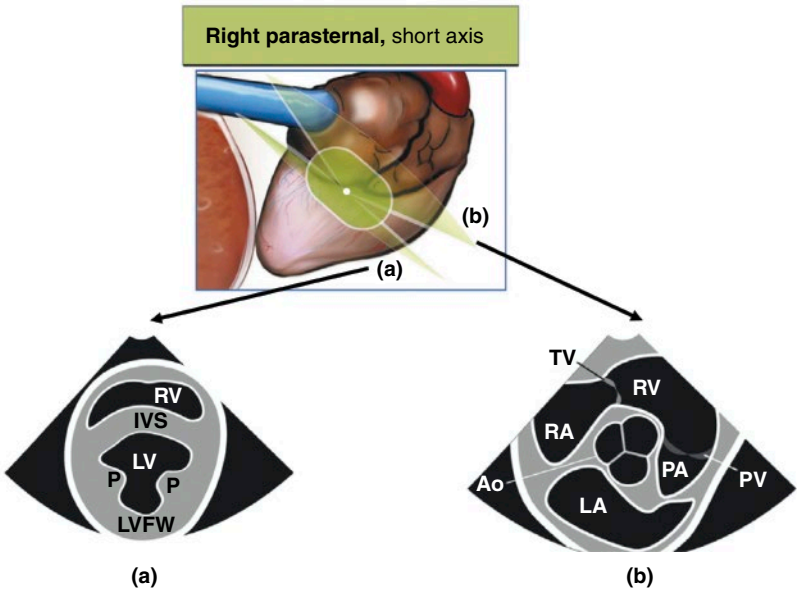


FIGURE 15.7 Obtain the La:Ao by fanning your probe from a to b. *Source*: DeFrancesco et al. (2021) with permission of Elsevier.

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Table 15.1 Mental checklist when assessing a patient for L-CHF. The more items a patient checks, the more likely they have L-CHF.

Checklist item	Present?
Appropriate signalment/history (older pet? Known cardiac disease?)	
Presence of loud heart murmur?	
Evidence of ≥3/confluent B-lines bilaterally or pleural effusion (cats)?	
Evidence of left atrial enlargement?	

or in sternal recumbency and then drop your wrist ventrally (fanning) until you obtain the La:Ao (Figure 15.7b).

Obtaining the La:Ao view is the first step, and then you have to interpret what you see. While you can certainly measure the La:Ao quantitatively with a value greater than 2 correlating to left atrial enlargement, depending on how good your image is, you may or may not obtain an accurate number. I will typically usually subjectively evaluate the La:Ao as normal or moderate/severe with the idea in my head of: "Is this big enough that L-CHF is a reasonable differential diagnosis?" In dyspneic dogs, the presence of left atrial enlargement (La:Ao > 2.1) was substantially associated with a cardiac diagnosis even though most dogs were correctly diagnosed based on history and physical exam alone (Hezzell et al. 2020). In dyspneic cats with experienced emergency clinicians, the presence of subjective left atrial enlargement was 100% specific and 97% sensitive for L-CHF (Ward et al. 2018), and integrating the La:Ao dramatically improved diagnostic accuracy in a similar study (Janson et al. 2020). The presence of pericardial effusion in cats was almost exclusively associated with L-CHF (Ward et al. 2018).

Finally, while point of care biomarker tests exist for cardiac disease (nt-proBNP), depending on your hospital and location they may or may not be available. In dogs, you cannot rely on a single point of care measurement to always differentiate between dogs with and without L-CHF (Riengvirodkij and Sakcamduang 2021, Chanmongkolpanit et al. 2024). Similarly, the use of a single point of care measurement was helpful but not perfect in diagnosing L-CHF in dyspneic cats with or without pleural effusion (Hezzell et al. 2016, Wurtinger et al. 2017, Ward et al. 2018) and the inclusion of that value only mildly improved diagnostic accuracy (Janson et al. 2020).

To summarize this section, I've included a checklist (Table 15.1) for approaching and diagnosing the potential L-CHF patient.

SO WHY DO I NEED A THORACIC RADIOGRAPH?

At this point you're probably wondering why you would ever need to do a chest X-ray! And while I think in many scenarios (the first time L-CHF) you probably don't, there are always exceptions to every rule.

As you likely noticed above, no *single* test has a 100% diagnostic accuracy. Even in L-CHF, it is the *combination* of test results and clinical information that yields the diagnosis. Furthermore, lung ultrasound may detect abnormal lung, but you may not have enough contextual information to say whether or not those changes represent pneumonia, neoplasia, or some other pathology. A thoracic radiograph is frequently helpful in making those differentiations. Similarly, it is not uncommon for dogs with mitral valve disease (even those with left atrial enlargement) to have concurrent lower airway disease that may mimic early L-CHF on lung ultrasound (Lam et al. 2022). For more inexperienced operators, this may lead to an inaccurate diagnosis of heart failure when, in fact, a thoracic radiograph might show changes more consistent with lower airway disease requiring a different treatment. Finally, as we discussed in the trauma section, having a thoracic radiograph can be helpful at identifying a myriad of concurrent non-pulmonary injuries that may or may not be easily visible on ultrasound.

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In conclusion, if after going through the steps listed above you still feel like you only have a tentative diagnosis and the patient is stable enough for a radiograph, then by all means proceed.

STABILIZING THE UPPER AIRWAY PATIENT

Patients with an upper airway crisis are understandably distressed because they cannot move an adequate amount of air into their lungs, which, at its extreme, leads to asphyxiation. Therefore, our stabilization goals are to decrease that distress through appropriate sedation and reestablishing a patent airway to improve ventilation using endotracheal intubation if necessary.

SEDATION FOR THE UPPER AIRWAY OBSTRUCTION

There is no one perfect drug for stabilizing these patients, and no studies exist that compare different protocols in patients presenting in an upper airway crisis with the majority of available information as expert opinion (Grubb 2010, Maczuzak and Silverstein 2015). For very mild cases, you can consider oral sedation such as gabapentin/trazodone, understanding that close monitoring is imperative to make sure they don't deteriorate. For patients requiring parenteral medications, the three most common options are butorphanol, acepromazine, and dexmedetomidine. For most clinicians (including myself), butorphanol is usually the first-line agent due to its relatively low impact on cardiovascular and respiratory parameters and moderate degree of sedation. My dose range is typically 0.2–0.4 mg/kg, depending on route of administration (IV versus IM) and degree of crisis. However, butorphanol frequently is inadequate at providing the required level of sedation and needs to be combined with another agent. Traditionally, the next agent in line was acepromazine due to its sedative abilities, acknowledging that it is nonreversible and can cause hypotension. And while this is a reasonable choice, I have transitioned away from acepromazine in most straightforward upper airway cases in favor of dexmedetomidine. Dexmedetomidine is an excellent sedative, is reversible, and will maintain a patient's respiratory status. In one veterinary study (albeit in more "stable" elective patients), brachycephalic dogs who received dexmedetomidine were more sedate and extubated faster with no increased complications when compared to acepromazine (Petruccione et al. 2021). My typical starting dose for dexmedetomidine is 0.001–0.004 mg/kg, depending on patient age (younger patients tend to need more), route of administration, and degree of distress. As discussed above, there have been no efficacy trials comparing these drugs for this patient population, and so my recommendations are solely "expert" opinion.

INTUBATION FOR THE UPPER AIRWAY OBSTRUCTION

Patients who fail to improve (or deteriorate) despite your sedation are ones who require endotracheal intubation to reestablish a patent airway. However, it is critically important to be prepared that intubating these patients can be challenging due to the presence of airway secretions and oropharyngeal swelling causing altered anatomy (Figure 15.8).

Frequently, you will only be able to place a smaller-than-normal endotracheal tube, which is okay in the short term. When it comes to airway diameter, even a small improvement can make a big difference (due to Poiseuille's law).

FALLBACK FOR A "FAILED AIRWAY"

For patients that you are unable to intubate, you have to be prepared for a surgical airway either through a tracheostomy (as shown in Lam 2015) or cricothyroidotomy (Hansen and Eriksen 2014, Hardjo 2021a both have good descriptions of this procedure, Delle Cave et al. 2024). The timing of when to initiate a surgical airway has not been fully defined in veterinary medicine, but I've included what I would consider reasonable criteria from the literature in Table 15.2.

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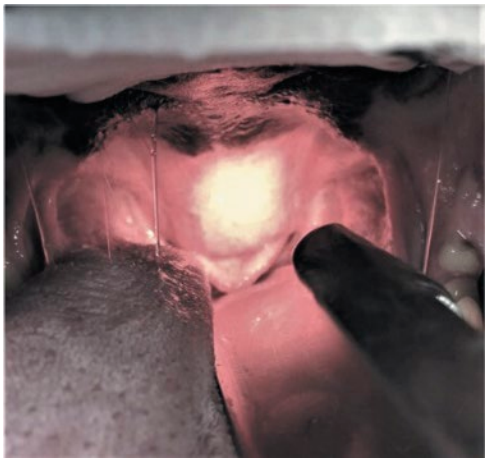


FIGURE 15.8 Upper airway disease, in this case, an elongated soft palate may obscure local anatomy, increasing the risk of failed intubation. *Source*: Sun et al. (2022) with permission of John Wiley & Sons.

Table 15.2 Recommended indications for surgical airway, such as cricothyrotomy or tracheostomy.

1 – Procedural indications	2 – Status indications: supercedes procedural indications
a. Three unsuccessful attempts at orotracheal intubation by a suitably qualified veterinarian	—
b. 10 minutes elapsed since anaesthetic induction without confirmed airway intubation	**a.** Rapid desaturation during orotracheal intubation attempts
c. Oxygen saturation falls below 75% (or the patient becomes cyanotic) at any point after the first or second intubation attempt	**b.** Criticalist, anaesthetist, or other suitably qualified veterinarian decides further attempts at orotracheal intubation would be futile
d. 2 minutes from the commencement of CPR without confirmed airway	**c.** Hypoxemic patient arrests during management of difficult airway.
(non-respiratory causes of arrest)	

Source: Hardjo et al. (2022)/MDPI/CC BY 4.0.

Between the two surgical procedures, the cricothyrotomy is faster (Hardjo et al. 2021b) and maybe technically easier, though it is less commonly taught than tracheostomy.

Once you've appropriately sedated your patient and improved airway patency, make sure to check a temperature, as many of these patients are hyperthermic and require active cooling. Other ancillary treatments are corticosteroids (anti-inflammatory dose), fluid therapy, and antiemetics. For patients where an underlying pneumonia may have triggered or potentiated their crisis, I will also consider antibiotics.

FALLBACK PLANS FOR EXTUBATION FAILURE

If you've had to intubate your patient, at some point you'll have to try and extubate them (unless you plan on ventilating them, which typically is unnecessary and resource-intensive). Depending on the underlying etiology and severity, the success of extubation will vary. For many patients with an uncomplicated laryngeal paralysis or brachycephalic obstructive airway syndrome (BOAS) crisis, I will leave them intubated for 1–2 hours with the hope that enough of the airway edema and swelling goes down enough to allow extubation. While some clinicians may advocate the placement of a temporary tracheostomy prior to extubation in BOAS patients, I have not found that to be necessary in the majority of patients. Again, there are no studies comparing these approaches, and unfortunately, the

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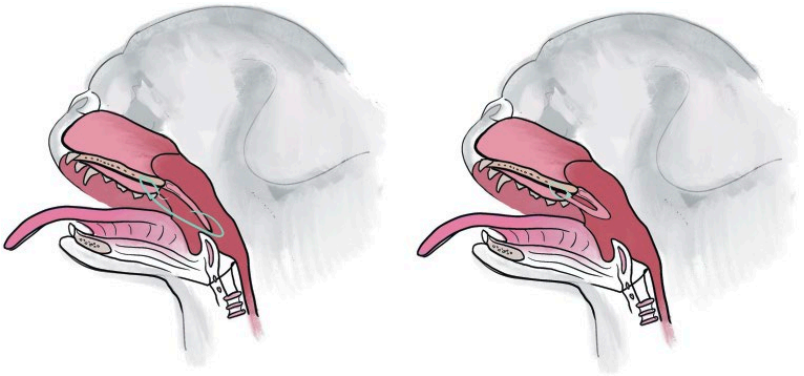


FIGURE 15.9 Temporary palatopexy may allow successful extubation of a BOAS crisis patient. *Source*: Sun et al. (2022) with permission of John Wiley & Sons.

majority of the literature deals with their perioperative, not emergent, management. Theoretically, if you've "broken" the crisis by reducing edema and thus improving airflow, these patients will go back to their baseline. Unfortunately, for a portion of these patients, their baseline is tenuous at best and so it's important to be prepared and have a backup strategy.

For the BOAS patient who fails to extubate, your options besides emergent airway surgery (which may or may not be available) are re-intubate and ventilate until surgery, perform a temporary tracheostomy, or a temporary palatopexy. Of these, the last option (palatopexy) is the least invasive and can be successful in many dogs, provided they do not have other conformational airway abnormalities such as laryngeal collapse or significant tracheal disease (Sun et al. 2022). While the study cited has some good schematics (Figure 15.9), there is also a recent video from JAVMA that also well illustrates the procedure (Sun 2025).

Continuous positive airway pressure (CPAP) was shown to reduce re-intubation in one study of postoperative BOAS patients and maybe an option if available (Vicenti et al. 2024).

Unfortunately, there aren't many nonsurgical or procedural interventions for patients with tracheal collapse or laryngeal paralysis who fail extubation. For patients with tracheal collapse, intraluminal tracheal stenting (it may be necessary to transport the patient intubated) is likely the best recommendation (Beal 2013). In dogs with laryngeal paralysis who fail extubation, options are either emergent tie-back surgery, ongoing intubation pending surgery/referral, or temporary tracheostomy (Karbe 2015, MacPhail 2020). However, a temporary tracheostomy in these patients has been associated with a significant increase in complications and mortality rate (Macphail and Monnet 2001). Even though there is likely some selection bias present in that study (the sicker patients are the ones requiring tracheostomy), it is important to educate owners about the increased risk if you decide to do that procedure.

OXYGEN THERAPY AND FALLBACK OPTIONS

With the exception of patients with head trauma (due to hyperoxemia), oxygen therapy is unlikely to worsen most clinical syndromes. The important exception is that if the provision of oxygen (usually via flow-by or mask) is causing increased patient stress and thus work of breathing, which is counterproductive. As many of you know, the goal of oxygen therapy is to increase the concentration of oxygen inspired to improve gas exchange and thus blood oxygenation.

The provision of standard oxygen therapy is well covered in many other resources (Boyle 2012), and so I won't cover it in detail here. The important part is recognizing when conventional oxygen therapy is *failing* and intervening appropriately. Some criteria for "failing" conventional therapy are a persistently abnormal respiratory effort/dyspnea, an elevated respiratory rate of greater than 40 breaths per minute, SpO_2 < 92–95%, or a PaO_2 < 70 mmHg despite $HFNO_2$

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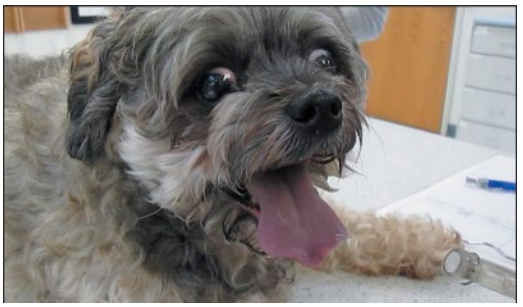


FIGURE 15.10 Signs of impending respiratory fatigue and failure. Note the almost panicked look in this dog's eyes and cyanotic tongue. Andrew Linklater DVM DACVECC.

(Jagodich et al. 2020, Pouzot-Nevoret et al. 2019, Frischer et al. 2023). However, I think it's also important to understand the visual signs of impending respiratory fatigue and failure. These dogs and cats will remain standing, unwilling to settle, orthopneic, with persistent respiratory effort and almost this panicked look in their eyes (Figure 15.10).

I think it's critically important that you intervene before the patient reaches an irreversible tipping point and goes into respiratory arrest.

For patients who have failed conventional oxygen therapy, there are two main options: high-flow nasal oxygen ($HFNO_2$) and mechanical ventilation. In the past decade, there have been multiple studies evaluating the efficacy of high-flow nasal oxygen therapy in both dogs and cats (Krawec et al. 2022, Hopper and Powell 2022, Her et al. 2024, reviewed in Whitney and Keir 2023). Ultimately, my experience is that it is most valuable when employed early, though it does not always stave off the need for additional ventilatory support. Demonstrated risk factors for failure of $HFNO_2$ are either a high respiratory rate-oxygenation index or a low ratio of pulse oximetry saturation to the fraction of inspired oxygen (SpO_2/FiO_2) (Duble et al. 2024, Eicher et al. 2024). It's important to recognize that some patients may require some level of sedation (typically butorphanol and/or low-dose dexmedetomidine CRIs) to tolerate this therapy. For reference, the following list provides a brief overview of machine parameters when starting high-flow nasal oxygen:

Sedation
- Only if required
- Based on patient assessment and comorbidities
- Butorphanol and/or α_2-agonists commonly used

Nasal prongs
- Occlude <50% opening of nares
- Suture to keep in place

Initial settings
- FiO_2: 100%
- Flow rate: 1–2 l/kg/min
- Temperature: 37 °C

Monitoring
- Continuous ECG
- Respiratory Rate, respiratory effort, SpO_2 every 1–2 hours
- PaO_2 and $PaCO_2$ at least every 12 hours

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Weaning

- Reduce FiO_2 by 5–10% every 1–2 hours based on SpO_2
- Reduce flow rate after stable RR and SpO_2 on 40% FiO_2 for 12–24 hours
- Consider discontinuing when FiO_2 < 30–40%

Indications for escalation

- No improvement after 1–2 hours of high FiO_2 and rate
- Persistent increased work of breathing
- Hypercapnia/hypoventilation

It would be impossible to cover the vast breadth of mechanical ventilation in this chapter. If you find yourself needing to intubate a patient and don't have or feel comfortable with a "true" ventilator, consider using an anesthesia ventilator as a temporizing measure. While you can look up some initial ventilator settings (Hopper and Powell 2013, Balakrishnan 2021), one reasonable option is to set roughly 8–10 ml/kg tidal volume with a 5 or 10 cm H_2O PEEP valve at a respiratory rate of roughly 10 breaths per minute. And then either refer to a place with mechanical ventilation or set up your own ICU ventilator.

However, there is one part about mechanical ventilation I do think is important here, which is to briefly cover the prognosis for patients requiring both high flow and mechanical ventilation. For patients requiring high flow, depending on the study cited, survival to discharge was between 36 and 66% with 27% requiring escalation to ventilation and between 27 and 54% passing away (Guieu et al. 2024). For mechanical ventilation, the prognosis is heavily diagnosis dependent (i.e. cardiac cases may do better than primary pulmonary) but overall is low at around 25% (Oppenheimer et al. 2022, Cagle et al. 2022).

CONCLUSIONS AND "MY" APPROACH

Promptly and accurately diagnosing the source of a patient's dyspnea is a core skill for the emergency clinician and requires the integration of a patient's signalment, history, physical exam, point of care ultrasound findings, and sometimes thoracic radiography. I've created a summary figure (Figure 15.11) that outlines my own diagnostic and therapeutic approach to these cases.

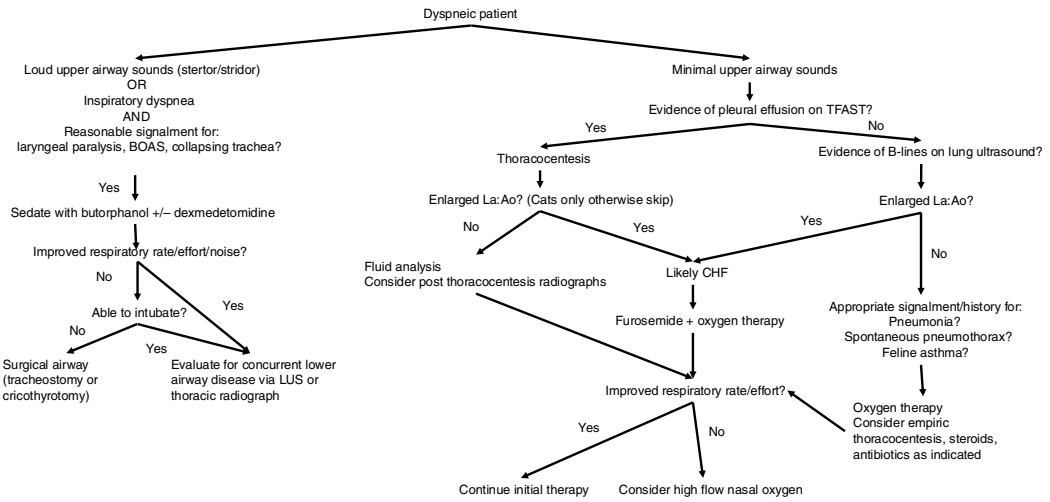


FIGURE 15.11 Flowchart for initial diagnosis and management of the dyspneic pet. Courtesy of the author.

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Neurologic Emergencies

CHAPTER 16

CRITICAL QUESTIONS

1. Is this likely an intracranial or extracranial source of seizures?
2. Should I start an anticonvulsant and which one is best?
3. Do I need an MRI and CSF sample for seizures in this patient?
4. How do I escalate anticonvulsant treatment?
5. Is this idiopathic old dog vestibular disease or something different?
6. Does this neck/back dog need an MRI and surgery?

INTRODUCTION

Neurologic emergencies can be scary both because they're very visible (unable to walk, actively seizing) and because every neurologist will manage them slightly different, which can make your life as an emergency clinician more challenging. In this section, we'll examine several common emergency presentations of neurologic disease and review some of the typical decision points you'll face in their management.

THE FIRST-TIME SINGLE SEIZURE PATIENT

Owners will frequently bring their pets in through the emergency room for a first-time single seizure event because it is such an obvious and scary symptom. When considering diagnostic testing (i.e. lab work and imaging), it's important to consider the likelihood of finding something abnormal. There is limited data about the diagnostic testing in the single, isolated seizure patient. My own personal practice is that unless there are risk factors for hypoglycemia, a portosystemic shunt, or other concurrent systemic symptoms, I will discuss but not strongly push screening lab work. There is likely no role for advanced imaging in the single, isolated first-time seizure patient. It is important to educate owners that while a reasonable percentage of dogs (likely around 20%) will never have further seizure activity ever, that still leaves many that will, usually within the first 3 months (Fredsø et al. 2016b, James et al. 2022) after their first event.

Starting an anticonvulsant is not currently recommended for a single, isolated seizure with recommendations for initiation listed as follows (Bhatti et al. 2015, Podell et al. 2016). We'll discuss more about the pros and cons of each anticonvulsant in a later section.

- Interictal period of $\leq$ 6 months (i.e. two or more epileptic seizures within a 6-month period)
- Status epilepticus or cluster seizures
- Severe or prolonged postictal signs
- Shortening interictal interval
- Increasing seizure duration or severity

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TREATING THE FIRST-TIME CLUSTER SEIZURE/STATUS EPILEPTICUS PATIENT

More commonly, you'll see a patient who is either having a cluster seizure event or in status epilepticus. The definition of a cluster seizure event is greater than two seizures within a 24-hour period, while status epilepticus is defined as greater than two seizures without complete interictal neurologic recovery OR a seizure lasting greater than 5 minutes (Berendt et al. 2015, Charalambous et al. 2024). We are starting with the treatment discussion prior to diagnostics because the most important stabilizing treatment you can perform is to stop ongoing and reduce future seizure activity.

Overall, the biggest questions we have to consider in the management of these patients are as follows:

1. How do I stop the seizures on an emergent basis? What if that doesn't work?
2. Which anticonvulsant is best?

INITIAL STABILIZATION

It's safe to say that most veterinarians would administer an intravenous benzodiazepine (diazepam or midazolam) as the first-line medication to terminate an active seizure as is currently recommended (Charalambous et al. 2024). Alternative routes of administration, if you cannot get intravenous access, would include intranasal or intramuscular midazolam in dogs (Charalambous et al. 2019, Charalambous et al. 2021) with limited evidence to support its use in cats. Between diazepam and midazolam, no clinical study has compared efficacy for intravenous administration, and in all likelihood, they are fairly equivalent. For patients whose seizure DOESN'T terminate with a single bolus dose, the first thing to ensure is that (i) the patient isn't hypoglycemic and (ii) the patient is actually seizing, not tremoring. If you feel comfortable that the patient is indeed having an ongoing seizure and it has been greater than 2 minutes since the first benzodiazepine bolus, then you can give a second and a third if necessary, but then start on a continuous rate infusion. While this category is uncommon, you will see it more commonly in patients with status epilepticus due to the internalization of the GABA receptors that benzodiazepines operate on (Charalambous et al. 2022).

INITIATING ANTIEPILEPTIC THERAPY

We'll talk more about managing the "chronic" seizure patient later and focus initially on the first-time cluster seizure/status epilepticus patient. The next step after terminating any ongoing seizure is to consider which anticonvulsant to start. Admittedly, this is a loaded question because there is substantial variation in recommendations depending on signalment, underlying etiology, and owner input. For simplicity, we'll divide our canine patient population into two categories: the younger patient who more likely has idiopathic epilepsy and the older patient who more likely has structural brain disease.

For the younger patient who likely has idiopathic epilepsy, the majority of clinicians will start phenobarbital, barring any obvious contraindication or owner concern about side effects (Meland et al. 2019, Griffin et al. 2022). Phenobarbital is a fairly effective drug that has the potential to reduce seizure frequency by about 85% in one study (Stabile et al. 2019). While levetiracetam is a reasonable choice for patients you suspect seizures due to a toxic etiology (Stabile and Di Risio 2021), evidence supporting its use as monotherapy in dogs with idiopathic epilepsy is lacking (Fredsø et al. 2016a). Finally, zonisamide monotherapy is also a reasonable option for idiopathic epileptics (Saito et al. 2024), though it has not been compared directly with phenobarbital. While phenobarbital does have a higher potential to cause some adverse events, it has not been linked to a decrease in quality of life as perceived by owners (Gristina et al. 2023) when compared to other medications. Important other considerations include owner's desire/ability to follow up for lab

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work (important for phenobarbital) and their ability to give medications every 8 hours (levetiracetam). Current recommendations for cats with idiopathic epilepsy are to use either phenobarbital or levetiracetam (Charalambous et al. 2018), though zonisamide may have some efficacy as well (Djani et al. 2024).

Patients who have experienced a seizure cluster (and especially status epilepticus) generally benefit from a loading dose of an anticonvulsant to rapidly achieve a therapeutic serum concentration. Interestingly, despite several different loading protocols (especially for phenobarbital), there is not a study that has directly compared their efficacy. My typical phenobarbital load is 12–16 mg/kg depending on age and 60 mg/kg for levetiracetam.

If we transition now to the older patient population who maybe at higher risk for structural epilepsy, the recommendations or decisions may shift. Choice of medication between phenobarbital and levetiracetam has not been shown to strongly influence seizure frequency and survival (Norona et al. 2020). Levetiracetam as monotherapy was effective in reducing seizures in approximately 50% of dogs with known structural epilepsy in one study (Kelly et al. 2017). My own practice is to discuss the pros and cons of each drug with a family and make a joint decision as to the best pathway forward, depending on the number of seizures and goals of care.

FALLBACK OPTIONS FOR THE REFRACTORY PATIENT

While infrequent, you may encounter a scenario where you've given a benzodiazepine AND a loading dose of an anticonvulsant, but your patient still continues to have seizure activity. Depending on the scenario, this is either recurrent or refractory cluster seizure/status epilepticus. The next option is a ketamine bolus +/− continuous rate infusion because NMDA receptors are upregulated with ongoing seizure activity, and ketamine has been successfully used in managing refractory cases (Roynard et al. 2021). I will typically reach for ketamine if a patient persistently has seizure activity despite three doses of a benzodiazepine and a loading antiepileptic dose. For patients who fail that treatment modality, one can consider propofol, alfaxalone, and inhalation anesthesia (Cagnotti et al. 2022, Al Kafaji et al. 2024, Sarpekidou et al. 2024). Thankfully, the frequency of these patients is rare.

Patients who have experienced a cluster seizure event or status epilepticus should be hospitalized for monitoring until they remain seizure- free for 24 hours as 50% of dogs will have additional seizure activity in the hospital (Kwiatkowska et al. 2018).

I've included a potentially helpful flowchart in Figure 16.1, though the order of ketamine versus benzodiazepine CRIs may be neurologist dependent. I think it provides a good overall summary, though, as you see, some of its recommendations differ from the text above.

WHEN IS AN MRI AND CSF SAMPLING HELPFUL?

At its core, the real question we are asking ourselves here is, could my patient have structural (i.e. not idiopathic epilepsy) intracranial disease such as meningoencephalitis of unknown etiology (MUE), neoplasia, or stroke whereby additional therapy beyond anticonvulsants might be helpful?

I think first it's important to understand what additional treatment options are even possible AND make sure an owner would consider them as viable approaches. Adjunctive therapy options for structural epilepsy might include corticosteroids (for autoimmune/inflammatory disease and certain neoplasia), radiation therapy, anticoagulation, and less commonly, surgery. For an older patient with a higher likelihood of neoplasia, if an owner would not pursue radiation therapy or surgery, the results of an MRI have no treatment implications. And while MRI +/− CSF sampling ARE the required tests to diagnose structural epilepsy, given the cost and logistics of advanced testing, we are often left treating empirically based on likelihoods and probability for certain diagnoses.

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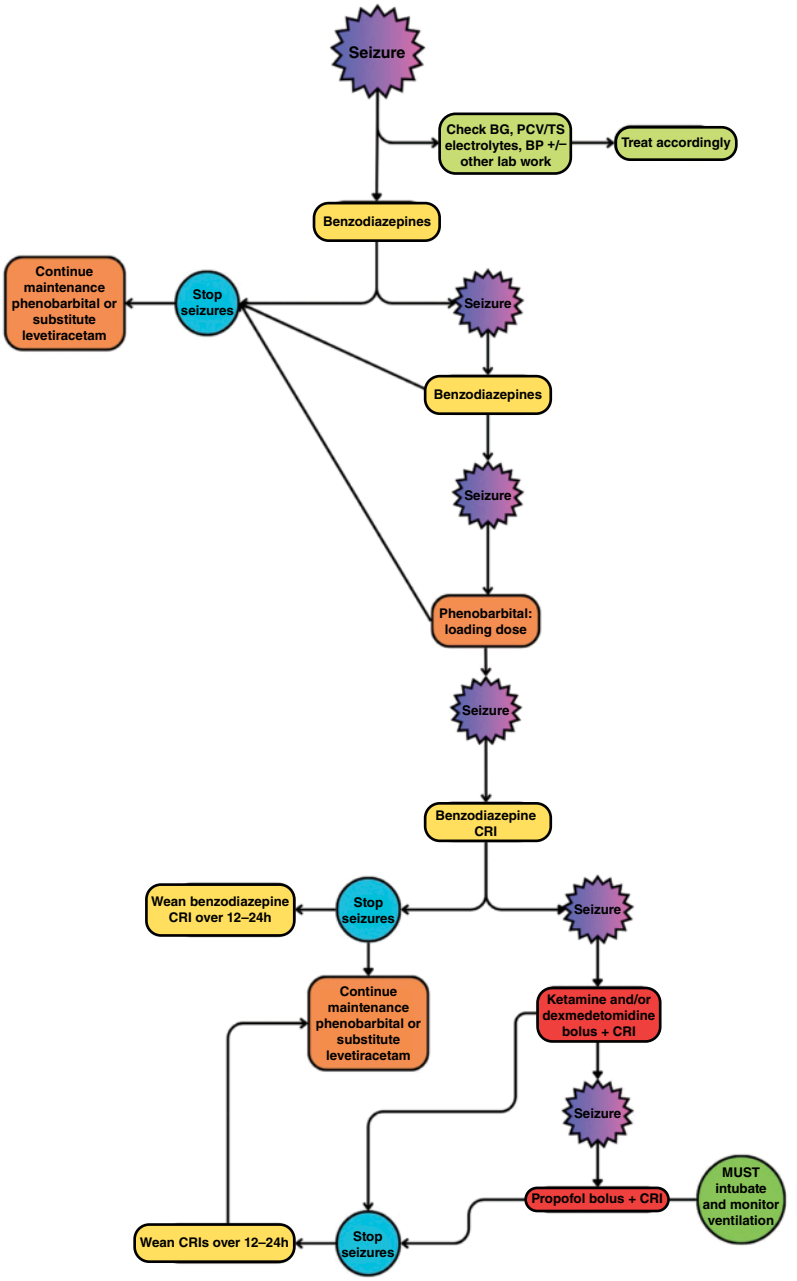


FIGURE 16.1 A reasonable flowchart for the initial management of cluster seizure/status epilepticus patients. *Source*: Munguia et al. (2024)/MDPI/CC BY 4.0.

To start, some broad differentials for cluster seizures are summarized in Table 16.1, and as you see, the first differentiation is an extracranial (reactive) versus an intracranial (structural or idiopathic epilepsies) etiology (Berendt et al. 2015).

Reactive seizures are less frequent than structural or idiopathic epilepsies in dogs and cats at 24 and 22%, respectively (Erlen et al. 2020, Schriefl et al. 2008). However, these are likely *overestimates*, especially in dogs, because many patients do not always receive complete workups.

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Table 16.1 Broad intracranial and extracranial differential diagnoses for seizures in the small animal patient.

Intracranial	Extracranial
Degenerative: • Thiamine deficiency (in cats) • Specific enzyme deficiencies Anomalous: • Hydrocephalus Metabolic: • Storage diseases Neoplastic: • Meningioma • Glioma • Lymphoma • Metastatic disease Inflammatory/Infectious: • Immune mediated (meningoencephalitis of unknown etiology) • Infectious (viral, protozoal, bacterial, fungal, rickettsial) Trauma/toxin: • Traumatic brain injury • Bromethalin Vascular: • Ischemia • Hemorrhage	Degenerative: • Severe hyperthermia Metabolic: • Hypoglycemia • Uremia • Hyperlipidemia • Hepatic encephalopathy • Electrolyte abnormalities (Na, Ca) • Hyperosmolar disorders (e.g. hyperosmolar hyperglycemic syndrome) • Urea cycle toxicity (rare) Toxicities: • Lead, organophosphates, chlorinated hydrocarbons, xylitol, caffeine, ethylene glycol Vascular: • Blood dyscrasias (anemia, severe thrombocytopenia) • Hypertension • Hyperviscosity (multiple myeloma, polycythemia, erythrocytosis)

Source: Munguia et al. (2024)/MDpI/CC BY 4.0.

Thankfully, the most common causes of reactive seizures in dogs such as drug ingestion and hypoglycemia are usually apparent due to either history or basic lab work (Brauer et al. 2011). Drug/toxin ingestion was also the most common etiology of reactive seizures in cats with less common causes of hepatic/renal disease and hypoglycemia that are also generally identifiable on lab work (Kwiatkowska et al. 2019). We won't discuss workup or treatment for these conditions further.

The other two sources of epileptic seizures are those with (structural) and without (idiopathic) evidence of macroscopic cerebral disease. In the absence of MRI and CSF findings, we have to try to evaluate risk factors when talking to owners about the likelihood that a patient has structural epilepsy. Dogs between 6 months and 6 years of age are most likely to have idiopathic epilepsy, and smaller breed dogs may be more likely to have inflammatory disease (Hall et al. 2020). Similarly, older dogs or those with cluster seizures and interictal neurologic deficit were at higher risk for having structural disease (Armaşu et al. 2014). Interestingly, a recent study used artificial intelligence to try and differentiate between idiopathic and structural epilepsy (Flegel et al. 2024). That study identified age, duration of seizure history, interictal frequency, and the presence of cluster seizures as important differentiating factors between idiopathic and structural epilepsy.

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Imaging and sampling maybe also be more fruitful in brachycephalic breeds as they likely have a higher incidence of structural disease when compared to other dog breeds (Prodger et al. 2025). In cats, increasing age over 6 years is also associated with an increased risk of structural epilepsy (Raimondi et al. 2017) with other risk factors including interictal neurologic deficits and patients with status epilepticus (Pákozdy et al. 2010).

I will give you my own opinion because the degree of overlap between dogs with structural and idiopathic epilepsy means there are no "hard and fast" rules you can apply. I will tend to push more for imaging in younger dogs that are high-risk breeds for meningoencephalitis of unknown etiology. Knowing a patient has MUE versus idiopathic epilepsy does have treatment as well as prognostic implications that are important for an owner to be aware of. For the older patient at higher risk for structural disease, I always discuss advanced imaging with the family in the context of their goals of care for the patient. The International Veterinary Epilepsy Task Force recommends imaging for patients who: are less then 6 months or greater then 6 years of age, have interictal neurologic deficits, present with status epilepticus or cluster seizures, or fail to respond to a single antiepileptic medication (De Risio et al. 2015).

CLIENT COMMUNICATION

Seizure emergencies are a common presentation and so having a prepared conversation can be helpful. I've included some bullet points and prognostic information below for nonreactive seizures.

1. Most seizures are either caused by idiopathic epilepsy (a wiring problem in the brain) or a structural issue such as a stroke, inflammation, or tumor.
2. Ultimately, an MRI of the brain and potentially CSF sampling is required to differentiate between these two categories.
3. Given the logistical and financial challenges of this testing, we will frequently initiate therapy empirically to treat seizures without knowing the underlying cause.
4. There are several medication options available to treat seizures, all with their pros and cons.
5. Phenobarbital is likely the most efficacious medication, and we have the ability to monitor drug levels to make sure it is in a therapeutic range. While it tends to cause more behavioral changes in the short term, those tend to resolve with time. It can also cause elevations in certain liver parameters, so it is important to monitor them over time (Charalambous et al. 2016). It is given every 12 hours.
6. Zonisamide is another reasonably effective medication, though likely not quite as good as phenobarbital. The incidence of side effects is reasonably low, and it can be given twice daily. We do not generally monitor drug levels for this medication.
7. Levetiracetam is the final option, and while it has the lowest side effect profile, it is also likely the least effective, especially in idiopathic epileptics. In addition, for many patients it has to be given three times daily and is much less effective if not given that way.
8. Our goal with any antiseizure medication is to reduce seizure frequency and reduce risk for cluster seizure or status events while maintaining a quality of life (Potschka et al. 2015).
9. Roughly 15% of patients with idiopathic epilepsy will be "seizure free" on medications. Positive prognostic factors for treatment success are older patients, lack of cluster seizures, female dogs, and neuter status (Packer et al. 2014).

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MANAGING THE CHRONIC SEIZURE PATIENT

Our final seizure emergency presentation is the patient with a history of seizures on medication who presents for ongoing seizure activity. The first crucial question is to make sure the patient is actually getting their medication and that there haven't been any missed doses. While the initial management for these patients is similar to above, also make sure to draw a blood tube to measure a phenobarbital drug level if they are on that medication. Finally, it is important to evaluate the drug doses that the pet is on to see whether or not any of them can be safely increased. If the patient has an appropriate serum drug level and is on the maximum dose of current antiepileptic medication, then it's time to consider adding another agent.

VESTIBULAR DISEASE IN THE EMERGENCY ROOM

Vestibular disease is also a common presentation through the emergency room because of its peracute onset and very visible clinical signs, including head tilt, ataxia, and nystagmus. The primary goal of this section is to differentiate patients who most likely have idiopathic old dog vestibular disease from those who do not. We'll also talk about the patient populations who might benefit from imaging to influence treatment beyond just symptomatic/supportive medications.

CAUSES OF VESTIBULAR DISEASE

As you already know, patients showing vestibular signs have a disease affecting either their peripheral or central vestibular systems with some differential diagnoses listed in Table 16.2.

Differentiating between central and peripheral disease on the basis of the neurologic exam can be challenging. While one study showed patients who are bright/alert and ambulatory with normal proprioception were more likely to have peripheral disease (Troxel et al. 2005), later studies show a lack of correlation between patients classified this way and MRI findings (Boudreau et al. 2018, Bongartz et al. 2020). The majority of the discrepancies were patients presumed to have peripheral lesions who actually had central disease. I've included certain risk factors on physical/neurologic exam (Table 16.3) for the most common diseases causing vestibular syndrome, understanding that exceptions inevitably occur and they should only be used to *rank* your differential list, not provide a definitive diagnosis.

Since patients with idiopathic/old dog vestibular signs have no relevant imaging abnormalities and no specific treatment, separating out this population is important to identifying patients who *would* benefit from MRI to guide more definitive treatment such as antibiotics, steroids, or antithrombotic agents. However, you can also use some of the risk factors in Table 16.3 for otitis interna/media and potentially MUE to start empiric therapy if imaging is not feasible.

Table 16.2 Common peripheral and central differential diagnoses for vestibular signs in the small animal patient. Courtesy of the author.

Peripheral vestibular disease	Central vestibular disease
Idiopathic/old dog vestibular disease	Neoplasia (primary or metastatic)
Otitis interna/media	Meningoencephalitis
Drug/toxin (i.e. metronidazole, aminoglycosides)	Vascular/stroke
Trauma	Metabolic (hypothyroidism)

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Table 16.3 Frequency and risk factors for the most common causes of vestibular signs in dogs.

Disease	Risk factors	Frequency (%)
Idiopathic/old dog vestibular	Older, larger breed, nystagmus, facial nerve paralysis	34
Otitis media/interna	Younger, male, hx of otitis externa, Horner's syndrome	24
Meningoencephalitis of unknown etiology	Younger, no hx of otitis externa, central vestibular signs	15
Vascular/stroke	Older, peracute onset, central vestibular signs	11
Neoplasia	Older, head tilt, concurrent cranial nerve deficits, central vestibular signs	11

Source: Adapted from Harrison et al. (2021).

DIAGNOSTIC OPTIONS FOR THE PATIENT WITH VESTIBULAR DISEASE

Traditionally, you may have learned about the utility of lab work, including a d-dimer level in raising/lowering your index of suspicion for stroke. Bloodwork and urinalysis are usually recommended to identify potential comorbidities that may have increased the risk of stroke, while an elevated d-dimer can be associated with clot formation/breakdown. While an initial study seemed to indicate a high prevalence (64%) of comorbidities such as chronic kidney disease (CKD), Cushing's disease and hypertension in stroke patients (Garosi et al. 2005), a later study showed a much lower prevalence (39%) with hypertension as the predominating comorbidity (Danciu et al. 2025). This is not to say that lab work is wrong in these patients but more to set expectations that you commonly won't find much directly related to the primary disease process. The utility of the d-dimer in making clinical decisions is unfortunately somewhat suspect. The largest study evaluating it in stroke patients showed significant overlap with a control group, poor sensitivity, and moderate to high specificity (DiPaola et al. 2024). While the authors of that study recommended using it as a screening test where a positive result should trigger additional testing, there are also many dogs with stroke with normal d-dimer levels (Koch et al. 2019). Therefore, a normal d-dimer does not rule out stroke, and an abnormal one may be suggestive, but certainly not definitive for one. In my opinion, with these data, a high d-dimer may support waiting to see if a patient improves (as would be expected with stroke) but is not sufficient evidence to start treatment by itself.

TREATMENT OPTIONS/PROGNOSIS

We'll focus next on the treatment options and prognosis for dogs with idiopathic vestibular disease as they are the most common presentations through the emergency service. A survey of neurologists indicated the most common treatments they employ are fluid therapy and maropitant as an antiemetic (Mertens et al. 2023), which is in line with my own practice and likely debunks the myth that medications prolong the course of the disease. What is fascinating is that there is no evidence supporting this medication (or meclizine) in vestibular dogs! The only studies to date have shown efficacy for ondansetron as an antinauseal (Foth et al. 2021, Henze et al. 2022), though it has not been directly compared with maropitant in a clinical trial. Roughly 50% of dogs will fully recover with the other half having persistent deficits (usually head tilt or facial paralysis) within roughly a week with a recurrence rate of approximately 22% (Orlandi et al. 2020, Radulescu et al. 2020).

FELINE VESTIBULAR DISEASE

Finally, while vestibular disease in cats is less common, there are some key species differences to be aware of in the diagnosis and treatment of this condition. Perhaps the biggest difference is the higher incidence of otitis media/interna in cats that can cause both peripheral and central signs

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if it extends into the brain (Negrin et al. 2010). This would appear to support the use of empiric antibiotics in vestibular cats if imaging is not pursued as the second most common cause in cats is idiopathic which would be neither harmed nor improved with antibiotic therapy. However, in older cats with more chronic signs and postural deficits were more likely to have neoplasia as their underlying cause for vestibular signs (Grapes et al. 2021).

ACUTE SPINAL CORD INJURY

In Chapter 10, we discussed traumatic vertebral fractures with secondary spinal cord injury. In this section, we'll address cervical and thoracolumbar myelopathies separately because their recommended management can be different. However, for each anatomic area, our critical questions will be the same:

1. How likely is this intervertebral disc disease?
2. Will medical management work?
3. What is the optimal medical management strategy?

Spinal pain or injury is always an important differential for your vaguely ill (lethargic, anorexic, not walking well) canine, especially in chondrodystrophic breeds (Parent 2010). Admittedly, the differential diagnosis for spinal pain can be broad as shown in the flowchart in Figure 16.2.

As we discussed in the seizure section, our goal as emergency clinicians is not necessarily to definitively diagnose the cause of the problem as that requires MRI, but to present a rational prioritized list of differential diagnoses to guide decision-making. Our discussion here is going to be focused on the *most likely* scenario, understanding that exceptions can and will occur.

APPROACHING CERVICAL PAIN

For patients with cervical pain and/or mild deficits (which I would argue anecdotally are the majority of the patients we see), the two most common differential diagnoses are intervertebral disc disease (IVDD) and steroid-responsive meningitis arteritis, or SRMA (De Strobel et al. 2019, Capasso et al. 2024). When broadly comparing these two patient populations, patients with SRMA tended to be younger, febrile, and have inflammatory changes on their complete blood count (CBC) (Grapes et al. 2020). While not directly evaluated in the previously cited study, certain breeds (especially larger breeds and beagles) are also likely at higher risk for SRMA (Wohlsein and Tipold 2023). The exception to the above maybe in juvenile (i.e. <18 months) dogs (especially

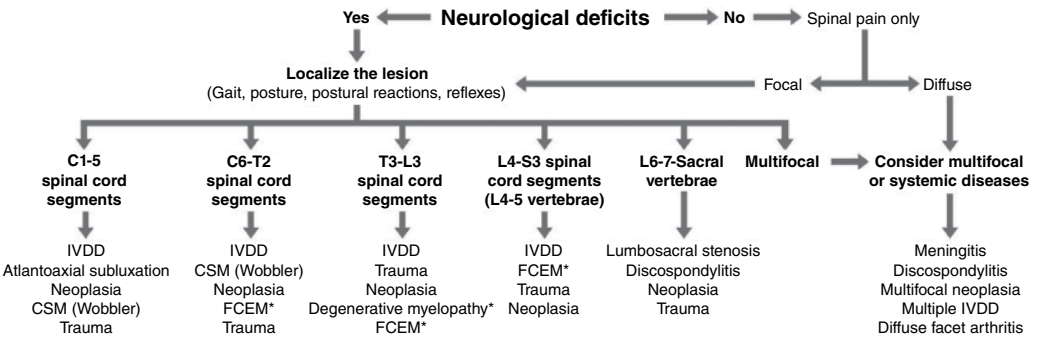


FIGURE 16.2 Broad differential diagnoses for myelopathies in small animal patients. *FCEM: Fibrocartilaginous embolic myelopathy*. Conditions that usually present without spinal pain (*). *Source*: Da Costa and Moore (2010) with permission of Elsevier.

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brachycephalics) where additional differentials should also include vertebral malformations and spinal diverticula (Pilkington et al. 2024).

Provided the patient would be considered "low risk" from a SRMA perspective, the presumptive diagnosis is usually going to be IVDD in the majority of patients with acute cervical pain. Therefore, the next question is, how likely is it that medical treatment will be successful? For patients with pain and/or mild neurologic deficits, the likelihood of successful medical management is roughly 50–75% (Levine et al. 2007a, Olby et al. 2020). The second question is, which medication is associated with the best outcome? Only one study indirectly evaluated this question and seemed to indicate that NSAIDs (though unclear if one NSAID is superior to another) led to better outcomes compared with corticosteroids (Levine et al. 2007a).

THORACOLUMBAR MYELOPATHY

Transitioning to the thoracolumbar spine, we'll attempt to answer the same questions as above to educate owners on the likelihood of IVDD and the pros and cons of advanced imaging/surgery versus medical management.

For dogs whose neurolocalization is the thoracolumbar spine, there are several signalment, history, and physical exam clues we can use to initially prioritize our differential diagnoses as shown in Figure 16.3.

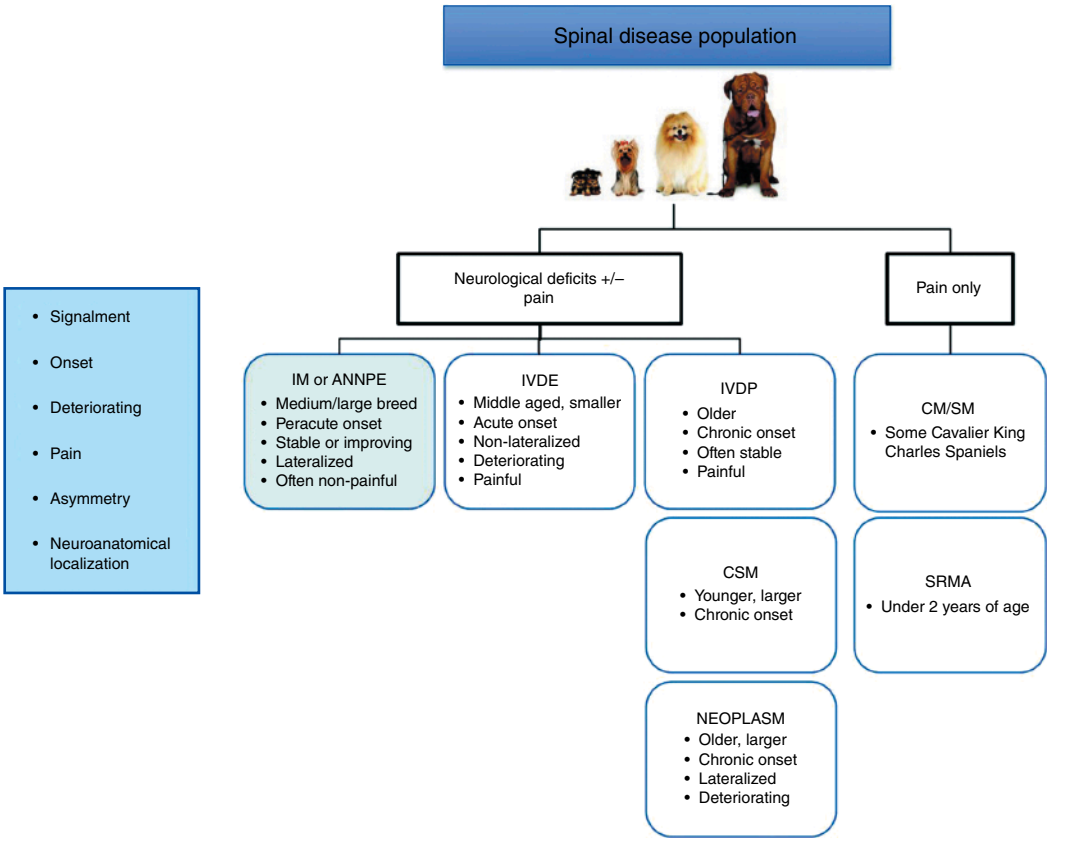


FIGURE 16.3 Differential diagnoses based on history, signalment, and physical exam findings. *ANNPE, acute non-compressive nucleus pulposus extrusion; CM/SM, Chiari-like malformation/syringomyelia; CSM, cervical spondylomyelopathy; IM, ischemic myelopathy; IVDE, intervertebral disc extrusion; IVDP, intervertebral disc protrusion; SRMA, steroid-responsive meningitis and arteritis. Source*: *Cardy et al. (2015) with permission of John Wiley & Sons.*

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Table 16.4 Success rate of medical versus surgical management for thoracolumbar IVDD of varying severities.

Injury severity	Number of cases reported	Medical management (%)	Surgical decompression (%)
Paraplegia with absent deep pain sensation	513	10	61
Paraplegia with intact deep pain sensation	603	62	93
Non-ambulatory paraparesis	354	79	93
Ambulatory paraparesis	152	48–84	95
Paraspinal hyperesthesia only	143	60–100	97

Source: Moore et al. (2020)/Frontiers/CC BY 4.0.

Even without imaging, a combination of those findings along with pain and lateralization of signs was shown to reasonably differentiate between IVDD and the next most common etiology, fibrocartilaginous embolism/acute non-compressive nucleus pulposus extrusion (Scalia and Freeman 2024). One interesting point of view was provided by Khan and Freeman who attempted to use Bayesian reasoning (see Chapters 2 and 3) in evaluating back pain patients without the use of advanced imaging (Khan and Freeman 2024), though their discussion was mostly theoretical, not a clinical trial.

MEDICAL VERSUS SURGICAL MANAGEMENT

As we did for cervical myelopathies, we'll again focus on IVDD as that is the most common disease process seen in this area (Cardy et al. 2015). The first question we'll consider is, when do MRI and surgery lead to a better outcome than medical management? In a survey of neurologists and surgeons, virtually all indicated that their "cutoff" for recommending surgery in a first-time patient was if their neurologic status was worse than or equal to nonambulatory paraparesis, though many (58%) also recommended surgery for ambulatory paraparetic/ataxic patients (Moore et al. 2016). This would be roughly in line with previous studies that have shown comparable outcomes between medical and surgical management until a patient loses motor function, as summarized in Table 16.4, and is in rough agreement with current practice guidelines (Olby et al. 2022). However, one recent study did indicate a higher success rate (48%) for medical management of small non-brachycephalic dogs lacking deep pain sensation than previously reported (Khan et al. 2024), consider this for owners without the financial ability to pursue surgery. It's important to understand risk factors for why medical management may *fail* so that owners can be aware of the potential for disease progression in the future. Unfortunately, no study has satisfactorily evaluated breed, neurologic status, history, and other parameters as risk factors for failure of medical management. However, two studies have shown a small signal that NSAIDs maybe more effective in preventing short-term progression (Levine et al. 2007b) and long-term recurrence (Mann et al. 2007) when compared to corticosteroids. However, current consensus recommendations vacillate between clinical equipoise (Moore et al. 2020) and recommending against corticosteroids (Olby et al. 2022).

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Immune-mediated Hemolytic Anemia and Thrombocytopenia

CHAPTER 17

CRITICAL QUESTIONS

1. Is it *actually* IMHA?
2. What diagnostics are helpful in IMHA and ITP?
3. Do they need a transfusion?
4. What initial immunosuppressant works best for IMHA and ITP?
5. How should I anticoagulate?
6. Do I have to give vincristine to the ITP patient?
7. How do I manage hemorrhage in the ITP?

INTRODUCTION

Both anemia and thrombocytopenia are common referrals from primary care veterinarians that you will encounter working in the emergency room. Having a workup and treatment protocol in mind along with an understanding of prognosis and monitoring is important for efficiently managing these patients. We'll focus almost exclusively on the initial diagnostic testing and medications for these diseases as the more chronic management decisions are outside the scope of this book.

IS IT ACTUALLY IMHA?

The first thing to consider when confronted with an anemic patient is to make sure they don't have a source of hemorrhage (abdominal, thoracic, or gastrointestinal). The second is to evaluate their CBC (if performed) to make sure the platelet and white blood cell counts aren't low, which would indicate dysfunction at the level of the bone marrow. You should also check the reticulocyte count to see whether the anemia is regenerative or nonregenerative as that can also help narrow down your differential as well. If the above process hasn't yielded a presumptive diagnosis, then it's time to consider the possibility of a hemolytic type anemia.

There are fairly strict criteria for the diagnosis of immune-mediated hemolytic anemia or IMHA, as shown in Figure 17.1. Unfortunately, many anemic patients with IMHA do not meet all of these criteria and so you should also consider other differential diagnoses such as precursor-directed IMHA (PIMHA), nonregenerative IMHA, a different hemolytic disease (i.e. microangiopathic hemolysis, hypophosphatemia, and heavy metal toxicosis), or hemophagocytic histiocytic sarcoma among others. Dogs with either PIMHA or nonregenerative IMHA can have some of the criteria seen in Figure 17.1 (Assenmacher et al. 2019, Woolhead et al. 2021), though thankfully, initial workup and treatment is similar, so we won't delve into them further.

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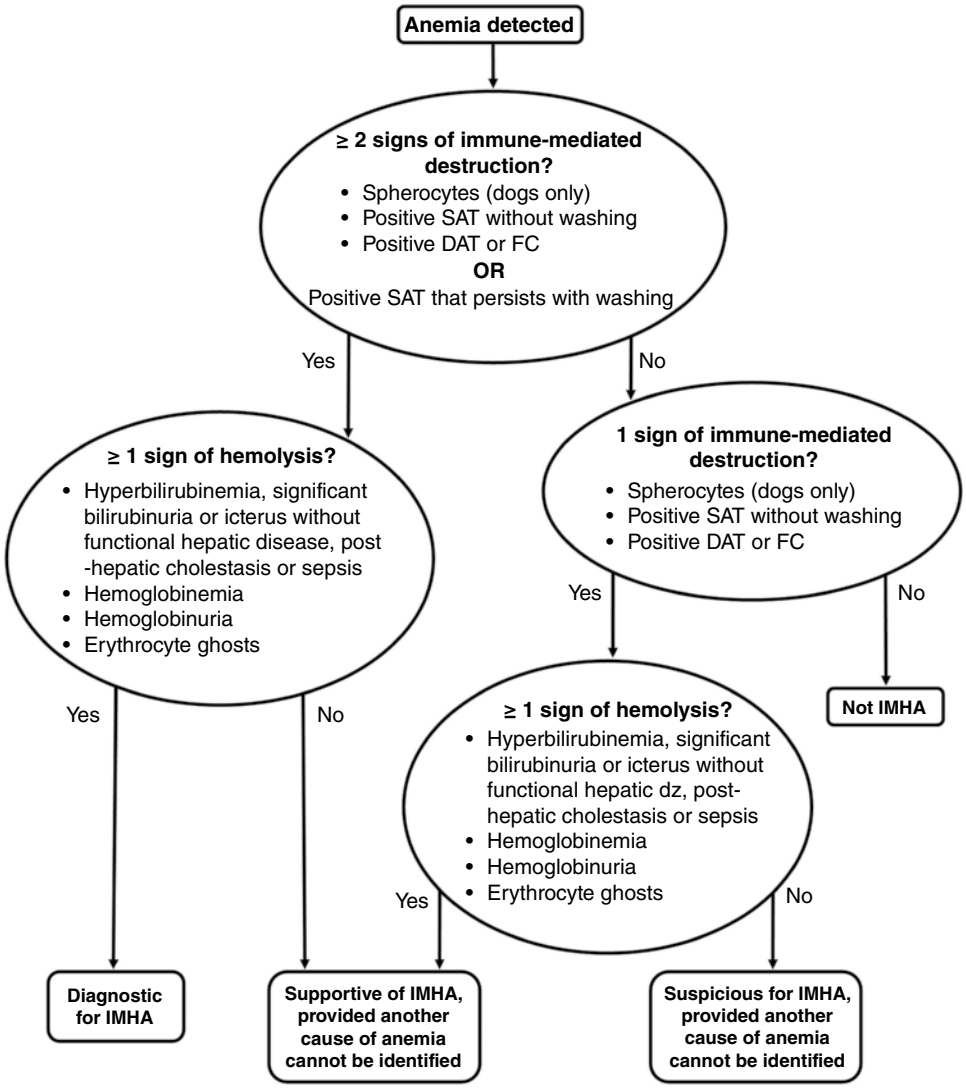


FIGURE 17.1 Diagnostic guidelines for canine IMHA. *Source*: Garden et al. (2019) with permission of John Wiley & Sons/CC BY 4.0.

WHAT DIAGNOSTICS ARE HELPFUL?

For a patient with peripheral IMHA, the next differentiation to make is whether or not this is associative (i.e. has an identifiable trigger factor) or nonassociative (i.e. idiopathic). Traditionally, this has involved whole body imaging and infectious disease testing with the concept being that if you can identify an underlying disease process, you may have additional treatment options beyond immunosuppression. Unfortunately, this testing does add significant expense to what is already an expensive disease and so I think it's important to consider the likelihood that you will obtain useful results with this approach. Thoracic radiographs will rarely show relevant abnormalities with 68–100% being normal, and while abdominal ultrasound will frequently show abnormalities, they infrequently change treatment (Andres et al. 2019, Woodward et al. 2020, De Laet et al. 2022). While certain metallic foreign bodies can cause a hemolytic anemia, it is exceedingly uncommon

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Table 17.1 Diagnostic workup options and hierarchy of importance for IMHA.

Required tests	Comments
PCV/TS	Required to verify anemia, icteric or hemolyzed serum also supports hemolytic process
Complete blood count	Required to evaluate platelet and WBC levels that might indicate bone marrow dysfunction
Reticulocyte count	Required to evaluate regenerative capacity
POCUS	Required to briefly evaluate for cavitary effusion/hemorrhage
Rectal exam	Evaluate for melena
Likely helpful	
Chemistry (total bilirubin)	Total bilirubin has prognostic significance and is evidence of hemolysis
Not mission critical	
Infectious disease testing	Infectious disease is an uncommon cause for IMHA
Whole body imaging	Has not been shown to influence treatment in vast majority of cases

PCV/TS, packed cell volume/total solids; POCUS, point of care ultrasound.

Table 17.2 Anemic Dog Clinical Assessment Score (ADCAS). The higher the total score, the more likely the patient is to benefit from a transfusion.

	Normal (0)	Mild (1)	Moderate (2)	Severe (3)
Mucosa color	Salmon pink	Slightly pale	Moderately	Severely pale
Pulse quality	Normal	Bounding	Weak	Weak
Heart rate	65–109 bpm	110–140 bpm	>140 bpm	>140 bpm
Respiratory rate	15–24 rpm	25–40 rpm	>40 rpm	>40 rpm
Mentation/exercise tolerance	BAR, walking	Quiet, able to walk	Lethargic, able to stand	Lethargic, unable to stand

Source: Kisielewicz et al. (2014) with permission of John Wiley & Sons.

(I have seen one case). Interestingly, board-certified specialists were more likely to run tests than primary care clinicians (Swann et al. 2021) for the diagnosis of IMHA! There is more limited data surrounding the utility of infectious disease testing in these patients and current recommendations are to test for the relevant vector-borne disease in your area (Garden et al. 2019, Macneil et al. 2019). I've summarized my opinion on required diagnostics in Table 17.1.

TRANSFUSION DECISION-MAKING

Essentially, all dogs presenting with IMHA are anemic to some degree. The hard part is both in predicting which ones will require a transfusion on presentation AND what their ongoing transfusion requirement will be. While one study evaluated the use of clinical parameters including mucous membrane color, heart rate, pulse quality, respiratory rate, and mentation to identify patients who would benefit from transfusion (Table 17.2), only about half of those patients had IMHA and so its generalizability is unclear.

Current expert opinion on transfusion therapy is mixed with current guidelines recommending transfusions for patients with evidence of decreased oxygen delivery and increased lactate with a minority suggesting a PCV cutoff of 12% (Swann et al. 2019a). Ideally, the "youngest" blood unit is used as it likely reduces the risk of a transfusion reaction (Maglaras et al. 2017). I've included my own list of anecdotal transfusion risk factors in Table 17.3, where the more factors present, the more likely

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Table 17.3 The author's transfusion risk factors in canine IMHA.

Transfusion risk factors
Tachycardia
PCV < 20%
Hyperlactatemia
Tachypnea

a transfusion is indicated. However, even more important than these initial factors is making sure that you monitor them through the course of hospitalization to ensure that they are not deteriorating.

While there is a large volume of literature surrounding prognostic factors for IMHA, which we'll discuss later, there haven't been any studies directly evaluating risk factors for transfusion dependency. The struggle is that the timeline for each patient's response to immunosuppression (thus liberating them from transfusion dependency) is variable, making prognostication difficult.

STARTING IMMUNOSUPPRESSION AND A SECOND AGENT

The overwhelming recommendation for first-line immunosuppression in IMHA is a corticosteroid with no known therapeutic superiority of prednisone, prednisolone, or dexamethasone. One study did show that twice-daily dosing of corticosteroids reduced bilirubin faster than once-daily dosing, though it did not appear to have an impact on any clinically significant patient outcome (Swann et al. 2019b).

While current expert opinion only suggests using a second agent in patients with severe/life-threatening disease, inability to maintain a stable PCV, or negative steroid side effects (Swann et al. 2019a), many clinicians will start a second immunosuppressive agent sooner as it may allow faster liberation from high dose steroid therapy. Current recommended second-line agents include azathioprine, cyclosporine, mycophenolate, and leflunomide (Goggs 2020). Studies comparing steroids versus multi-agent protocols showed no difference in initial outcome, though the use of a second agent was shown to reduce relapse rate in one of two studies (Weng et al. 2023, Agnoli et al. 2024). As an emergency clinician, frequently starting and selecting a second immunosuppressive agent is a decision for the inpatient doctor, and so we won't discuss them further here.

THROMBOEMBOLISM/THROMBOPROPHYLAXIS

There are multiple mechanisms by which dogs with IMHA are hypercoagulable (Kidd and Mackman 2013), and thromboembolism was responsible for 60% of fatalities in one study (Duclos et al. 2024). Patients with thrombocytopenia, severe hyperbilirubinemia, and hypoalbuminemia are at the highest risk for thromboembolic events (Carr et al. 2002). Common clinical signs of thromboembolism in these patients include new onset dyspnea (indicative of pulmonary thromboembolism), neurologic signs (head tilt, seizures, nystagmus, ataxia), or new onset of severe gastrointestinal signs/ascites that may indicate a portal vein thrombus.

Given the high rate of thromboembolism in IMHA, all patients with this disease should be started on some form of antithrombotic (deLaforcade et al. 2019). Pharmaceutical options for thromboprophylaxis include aspirin (Weinkle et al. 2005), clopidogrel (Mellett et al. 2010), fractionated/unfractionated heparin (Breuhl et al. 2009, Panek et al. 2015), and rivaroxaban (Morassi et al. 2016). There are clear cost (rivaroxaban) and logistical (need for injections with heparin) disadvantages to anticoagulants compared with antiplatelet agents, though current (expert opinion) recommendations do prioritize their use due to a higher prevalence of venous thrombi in IMHA (Goggs et al. 2018, Swann et al. 2019a). That being said, the one retrospective study comparing clopidogrel to an anticoagulant protocol showed no difference in the rate of thrombosis, so it is reasonable to start clopidogrel only in most "normal risk" patients. My own current practice is to start clopidogrel in these patients.

For patients with recalcitrant IMHA who are not responding to initial therapy, other options, including human intravenous immunoglobulin infusions, or hIVIG (Whelan et al. 2009), plasmapheresis (Francey et al. 2021), and splenectomy (Bestwick et al. 2022) can be considered. None of these are recommended as first-line therapy and most studies have not indicated a survival benefit

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for IVIG (Kane and Greer 2023). The timing of these treatments is also debated as is the definition of treatment-resistant or recalcitrant IMHA (Swann et al. 2019a). The more practical debate is that for patients who are requiring multiple transfusions, the financial cost rapidly adds up, as does the risk of transfusion complications that may favor earlier second-line intervention.

PROGNOSTIC FACTORS WITH IMHA

As we mentioned above, IMHA is an expensive disease to both diagnose and treat. Therefore, setting appropriate client expectations surrounding prognosis is imperative. Table 17.4 summarizes survival rate and prognostic factors from multiple IMHA studies.

While there have been two IMHA severity scores developed (Table 17.5), their discriminatory ability for mortality is only moderate at best with the CHAOS scores (Whelan et al. 2006)

Table 17.4 Studies evaluating prognostic factors in canine IMHA.

Study	Number of patients	Short-term survival (%)	Negative prognostic indicators	Relapse rate
Carr et al. (2002)	72	42	Thrombocytopenia, increased bilirubin	n/r
Weinkle et al. (2005)	151	75	Leukocytosis, thrombocytopenia, coagulopathy, hypoproteinemia, hyperbilirubinemia	15%
Goggs et al. (2015)	276	74	Increased CHAOS score, increased ASA status, AKI, increased bilirubin	n/r
Weng et al. (2023)	242	77	Not evaluated	7%
Agnoli et al. (2024)	43	93	Not evaluated	7%
Sparrow et al. 2024)	73	n/r	Not evaluated	11% at 1 year, 18% at 2 years
Duclos et al. (2025)	104	87	Increased CHAOS score, higher bilirubin, thrombocytopenia	7%

AKI, acute kidney injury; n/r, not reported; CHAOS, canine hemolytic anemia objective score.

Table 17.5 Two IMHA clinical severity scores.

Canine hemolytic anemic objective score (CHAOS)	
Age (year)	If ≥7 score 2, otherwise score 0
Temperature (°F)	If ≥102.0 score 1, otherwise score 0
Agglutination	If present score 1, otherwise score 0
Albumin (g/dl)	If <3.0 score 1, otherwise score 0
Bilirubin (mg/dl)	If ≥5.0 score 2, otherwise score 0
Total	Maximum score 7
Tokyo score	
Sex	Male score 1, Female score 0
Season	April–September score 1, October–March score 0
Packed cell volume (%)	If <20 score 1, otherwise score 0
Platelet count ($\times 10^3$/μl)	If <200 score 1, otherwise score 0
Total protein (g/dl)	If <6.0 score 1, otherwise score 0
Total	Maximum score 5

Source: Goggs et al. (2015)/John Wiley & Sons/CC BY 4.0.

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performing better than the Tokyo scoring system (Ishihara et al. 2010, Ke et al. 2015, Goggs et al. 2015, Hall et al. 2022, Duclos et al. 2024).

A third proposed scoring system based on Systemic Inflammatory Response Syndrome (SIRS) criteria, and ASA status along with certain biochemical parameters has not been prospectively evaluated (Goggs et al. 2015). For future relapse, only a higher total bilirubin was found to be a predictive factor (Bannister et al. 2024).

DIAGNOSIS OF ITP

We'll approach ITP in a similar manner to IMHA in terms of diagnosis, diagnostic testing, and treatment, acknowledging that there is more limited data on that disease.

While there is an exhaustive flowchart (Figure 17.2) for the diagnosis of ITP, my own personal experience is that the vast majority of patients will present with petechiae and/or ecchymoses with a platelet count of less than 10 000 platelets/mcl.

Interestingly, there is more data to support the use of empiric imaging (especially abdominal ultrasound) in ITP patients compared to those with IMHA (Scuderi et al. 2016, Spear et al. 2024, López-Bailén et al. 2025). Furthermore, given the higher association of certain vector-borne diseases with ITP, PCR/serology testing for *Ehrlichia, Babesia, Anaplasma, Leishmania*, and *Angiostrongylus* maybe indicated for these patients depending on your local prevalence patterns (LeVine et al. 2024a).

IMMUNOSUPPRESSION AND ADJUNCTIVE THERAPIES IN ITP

As with IMHA, corticosteroids are the first-line immunosuppressants (LeVine et al. 2024b), though there is one case series of patients managed on mycophenolate alone (Yau and Bianco 2014). Based on the current ACVIM consensus guidelines, there is no current evidence that one

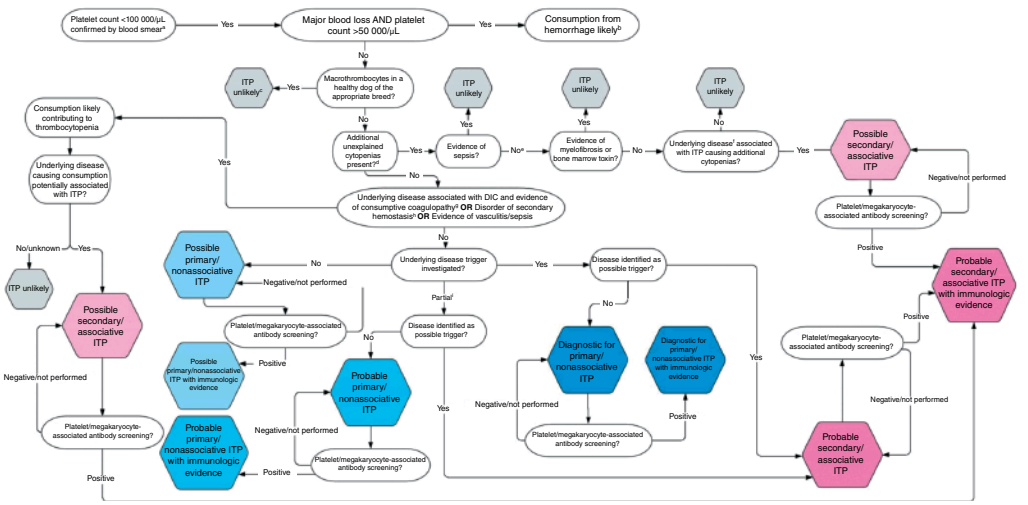


FIGURE 17.2 Diagnostic algorithm/flowchart for the diagnosis of canine ITP. Confirmed via blood smear evaluation (a), unless concurrent disseminated intravascular coagulation, Vasculitis or splenic sequestration present (b), requires confirmatory genetic testing (c), ensure no other plausible explanation (i.e. hemorrhage induced anemia) for thrombocytopenia (d), consider bone marrow sampling (e), ruleout concurrent disease such as lymphoid neoplasia/ehrlichiosis (f), evaluate for prolonged coagulation time (PT, aPTT), elevated D-dimer level consistent with consumptive coagulopathy (g), as evidenced by prolonged PT/aPTT (h).
Source: LeVine et al. (2024a) with permission of John Wiley & Sons.

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steroid (i.e. dexamethasone, prednisone, or prednisolone) is superior to another. They recommend the immediate initiation of a second immunosuppressive agent (identical options as with IMHA) in patients with active hemorrhage or when a comorbidity would benefit from a more rapid steroid taper. No difference in outcome has been demonstrated for patients treated with multiple agents compared with steroid monotherapy (Scuderi et al. 2016). And while a literature review did not identify one secondary agent as superior to another, the authors cautioned that many of the studies had limitations and methodologic flaws (Kristiansen and Nielsen 2021). Interestingly, despite its expense, hIVIG has been shown to hasten platelet count improvement in the majority (but not all) of trials it has been tested in (Bianco et al. 2009, Balog et al. 2013, Zoia et al. 2024). For patients who either test positive for a vector-borne disease or have a high clinical suspicion for one, doxycycline would be indicated pending test results.

Vincristine has been shown to hasten platelet count recovery and decrease hospitalization time (Rozanski et al. 2002), though **it should not be coadministered with cyclosporine** due to a risk of neutropenia (LaQuaglia et al. 2021).

For patients with more refractory ITP, treatment with plasmapheresis can be considered, though the data are mixed. A preliminary case series had 3/4 dogs with refractory ITP respond to this treatment (Kopecny et al. 2020), while a subsequent study did not show any difference compared to conventional controls (Francey et al. 2021). However, I think it is important to recognize that most dogs treated with plasmapheresis in both studies had already failed conventional therapy, resulting in some degree of selection bias. Finally, while expensive, the use of a thrombopoietin receptor agonist romiplostim has been described for primary ITP (Ryu et al. 2024) and may play a larger role in the future as many dogs with ITP have low thrombopoietin (Brooks et al. 2024b).

MANAGING HEMORRHAGE IN THE ITP PATIENT

If the largest driver of mortality in IMHA was thromboembolism, the most common cause for mortality in ITP is likely hemorrhage in the lungs, central nervous system, or gastrointestinal tract (O'Marra et al. 2011). A clinical bleeding score for canine patients with ITP was developed (Table 17.6), though it did not correlate with survival or transfusion requirement in a validation cohort (Brooks et al. 2024a). Therefore, while it is a helpful monitoring tool, it should be taken in the context of the patient's cage side exam.

There are several tools available to try and both prevent and treat hemorrhage. Ideally, you can prevent a bleed from happening in the first place. While the use of antacids (such as pantoprazole/omeprazole) theoretically makes sense, no trial has actually documented their efficacy. Current recommendations are split with one citing insufficient evidence to make a recommendation (Marks et al. 2018) and the other making a weak recommendation for use in the presence of melena (LeVine et al. 2024b). Patients with evidence of ongoing systemic hemorrhage should receive both vincristine and hIVIG in an attempt to raise their platelet count faster (LeVine et al. 2024b). While the use of platelet transfusions again may make sense conceptually and has been described in ITP patients (Saint-Pierre et al. 2023), that study did not document efficacy outcome and so the role of platelet transfusions is unclear. If you are going to give platelets, no difference has been shown between fresh platelet concentrate and lyophilized platelets (Davidow et al. 2012), though there may be a small clinical difference between cryopreserved and lyophilized platelets (Goggs et al. 2020). Platelet transfusions (or fresh whole blood) should not be first-line therapy but could be considered for patients with life-threatening hemorrhage or who need an invasive procedure. While the use of aminocaproic acid increased clot strength, it did not improve outcome or transfusion requirement (Wolf et al. 2024). A small study showed similar results for tranexamic acid as well (Olivares et al. 2023).

A summary of medications to consider is summarized in Table 17.7.

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Table 17.6 "DOGIBAT" score using to quantify bleeding in ITP.

	Bleeding grade		
Site	**0**	**1**	**2**
Skin	No	Petechiae/ecchymoses single site	Petechiae/ecchymoses >1 anatomic site
Catheter/venipuncture/ other cutaneous bleed	No	Self-limiting and <5 minutes	>5 minutes and/or intervention to control
Oral mucosa	No	Petechiae	Frank hemorrhage
Intraocular	No	Funduscopic	Hyphema
Epistaxis	No	Unilateral and <5 minutes	Bilateral and >5 minutes
Gastrointestinal	Occult blood (−); (Hema-chek™, Siemens Healthcare Diagnostics Inc., Tarrytown, New York)	Occult blood (+); (Hema-chek, Siemens Healthcare Diagnostics Inc.)	Hematemesis, hematochezia, melena
Urinary	No	Microscopic (dipstick)	Macroscopic
Pulmonary hemorrhage (suspected/observed)	No	N/A	Yes
Intracranial hemorrhage (suspected/observed)	No	N/A	Yes

Source: Makielski et al. (2018) with permission of John Wiley & Sons.

Table 17.7 The author's initial treatment recommendations for canine ITP.

Always start	**Rationale/comments**
Corticosteroids	First-line therapy
Antacids	May help reduce risk of GI ulceration
Vincristine	Will hasten platelet recovery, do not give if starting cyclosporine
Consider starting	
Doxycycline	Usually start pending infectious disease testing
hIVIG	Will hasten platelet recovery, always administer for patients with active bleeding
Second immunosuppressive agent	Can hold for receiving clinician decision, but likely indicated in hemorrhaging patients
Rarely given	
Platelet transfusion	Consider planning invasive procedure

TIME TO RECOVERY AND PROGNOSIS

The expected time for platelet recovery (i.e. above clinical bleeding threshold) is roughly 4–5 days with patients treated with vincristine generally toward the lower end and the use of hVIG lowering it further to less than 3 days (LeVine et al. 2024b).

Some survival statistics, negative prognostic indicators, and relapse rates are summarized in Table 17.8. The only risk factors for transfusion identified were a low hematocrit on presentation and longer duration of hospitalization (Brooks et al. 2024a).

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Table 17.8 Mortality, prognostic indicators, and relapse data from selected studies on canine ITP.

Study	Number of patients	Short-term survival (%)	Negative prognostic indicators	Relapse rate
O'Marra et al. (2011)	73	84	Melena, increased BUN	9%
Simpson et al. (2018)	45	90	Not evaluated	31%
Brooks et al. (2024)	58	85	Anemia, increased BUN	10%
Sparrow et al. (2024)	55	n/r	Not evaluate	11% at 1 year, 23% at 2 years
López-Bailén et al. (2025)	49	67	Anemia	n/r

n/r, not reported.

CLIENT COMMUNICATION PEARLS

There are a couple of important points to communicate with clients when you diagnose (or suspect) either IMHA or ITP. The first is making sure that the owner has a desire to treat their patient. These patients require lengthy medication courses and follow-up that owners may or may not be logistically/financially able to undertake. Beyond that, some client communication points to make are listed below:

- Diagnostic imaging is likely not helpful in IMHA but may be more helpful in ITP. If the results of imaging would change an owner's course of treatment, then they should be considered.
- Initial therapy involves immunosuppression with steroids, though some patients go on additional immunosuppressive medications.
- For patients who fail initial therapy, adjunctive treatments such as hIVIG and TPE are options, though due to expense, they are not considered first line.
- Most patients with IMHA require at least one blood transfusion with several requiring more than one.
- Patients with IMHA are prone to developing blood clots and require antiplatelet therapy. For patients who do develop clots, they can be fatal.
- While many ITP patients don't need transfusions, they do have the potential to bleed into critical areas such as the lungs and central nervous system, causing dramatic and potentially life-threatening clinical signs.
- The mortality rate for IMHA is roughly 30–40% with a slightly lower rate around 20% in ITP. Negative prognostic factors for mortality both diseases are in Tables 17.4 and 17.8.

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Approach to the Azotemic Pet

CHAPTER 18

CRITICAL QUESTIONS

1. Is this pre-renal, renal, or post-renal azotemia?
2. Is this acute, acute on chronic, or chronic renal failure?
3. Should I measure urine output?
4. How do I medically manage oligo/anuria?
5. When is hemodialysis indicated?

INTRODUCTION

Similar to immune-mediated hemolytic anemia (IMHA) and thrombocytopenia (ITP), azotemia is common referral with an estimated prevalence of roughly 12% in dogs presenting to one academic referral hospital (Babyak et al. 2017). In my eyes, the challenge in managing these pets is the broad differential for azotemia combined with a paradoxically diverse but limited array of treatment options. In this chapter, we'll first address the varying causes for azotemia, followed by a discussion of the management of acute kidney injury (AKI) and acute on chronic kidney disease (AoCKD), acknowledging the species variations between dogs and cats. As this chapter is focused on the emergent diagnosis and management of these patients, we won't focus on managing the chronic kidney disease (CKD) patient.

THE PRE-RENAL AZOTEMIA

Pre-renal azotemia is the elevation of BUN and creatinine either due to dehydration or decreased renal perfusion from hypotension/low cardiac output (Juncos and Juncos 2013). It is typically associated with an azotemia associated with oliguria, high urine specific gravity, history of fluid loss, and rapid correction of the azotemia after fluid therapy (Polzin and Osbourn 2012). There are limited data on the prevalence and outcome of dogs and cats with pre-renal azotemia likely because the majority of them get better with fluid therapy. By correcting the source of the hypovolemia or hypotension either through fluids and/or vasopressors, you will likely correct or improve the azotemia.

RENAL OR INTRINSIC AZOTEMIA

Renal azotemia is the elevation of BUN and creatinine due to intrinsic renal damage and/or insufficiency leading to an inadequate number of functional nephrons. Beyond the urine specific gravity, pre-renal and renal azotemia can be differentiated using urine electrolytes, though it is infrequently employed for this purpose (Troìa et al. 2018a). However, understanding the underlying etiology from your history (i.e. ingested ibuprofen 3 days prior) is also frequently helpful in making this determination.

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Table 18.1 Some differentiating characteristics of acute and chronic kidney disease, acknowledging that in patients with acute on chronic disease, there may be some overlap.

	Acute kidney disease	Chronic kidney disease
Historical factors	No known renal disease	History of CKD, PLN, renal dysplasia
Physical exam	Good body/muscle condition, enlarged, and painful kidneys	Thin, muscle-wasted, small/misshapen kidneys
Laboratory findings	Generally normal RBC	Nonregenerative anemia
Renal ultrasound	Can be normal or enlarged, can have retroperitoneal effusion	Typically small and irregular, can be asymmetric, and occasionally have renal cysts

PLN, protein losing nephropathy.
Source: Myott and Langston (2011).

POST-RENAL AZOTEMIA

Post-renal azotemia is the inability of urine to be excreted from the body either due to a blockage in the urethra or both ureters from stones, mucus plug, stricture, clot, or neoplasia. I would also include uroabdomen in this section as well, though we previously discussed that disease process in the chapter on trauma. Since urethral obstruction was covered in Chapter 13, the main focus later on in this chapter will be on ureteral obstructions.

DIFFERENTIATING AKD, AOCKD, AND CKD

Once you've established this is a renal azotemia, the next step is to ideally distinguish whether or not this is an AKI, acute on chronic renal failure, or progression (end stage) of a patient's renal disease. This is important not just from a workup and treatment perspective but also prognostically since patients with acute on chronic or progressive chronic disease may not regain as much (if any) renal function.

Unfortunately, there have not been many studies differentiating these patients on presentation, and the vast majority of symptoms are identical between both groups. Some general guidelines to help differentiate these patients are shown in Table 18.1.

And while understanding the underlying etiology for renal injury is always helpful, it is frequently hard to determine in patients with an acute on chronic injury (Dunaevich et al. 2020, Chen et al. 2020). Certain biomarkers, such as neutrophil gelatinase-associated lipocalin (NGAL), have been shown to differentiate AKI and CKD patients (Steinbach et al. 2014) whose value is not available on most chemistry panels. Unfortunately, the more ubiquitous biomarker symmetric dimethylarginine (SDMA) cannot adequately differentiate between acute and chronic disease processes (Dahlem et al. 2017, Loane et al. 2022). Finally, while there is also some interesting data on the utility of ultrasound for differentiating acute and chronic kidney disease using the thickness of the renal cortex (Choo et al. 2023), you may have to talk to your radiologist about their comfort level in obtaining that value.

INITIAL DIAGNOSTICS AND MANAGEMENT FOR THE AKI PATIENT

CRITICAL QUESTIONS

1. What testing is helpful?
2. When should I monitor urine output?
3. How much fluid should I give?
4. What medications are indicated?
5. How do I manage oligo/anuria?

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For the non-septic/non-toxic AKI patient, beyond your baseline CBC, chemistry, urinalysis, and urine culture, you should consider testing for any infectious disease endemic in your practice area (Segev et al. 2024b). Depending on the area you practice in, this could range from leptospirosis to borreliosis. Unfortunately, while point of care testing is very accessible, its sensitivity and specificity for these diseases can be lacking (Liu et al. 2018, Troìa et al. 2018b), and so testing at the reference lab is frequently required. The second test to consider is ultrasonographic imaging. While the primary goal of ultrasound is to rule in/rule out ureteral obstruction requiring procedural intervention, it is also helpful for detecting renal lymphoma, Feline Infectious Peritonitis (FIP), and the presence of retroperitoneal fluid, which can be associated with oligo/anuria (Cole et al. 2019). There is some interesting research ongoing that is using contrast-enhanced ultrasound both from a diagnostic (Mannucci et al. 2019) and prognostic (Puccinelli et al. 2025) standpoint that may become relevant in the future. Similarly, while there are a large number of biomarkers that have been evaluated in AKI (reviewed in Nabity and Hokamp 2023), they are not widely available and are not required in the most recent diagnostic guidelines for veterinary AKI (Segev et al. 2024b).

Identifying patients who either currently are or are at risk for oliguria/anuria is crucial because it indicates a need for urinary catheter placement to monitor urine output (UOP) and is a negative prognostic factor (Worwag and Langston 2008, Rimer et al. 2022). While there are likely some biomarkers (i.e. NGAL) and ultrasonographic (i.e. retroperitoneal effusion) changes that could be useful red flags on presentation, as we discussed above, they are not always available. In my own practice, criteria on presentation that I consider "high risk" for oliguria/anuria are underlying disease (i.e. leptospirosis is high risk) and the presence of even a mild hyperkalemia on initial labs, along with retroperitoneal effusion on ultrasound. For these patients, I am much more vigilant about tracking UOP over the first 12–24 hours to promptly identify patients with decreased to absent UOP. I've included some risk factors that I look for when considering UOP monitoring in Table 18.2.

Unfortunately, there are times when urinary catheterization is technically challenging or requires sedation, such as with female dogs/cats and male cats. While there are ultrasound methods to serially evaluate urinary bladder volume (Yiew et al. 2019, Yang et al. 2022, Kendall et al. 2020, DiFazio et al. 2020), they are highly operator dependent and maybe unreliable when trying to gain anything more than a subjective idea of bladder size.

Appropriate fluid therapy can be challenging in the AKI patient because too little fluid risks ongoing renal damage due to inadequate perfusion, while too much risks volume overload that can have deleterious multisystemic effects (Langston and Gordon 2021). Unfortunately, there have been no randomized control trials in veterinary medicine around fluid therapy, with current expert opinion emphasizing the need for appropriate resuscitation and rehydration combined with fluid and electrolyte balance monitoring (Langston and Gordon 2021, Segev et al. 2024b). One plausible approach based on these principles is listed as follows (From Langston and Gordon 2021), understanding that monitoring patient response to therapy is more important than the initial treatment

Table 18.2 Some clinical risk factors the author uses when considering urinary catheter placement and urine output monitoring.

Considerations for urine output monitoring in nonobstructive AKI
Any hyperkalemia at presentation
Known predisposing disease (Leptospirosis, ethylene glycol/lily toxicosis, sepsis)
Failure of spontaneous urination or persistently small bladder on ultrasound after 6 hours of fluid diuresis
Lack of historical urination at home
Small bladder on presentation (unless has just urinated)

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protocol. You can also refer to Chapter 4 on fluid therapy for additional thoughts on intravenous (IV) fluid rates in this patient population.

- Resuscitation phase: 10 ml/kg over 15 minutes, up to 3 boluses.
- Optimization phase: 5 ml/kg over 20 minutes.
- Stabilization phase: Replace insensible lass at 22 ml/kg/day plus replacement of urine volume and additional loss (such as vomiting). The volume of medications and nutrition account for part of the fluid administered; the remaining volume is typically administered as a crystalloid fluid.
- Evacuation phase: Decrease fluid dose by 10–20% every 12–24 hours and monitor for dehydration.

Beyond fluid therapy, other medications are dictated by symptoms and the underlying disease process. As the majority of these patients will present with gastrointestinal signs, the use of antiemetics and antacids is likely beneficial (Ross 2022), though that has not necessarily been empirically proven. Many patients with AKI will have high blood pressure with amlodipine likely the treatment agent of choice (Geigy et al. 2011). Antibiotic selection can be slightly more challenging in term of drug choice and dose. For dogs, ampicillin/sulbactam (a renally excreted drug) is likely a reasonable choice, especially for those in Leptospirosis endemic areas. The exception to this would if you suspected Lyme nephritis in which case doxycycline would be a better empiric choice. Ampicillin has been shown to have altered pharmacokinetics in azotemic patients (Monaghan et al. 2021) and one study showed that twice-daily dosing is likely sufficient in azotemic dogs (Wang et al. 2025). In cats with AKI, a fluoroquinolone is likely a better empiric option given high rates of resistant *Escherichia coli* infections (Whitehouse et al. 2024), though dose adjustment is not required (Foster et al. 2023).

For a dehydrated patient with oliguria/anuria, the treatment goals are to either increase UOP pharmaceutically or externally reduce uremia via renal replacement therapy (RRT). While the complexities of RRT are beyond this book, it IS important for an emergency clinician to understand its indications and when to refer a patient for this modality. Medication options to try increase UOP include furosemide, fenoldopam, diltiazem, and mannitol. Unfortunately, as we'll see, none of these medications are consistently successful at achieving the desired diuretic effect. Furosemide (a loop diuretic) has several putative benefits in AKI for improving potassium excretion and reducing volume overload; however, these have not been proven in a veterinary clinical study. Fenoldopam (a dopamine agonist) has been shown to improve glomerular filtration (GFR) in healthy dogs (Kelly et al. 2016), though that benefit has not translated to improved outcomes in clinical AKI patients (Nielsen et al. 2015, Segev et al. 2018). Diltiazem was reported to improve renal recovery in leptospirosis patients in a very small study (Mathews et al. 2007), but subsequent work in healthy dogs showed no increase in GFR in response to this medication (Kelley et al. 2022). The osmotic diuretic mannitol may increase BUN and sodium excretion (Segev et al. 2019), though its efficacy in AKI patients has not been documented with the exception of one cat who received it for clothes dryer-induced heatstroke/AKI (Cudney et al. 2021). My own personal practice is that I will trial a single bolus dose of furosemide to see if it improves the UOP, and I will trial diltiazem in patients with suspected leptospirosis. However, I only give those patients a short amount of time to show improvement prior to recommending RRT.

The goal of RRT is NOT to heal the kidneys directly but rather to reduce the level of life-threatening uremia/hyperkalemia to give them the time to do so. Generally, accepted indications for RRT include serum creatinine >5 mg/dl that worsens despite fluid therapy, persistent (>6 hours) oliguria or anuria that has failed medical management, overhydration/fluid overload, and hyperkalemia causing altered cardiac function (Segev et al. 2024a). Overall prognosis with RRT can vary and be disease process dependent, so talking to the clinician who would be managing the patient after RRT is imperative.

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PROGNOSIS AND CLIENT COMMUNICATION

Overall mortality for feline AKI is 53% and 45% in dogs, with infection-induced AKI associated with a better prognosis than noninfectious (Legatti et al. 2018). I've listed some prognostic factors and scores in Tables 18.3–18.6, though I did not include any predictive scores for hemodialysis patients as that is not a modality we would unilaterally initiate as emergency clinicians.

Table 18.3 Common scoring systems for veterinary acute kidney injury, including the Veterinary Acute Kidney Injury (VAKI).

VAKI stage	Criteria
Stage 0	Creatinine increase <150% from baseline
Stage 1	Creatinine increase of 150–199% from baseline or creatinine increase of 26.5 µmol/l (0.3 mg/dl) from baseline
Stage 2	Creatinine increase of 200–299% from baseline
Stage 3	Creatinine increase of ≥300% from baseline or an absolute creatinine value >354 µmol/l (4.0 mg/dl)

Source: Thoen et al. (2011) with permission of John Wiley & Sons.

Table 18.4 Risk, Injury, Failure, Loss, End Stage (RIFLE).

RIFLE category	Serum creatinine criteria	Urine output criteria
Risk	≥1.5-fold from serum creatinine baseline or 50–25% decrease in GFR	<0.5 ml/kg/h for ≥6 hours
Injury	≥2.0-fold from serum creatinine baseline or 75–50% decrease in GFR	<0.5 ml/kg/h for ≥12 hours
Failure	≥3.0-fold from serum creatinine baseline or decrease in GFR ≥75%, or an absolute serum creatinine ≥354 µmol/l or with an acute rise of at least 44.2 µmol/l	<0.3 ml/kg/h for ≥24 hours or anuria ≥12 hours

Source: Lee et al. (2011) with permission of John Wiley & Sons.

Table 18.5 International Renal Interest Society (IRIS) AKI grade.

AKI grade	Blood creatinine	Clinical description
Grade I	<1.6 mg/dl (<140 µmol/l)	Nonazotemic AKI: **a.** Documented AKI: (historical, clinical, laboratory, or imaging evidence of AKI, clinical oliguria/anuria, volume responsiveness) and/or **b.** Progressive nonazotemic increase in blood creatinine: ≥0.3 mg/dl (≥26.4 µmol/l) within 48 hours **c.** Measured oliguria (<1 ml/kg/h) or anuria over 6 hours
Grade II	1.7–2.5 mg/dl (141–220 µmol/l)	Mild AKI: **a.** Documented AKI and static or progressive azotemia **b.** Progressive azotemic: increase in blood creatinine; ≥0.3 mg/dl ≥26.4 µmol/l) within 48 hours), or volume responsiveness‡ **c.** Measured oliguria (<1 ml/kg/h) or anuria over 6 hours
Grade III	2.6–5.0 mg/dl (221–439 µmol/l)	Moderate to severe AKI: **a.** Documented AKI and increasing severities of azotemia and functional renal failure
Grade IV	5.1–10.0 mg/dl (440–880 µmol/l)	
Grade V	>10.0 mg/dl (>880 µmol/l)	

‡ Volume responsiveness is defined as an increase in urine output to greater than 1 mg/kg/hr over 6 hours and/or a decrease in serum creatinine to baseline over 48 h.

Source: Adapted from Cowgill (2023).

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Table 18.6 Prognostic factors and scoring systems (see also Tables 18.3–18.5) for feline and canine AKI.

Study	Prognostic factors	Comment
Thoen et al. (2011)	VAKI score >0, age	See Table 18.3 for VAKI score, canine only
Lee et al. (2012)	Lower PCV, WBC, RBC, LDH, albumin, hypothermia, age	Feline only
Lee et al. (2011)	Worse RIFLE score, presence of diarrhea, high phosphorus	See Table 18.4 for RIFLE scoring system, canine only
Rimer et al. (2022)	Worse IRIS AKI grade, anuria	See Table 18.5 for IRIS AKI grade

PCV, packed cell volume; LDH, lactate dehydrogenase.

When I speak to clients about AKI, I try to make the following points:

1. Depending on where we are in the renal injury process when we start treatment, things may get worse before they get better.
2. Patients who go on to develop oliguria/anuria may need consideration for intermittent hemodialysis, which is expensive and can have some complications.
3. The kidneys are not fast healers, and while many animals with AKI can normalize their renal values (Bar-Nathan et al. 2022), the timeline for this is highly variable.
4. Sometimes patients are transitioned to subcutaneous fluid therapy at home to prevent a pre-renal exacerbation of their kidney injury during the healing process.

ACUTE ON CHRONIC KIDNEY DISEASE

Patients with underlying CKD are prone to having episodes of acute worsening/progression of their renal function that are labeled acute on chronic kidney disease, or AoCKD. If I were to ask a new graduate or senior veterinary student what the cause for this acute deterioration was, they would likely answer "pyelonephritis," even though that is an infrequent cause of this disease in dogs and cats (Dunaevich et al. 2020, Chen 2020). Interestingly, there is evolving thought that pancreatitis maybe a trigger factor for these flares, especially in cats (Dulude et al. 2024), which I think makes sense. There is also emerging evidence that supports the use of serum amyloid A to diagnose pyelonephritis (Kurtz et al. 2024) in cats, though as with many of the biomarkers, it is not clinically available in the emergency room. Ultimately, the workup and treatment for these patients is similar to that for AKI, though the long-term prognosis is much worse, likely because these patients had compromised renal function to begin with (Dunaevich et al. 2020, Chen 2020). One study using artificial intelligence/machine learning showed that creatinine levels after 48 hours of hospitalization correlated with a higher mortality risk. Other potential prognostic factors identified in that study were older age, presence of nephroliths, lack of spontaneous eating at 48 hours, and lower body weight (Renard et al. 2021).

MANAGING URETEROLITHIASIS

Bilateral ureterolithiasis causing a post-renal azotemia can occur either as an acute or acute on chronic disease process with the former more common in younger cats and more likely to cause complete obstruction (Geddes et al. 2023). As we discussed above, evaluating for this disease process is one of the main indications for abdominal ultrasound in the azotemic patient, and I would argue its importance only increases in animals with palpably large, painful kidneys. In one study,

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the presence of renal pelvic dilation on point of care ultrasound (Figure 18.1) was a large risk factor for ureteral obstruction in cats (Beeston et al. 2023).

For patients with evidence of ureterolithiasis, the questions to answer are, are they causing an obstruction and do they require procedural intervention to remove? While traditionally, patients with renal pelvic dilation and ureteral dilation proximal to an object were considered obstructed, that maybe an underestimate of cats when compared to other modalities such as CT and pyelography (Lamb et al. 2018, Testault et al. 2021, Lemieux et al. 2021). However, for our purposes, we will only consider those with clear evidence of obstruction as I do not think CT on an emergent basis for the diagnosis of ureterolithiasis is indicated because we are unlikely to immediately send that patient for procedural intervention. While ureteral stent or subcutaneous ureteral bypass (SUB) likely leads to prompter relief/renal decompression, they can be expensive, and the need for follow-up maybe challenging for some owners. Therefore, our job as emergency clinicians is to both consider medical management options as shown in Table 18.7 and discuss their probability of success.

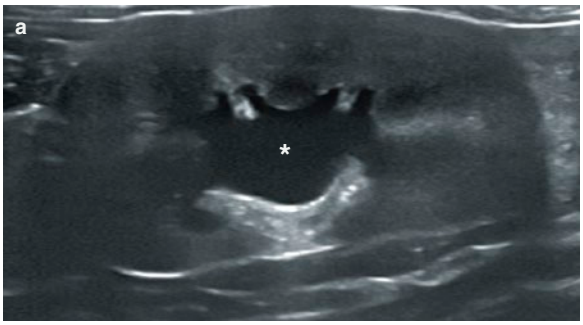


FIGURE 18.1 Renal pelvic dilation in an azotemic feline on point of care ultrasound (POCUS) should raise your index of suspicion for ureteral obstruction in the azotemic feline. Dilated renal pelvis secondary to ureteral obstruction (*). *Source*: Griffin (2020) with permission of SAGE Publications.

Table 18.7 Medical management options for feline ureterolithiasis. The author typically reaches for prazosin in these cases.

Drug	Drug type and mechanism of action	Dose in cats	Comments
Amitriptyline HCl	Tricyclic anti-depressant	0.5–2 mg/kg orally every 24 hours	May cause sedation
	Ureteral smooth muscle relaxation of ureter by opening of potassium dependent voltage channels	(Extra-label use and dose)	Multiple potential drug interactions, consult veterinary formulary prior to use
Glucagon	Polypeptide hormone	0.05–0.1 mg/cat intravenous every 8–24 hours	Vomiting, diarrhea, nausea, and hypokalemia may result
	Ureteral smooth muscle relaxation and relief of ureteral colic	(Extra-label use and dose)	
Prazosin	Alpha-1 antagonist	0.25–0.5 mg/cat orally every 12–24 hours	Sedation, third eyelid elevation, and hypotension can result
	Ureteral smooth muscle relaxation		

(Continued)

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Table 18.7 (Continued)

Drug	Drug type and mechanism of action	Dose in cats	Comments
Tamsulosin	Alpha-1 antagonist	0.004–0.006 mg/kg orally every 12–24 hours	Safety, efficacy, dose have not studied in cats
	Ureteral smooth muscle relaxation	(Extra-label use and dose) May take 3 days to be effective	May cause lethargy, vomiting, and hypotension, dose size will require compounding
Mannitol	Osmotic diuretic to increase flow of urine through the ureter	0.25–1 g/kg intravenous over 20–30 minutes; may repeat if effective	Contraindicated with anuria and fluid overload
		60–120 mg/kg/hour CRI	May cause fluid overload so use caution with cardiac disease and overhydrated patients
Furosemide	Loop diuretic for increased flow of urine through the ureter	0.1–1 mg/kg intravenous, if effective, can use 0.25–1 mg/kg/hour CRI	Can increase urine output but does not affect GFR
		(Extra-label use and dose)	Can result in hypochloremia and hypokalemia
Amlodipine besylate	Calcium channel blocker	0.625–1.25 mg orally per cat every 24 hours	Limited veterinary evidence for ureteral disease
	Smooth muscle relaxation via blockade of calcium channels	(Extra-label use and dose)	Slow onset of action May cause anorexia and hypotension

Source: Clarke (2018)/John Wiley & Sons.

The original study on medical management for UO appeared to show a low success rate of around 10% (Kyles et al. 2005), while a later study was a bit better at around 30–40% with smaller (<2 mm) and more distal obstructions most likely to be passed (Merindol et al. 2023). In the absence of life-threatening azotemia or hyperkalemia, I will typically allow roughly 24 hours on medical management.

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Managing the Chronic Disease Patient

CHAPTER 19

CRITICAL QUESTIONS

1. Is this a progression of their previously diagnosed disease?
2. Is this a side effect of the medications treating the patient's chronic disease?
3. Is there room to increase the dose of a current medication or a potential additional medication to start?

INTRODUCTION

As a 24-hour service, we are frequently called upon to see patients being managed for chronic diseases either because it is after hours or because their primary clinician is unavailable to see them for follow-up. While these patients can be initially daunting, I have found that utilizing the critical questions above to be very helpful in guiding my diagnostic thought process. We've already discussed the evaluation of the chronic seizure, diabetic, cardiac, and renal patients in other chapters. The goal is not to cover every single disease process, but we'll expand our analysis to some other common diseases not otherwise discussed and also review the common medication side effects used to manage some of those diseases. In human medicine, the most common "chronic" diseases (that are relevant to veterinary medicine) are respiratory disease, diabetes, hypertension, epilepsy, and pain (Chiu et al. 2022). As you'll notice by a general lack of citations, there hasn't been a large amount of research into the presentation and management of these cases through the emergency service. I've included some general history tips in Table 19.1 that we'll discuss further later in the text.

IS IT AN OLD OR A NEW PROBLEM?

The first question to ask is whether or not the patient's presenting complaint could plausibly be due to a progression of a previously diagnosed disease. While some of these are fairly simple associations (i.e. the pale dog with a history of immune-mediated hemolytic anemia [IMHA]), others may require a bit more thought and diagnostic testing. Our core concepts discussed in earlier

Table 19.1 An appropriate clinical history is critical for raising or lowering your index of suspicion that the patient's presenting complaint is related (or not) to a previously diagnosed disease.

History tips for the chronic disease patient
Are these symptoms similar to when he/she was originally diagnosed?
Has he/she had previous flareups of this disease before? Were they similar?
Have any new medications been added? Or medication doses changed?
When was he/she last seen by their primary clinician for this disease?
When was the last time he/she had any lab work or imaging performed that evaluated their underlying disease?

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Table 19.2 Some considerations when evaluating the oncology patient through the emergency room.

Historical questions	Comments
When was the cancer diagnosed?	This may give you an idea of how close the patient is to their particular cancer's median survival time
How has it been treated to this point?	—
When was the last dose of chemotherapy?	The majority of chemotherapy nadirs are usually 5–7 days post administration
When was the last "staging" of the cancer?	The longer it's been since the last staging, the higher the likelihood of relapse
Physical exam (PE) findings	
Fever present?	May indicate chemosepsis in patients undergoing chemotherapy
Evidence of cancer/tumor regrowth on PE or imaging?	If there are enough historical risk factors, always consider imaging to evaluate for progressive disease
Overall considerations	
Is the patient sick enough to warrant hospitalization?	See Chapter 7 for more details
What are the owner's goals of care?	—

Chapters 2, 3, and 7 for diagnostic testing/imaging and hospitalization are still valid here, though they have to be taken in the context of each individual patient.

Perhaps the easiest disease category to conceptualize this question for is with oncology patients. Barring complete surgical removal, the vast majority of cancers will either recur or progress despite medical management. Therefore, in patients with a previously diagnosed malignancy, you should evaluate through your physical exam, lab testing, or imaging whether a tumor has recurred locally or if their signs are related to metastatic disease. For patients with progressive disease, ascertaining the clients' goals of care (i.e. definitive care, palliation, euthanasia) is crucial in guiding treatment approach as we'll discuss in a later chapter. I've included a checklist for evaluating the oncologic patient in Table 19.2.

The next set of patients we'll discuss is those with chronic cough who present for an acute exacerbation of their clinical signs. The most common causes of chronic (noncardiac) cough include tracheal collapse, chronic bronchitis, and neoplasia in that order (Blazquez et al. 2025). For dogs with minimal risk factors of pneumonia or lung changes on point of care ultrasound (POCUS), thoracic radiographs are likely of low diagnostic yield. Since patients with these diseases are at higher risk for having bacterial tracheobronchitis compared to the general population (Blazquez et al. 2025), empiric antibiotics maybe reasonable if their signs were reasonably controlled on their prior medication (if any). Similarly, dogs with chronic bronchitis have also been shown to have an altered pulmonary microbiome (Ericsson et al. 2020) that may leave them at increased risk for secondary infection. If you feel like the risk of infection is low, then considering modification to their steroid dose, antitussive medications, and/or inhalers is reasonable. Figure 19.1 shows a summary flowchart of diagnostic and therapeutic options to consider for acute on chronic coughing dog. Note that for cats who are coughing, always evaluate their historical and risk factors for feline asthma (history and signalment) and consider thoracic radiographs for cases concerning other causes.

For the acute on chronic coughing patient, regardless of etiology, you should always make sure that they aren't having any difficulty breathing that may warrant hospitalization for oxygen therapy. These patients should also usually have repeat thoracic imaging to evaluate for new pathology, including a potential echocardiogram to look for pulmonary hypertension.

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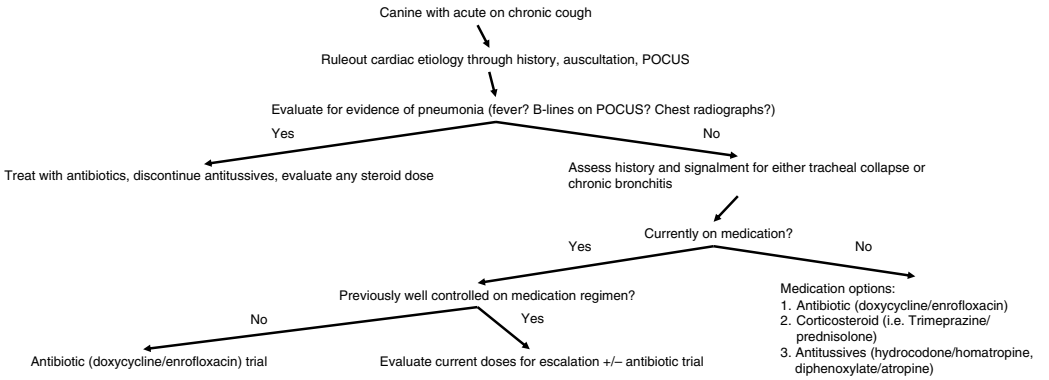


FIGURE 19.1 The author's flowchart and approach to the acute on chronic coughing dog. The main points are to rule out a cardiac cause and to evaluate risk factors for an infectious (pneumonia, infectious tracheobronchitis) etiology. Courtesy of the author.

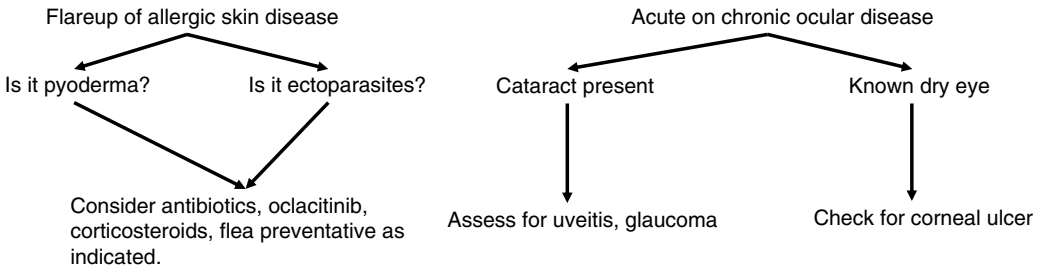


FIGURE 19.2 Some considerations for assessing select chronic conditions. For additional information on allergic disease, consult Miller et al. (2023), and for ocular disease, consider Wynne (2020).

Patients with chronic gastrointestinal disease who present with gastrointestinal signs are certainly frustrating since the majority of these are already on symptomatic management. For these patients, our job as emergency clinicians is to assess for disease severity using lab work/imaging and need for hospital admission, as discussed in Chapter 14. For patients who do not meet hospitalization criteria or who do not necessarily warrant emergent diagnostics, I will typically look for areas of symptomatic management to augment (i.e. additional antinauseals, antidiarrheals, appetite stimulants, diet change, etc.). However, you should always evaluate the need for these patients for hospitalization (see Chapter 7) in terms of their hydration status and appetite. Any patient with "new" signs, such as new hematemesis, or new onset hematochezia/melena should also have repeat abdominal imaging to evaluate for additional pathology.

There are some miscellaneous chronic diseases that, while I wouldn't necessarily classify as "emergencies" such as dermatologic and ocular, will still sometimes present through the emergency room. Some general considerations for these patients are shown in Figure 19.2, though other sources exist that cover their management in more detail.

Finally, if you feel like a chronic disease patient's clinical signs are due to an exacerbation of their chronic disease, I always try to consider whether or not there is room for dose increase in their current medication or a potential additional medication that can be considered. You should also always recommend that the owner contact the pet's primary clinician to arrange follow-up and mention that you will notify that doctor of the patient's visit (via a referral letter) through the emergency service.

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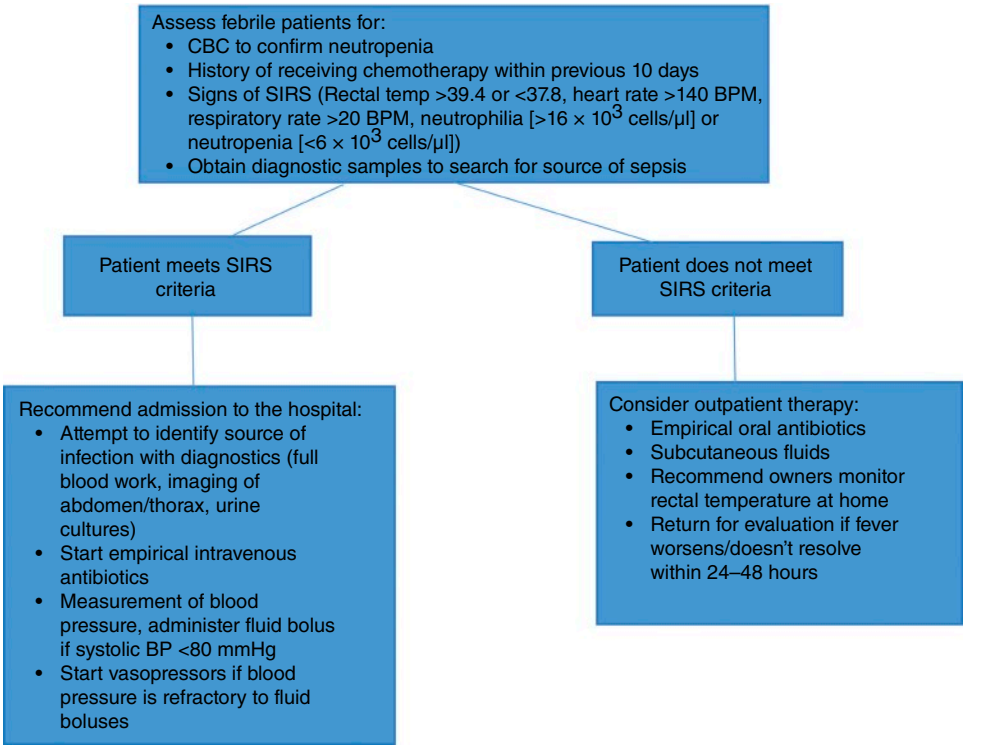


FIGURE 19.3 Initial approach and hospitalization decision-making for febrile oncology patients. *Source*: Tumielewicz et al. (2019) with permission of John Wiley & Sons.

IS IT THE MEDICATION?

The second important question to consider for the chronic disease patient is whether their medications could be the cause of the presenting complaint. Always ask the owner if they are actually *giving* the medications as prescribed and also whether or not there have been any recent dose adjustments.

As discussed above, the easiest "sample" group of medications to grasp this concept is with oncology patients on chemotherapeutics. Many patients in chemotherapeutic protocols are at increased risk for infection and potentially chemosepsis. The neutrophil nadir point for most chemotherapeutics is roughly 5–7 days post administration, though patients receiving carboplatin or lomustine may have either delayed or second nadirs (Bisson et al. 2018). Current treatment recommendations for chemosepsis in terms of inpatient versus outpatient care are shown in Figure 19.3.

One other drug class to mention here is the tyrosine kinase inhibitors (i.e. toceranib among others), which are becoming prescribed for a broadening array of cancers. Beyond the "standard" myelosuppression, these drugs have the potential to cause hepatoxicity/proteinuria in cats as well as gastrointestinal hemorrhage in dogs (Bavcar and Argyle 2012, Žagar and Schmidt 2023). For patients on these medications, make sure to evaluate for any evidence of melena or anemia as well as any new onset liver enzyme elevations.

Beyond the chemotherapeutics, almost every medication has the potential to cause some degree of side effects. I've listed some of the most common ones used in chronic disease management that might present acutely in Table 19.3, categorized by mechanism/indication.

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Table 19.3 Some common drugs used to treat chronic disease and their associated side effects.

	Medication	Adverse effects	Additional information		Medication	Adverse effect	Additional information
Cardiac				**Endocrine**			
	Furosemide	Acute renal failure	Abbott and Kovacic (2008)		Methimazole	Worsened renal function, cytopenias, hepatotoxicity	Daminet et al. (2014)
	ACE inhibitors	Acute renal failure	Lefebvre et al. (2007)		Trilostane	Iatrogenic Addison's	Lemetayer and Blois (2018)
	Antiarrhythmics	Hypotension	Romito et al. (2024), Romito et al. (2025)				
Immunomodulators							
	Prednisone	Diabetes mellitus, GI ulceration, iatrogenic Cushing's volume overload (cats)	Aharon et al. (2017)				
	Cyclosporine	Gastrointestinal, gingival hyperplasia, hepatotoxicity	Viviano (2022)				
	Mycophenolate	GI ulceration, cytopenias	Fukushima et al. (2021)				
	Leflunomide	Diarrhea, hemorrhage, thrombocytopenia	Sato et al. (2017)				

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Table 19.4 Summary approach to the chronic disease patient presenting through the emergency services.

Clinical considerations
Is the presenting complaint similar to when the pet was originally diagnosed or similar to previous flares?
What medications are being used to treat the disease and have there been any missed doses or dosage changes?
Are repeated laboratory or diagnostic testing tests used to evaluate for either chronic disease progression or new pathology?
Are the patient's signs severe enough to warrant hospitalization?
Are there symptomatic or empiric medications I can prescribe to alleviate their clinical signs?

CONCLUSIONS

While the chronic disease patient can be a challenge for the emergency clinician, the checklists above as well as the summary approach seen in Table 19.4 should provide a solid foundation in evaluating these patients. Most importantly, the goal of the visit is NOT to cure the chronic disease but rather to evaluate the severity of clinical signs and hopefully improve them.

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Toxin Ingestion Management

CHAPTER 20

CRITICAL QUESTIONS

1. Should I induce emesis?
2. Should I give charcoal?
3. Should I give intralipid emulsion?
4. Is fluid diuresis helpful?
5. Should I refer for extracorporeal therapy?

INTRODUCTION

Accidental ingestions are an incredibly common presentation to the emergency service. While it is never wrong to consult a poison control center for information (especially uncommon ingestions), there are some common themes for the management of most toxicities that you should be aware of as an emergency clinician. My goal with this chapter is not to provide an exhaustive list of management approaches for every individual ingestion, but rather to talk broadly about the treatment options you should consider implementing.

EMESIS

We discussed the use of emesis to remove gastric foreign bodies in Chapter 19, but here we consider it as a decontamination technique. Within this section, there are two questions to answer: what is the longest time period since ingestion where emesis is helpful, and second, what is the best agent to use?

While ideally emesis is induced as close to ingestion as possible to maximize the amount of toxin retrieved, this is frequently a challenge given the unknown timeframe since many ingestions. There is no "hard" data to support a time limit on when emesis is ineffective (Bates et al. 2015), though one expert opinion level reference does have a table and lengthy flowchart surrounding decision-making for emesis induction (Stern 2025). However, the better question to ask is whether or not there is a general contraindication to inducing emesis (Table 20.1) rather than placing a strict time limit post ingestion as gastric transit time can significantly vary (Tolbert et al. 2022) between pets. While it may seem counterintuitive, brachycephalic pets do not have a higher complication rate with emesis induction compared to other breeds (Harkins et al. 2024).

The second question is what is the best agent to use to induce emesis? In dogs, my agent of choice is likely apomorphine, which has been shown to be superior to ropinirole (albeit in one study) and can be given via several different routes, including both intravenous (IV) and intranasal routes (Manley et al. 2024, Reeves et al. 2025).

Table 20.1 Contraindications for induction of emesis in the intoxicated patient.

Contraindications for emesis induction
Compromised upper airway function (i.e. laryngeal paralysis)
Concurrent megaesophagus
Mentally inappropriate, unable to protect airway
Caustic substance ingestion (i.e. bleach)
Volatile substance ingestion (i.e. hydrocarbons)

Source: Lee (2015).

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To provide both sides though, another study said the two agents were of equivalent efficacy (Lee et al. 2025). If one agent fails, it is also safe to administer the other (Saloranta et al. 2024).

Multiple agents have been described to induce emesis in cats including xylazine, hydromorphone, and dexmedetomidine (Thawley and Drobatz 2015, Willey et al. 2016, Nystrom et al. 2019). However, the route I think is the most effective is 20 mcg/kg of dexmedetomidine given transmucosally and then reversed with half the volume of atipamezole intramuscularly post emesis (Maxwell et al. 2024).

ACTIVATED CHARCOAL

The next potential decontamination step post emesis is considering whether or not activated charcoal is beneficial. Despite a general move away from activated charcoal in human medicine except for selected toxicities (AACT 2005, Hoegberg et al. 2021), the use of single dose to multidose activated charcoal continues to be recommended in veterinary medicine (Lee 2013, Peterson 2013, Peterson and Fluegeman 2013) especially for NSAIDs, bromethalin, and cholecalciferol. However, if you follow the chain of citations, the majority of the evidence for its use is limited to expert opinion, conference proceedings, or studies from over 40 years ago. As with the induction of emesis, I think it's important to consider contraindications to charcoal administration (Table 20.2) and I do not personally use multiple doses for NSAID ingestion as it has not been associated with better outcomes when studied (Koenigshof et al. 2015).

My opinion is that single-dose charcoal is reasonable in the majority of cases and multidose charcoal maybe reasonable in certain cases for high-dose bromethalin or extended-release productions (Bates 2025) despite a lack of proven efficacy. Poison control will continue to recommend its administration and so from a liability standpoint, you may need to abide by some of them while acknowledging the low risk of clinically significant dehydration hypernatremia (Mix et al. 2019, Young et al. 2025) and rarely, gastrointestinal obstruction (Farrell et al. 2020). I do not routinely recheck electrolytes for single-dose charcoal administration due to the very low incidence rate of clinically significant hypernatremia but will usually give a dose of subcutaneous fluids unless otherwise contraindicated.

Table 20.2 Contraindications for the administration of activated charcoal (AC).

Contraindications for Administration of AC	
Central nervous system depression (decreased level of consciousness)	Diminished gag reflex or compromised airway (increased risk of aspiration pneumonia)
Time frame for benefits of AC is exceeded (e.g. late stage presenting with clinical signs already present)	Toxicants that do not reliably bind, including ethylene glycol, xylitol, and heavy metals
Ingestion of corrosive/caustic agents	Ingestion of hydrocarbons
Dehydration or hypovolemia	Ingestions of salt (e.g. paintballs, homemade play dough, ocean water, table salt)
GI obstruction or perforation	Ileus GI stasis
Imminent endoscopy or surgery	Hyperosmolar states (e.g. renal disease, diabetes mellitus, psychogenic polydipsia, diabetes insipidus)
Hypernatremia	Underlying medical condition predisposing to aspiration pneumonia (e.g. laryngeal paralysis, megaesophagus, upper airway disease)

GI, gastrointestinal.
Source: Lee (2015) with permission of Elsevier.

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INTRALIPID THERAPY

The use of intralipid emulsion (ILE) was originally described for lidocaine toxicity but has since expanded in an attempt to decontaminate other lipophilic drugs or unknown toxicities with varying success (Markert et al. 2023). Currently, it has shown probable benefit in feline permethrin toxicosis (Peacock et al. 2015), ivermectin toxicosis (Jourdan et al. 2015), tremorgenic mycotoxicosis (Kormpou et al. 2018), and several others (reviewed in Gwaltney-Brant and Meadows 2018). While there are multiple case reports of ILE improving signs in other toxicities, the number of confounding treatment factors makes it difficult to determine how much of an impact lipids had on the patient's improvement. Unfortunately, the role of intralipid for THC toxicosis (for which I use it most frequently) has not been fully elucidated through a randomized control trial, though its use is well described in case reports and case series (Williams et al. 2015, Markert et al. 2023). Even in human medicine, the current recommendations for use of ILE are mostly neutral due to a lack of evidence for the majority of medications (Gosselin et al. 2016). Theoretically, a patient with toxic signs from a lipophilic substance would be expected to benefit from ILE. If you are in doubt as to whether a drug is lipophilic, there are multiple open-source resources such as PubChem where you can look up the octanol/water coefficient or LogP/LogD where values greater than 1 correlate to lipophilicity (Jones et al. 2024). My personal practice is to use ILE for lipophilic intoxications or in neurologic cases that I suspect to be due to an unknown toxicity. When weighing the risk/benefit of this intervention, ILE has been shown to occasionally lead to a transient worsening of mentation as well as some potentially significant acid/base and electrolyte changes, likely due to an increase in serum triglyceride levels, though almost all changes resolved within 24–48 hours (Markert et al. 2023, Kiwitz et al. 2024). The implication of this is that if something changes with your patient post treatment, make sure you are reassessing those values.

INTRAVENOUS FLUID THERAPY

Intravenous fluid therapy is frequently recommended for patients who have ingested potentially nephrotoxic medications such as NSAIDs, grapes/raisins, ethylene glycol, cholecalciferol, and lilies in cats. Traditionally, high fluid rates were recommended to "flush" or "diurese" the kidneys (Lee 2013) with the thought being that a higher urine output would hasten renal excretion of the toxin and thus lead to a faster reduction in plasma toxin levels. And while one study in piglets did show fluid therapy led to increased clearance of amikacin (Dhondt et al. 2021), indicating there is occasionally an effect of fluids on renal clearance, the appropriate rate, route, and duration of therapy are unknown. Furthermore, the effect of intravenous fluids on urine output is mediated more by increasing renal blood flow than increasing the glomerular filtration rate (McClellan et al. 2006). My own practice is to recommend fluid therapy (though not high rate) for patients who have ingested a nephrotoxic dose of NSAIDs and lilies to maintain renal perfusion. A recent study in lily toxicosis cats showed there was a 14.5% survival difference between those managed as inpatients and outpatients, indicating a probable benefit to IV fluid therapy (Lam et al. 2025). The incidence of ethylene glycol and cholecalciferol ingestion is less common, though for the latter, current initial recommendations are to monitor serial calcium/phosphorus levels for potential elevation as the goal of fluid therapy is to increase calciuresis, not improve excretion (Peterson and Fluegeman 2013). Grape and raisin ingestion raises several challenges due to an unknown toxic dose and potential for AKI. The largest literature review indicated that 75% of dogs who ingested grapes/raisins were asymptomatic with a 15% rate of AKI (Downs et al. 2024). However, if you read deeper, the majority of the AKI cases were from older studies with the larger, more recent studies having a much lower rate of 1–7% with most only having a single case reported. Furthermore, the single largest study had only one case out of 600, and there appeared to be no difference in renal complications between patients treated as inpatients or outpatients (Croft et al. 2021) with another recent study showing 1 patient out of 95 developing AKI (Dijkman et al. 2022). My own approach

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is to discuss the pros and cons with owners and ultimately let them make a decision. Since we don't know the toxic dose, it is impossible to guarantee that their individual dog won't develop AKI, though the incidence is rare (I have never seen one). While there are potential physiologic advantages to inpatient as opposed to outpatient care, definitive benefit has not been proven. One middle ground I have employed for treating patients as an outpatient is to have them come back for several days after presentation to check renal values.

EXTRACORPOREAL THERAPY

Besides emesis/charcoal, extracorporeal therapy represents another mechanism to "decontaminate" the intoxicated patient by directly removing the drug or toxin from the bloodstream. While the mechanisms behind these modalities are beyond the scope of this chapter, you should be aware of some general indications for their use so you can make appropriate recommendations for their implementation. The first question you should ask is, is this a drug or a toxin that is even amenable to removal? The most common drugs/toxins where you should consider extracorporeal therapy are NSAIDs and baclofen (Groover et al. 2022) though there are case reports where it was helpful in treating toxicities from 5-hydroxytryptophan (Her et al. 2024), metaldehyde (Teichmann-Knorrn et al. 2020), and cannabinoids (Culler and Vigani 2019) among others.

The second question, given its expense and potential complications, is whether or not extracorporeal therapy will be beneficial to the patient. Current recommendations for use of extracorporeal therapy are when you can't decontaminate the patient, there is a high risk for severe toxic signs, no available antidote, or if the patient can't metabolize the toxin on their own (Londoño 2023). While there have been no randomized control trials comparing extracorporeal therapy with conventional management, one retrospective study did appear to show a reduced risk of AKI in patients receiving therapeutic plasma exchange who had ingested nephron/neurotoxic doses of NSAIDs compared to conventional therapy (Chalifoux et al. 2023).

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Client Communication and Palliative Care

CHAPTER 21

CRITICAL QUESTIONS

1. Will the diagnosis be helpful?
2. What are the owners' goals of care?
3. What clinical signs can I palliate?
4. How do I recommend euthanasia?

INTRODUCTION

Client communication in the emergency room is a distinct challenge because we are meeting families for the first time under stressful circumstances. Furthermore, since the vast majority of our clients have little to no medical knowledge, we also have to quickly educate these families about complex medical topics while forcing them to make critical decisions in a short time period (Bateman 2007). While we've talked about certain medical communication points for specific diseases before, this chapter is more focused on communicating about the difficult choices when there is no one obvious or perfect pathway forward. My hope is that the approach we talk about here will help you share the decision-making process with those families, which will not only relieve some of the burden on you but also improve the client's satisfaction with your care (Ito et al. 2022). There are also multiple resources about client communication that I will include at the end of the chapter.

IS THE DIAGNOSIS HELPFUL OR IMPORTANT?

While it may sound overly simplistic, there really are only three treatment options for ANY disease process: definitive management (i.e. antibiotics for a urinary tract infection), symptomatic (i.e. antiemetics for vomiting), and euthanasia. Our job as veterinarians is not always to just recommend the next test toward getting the "answer" nor is it to always push for the treatment with the "best" long-term outcome. The real job is to take the diagnostic/treatment options we know, and best align them with the owners' goals for their pet, as shown in Figure 21.1.

In reality, there are medical, pet, and family factors that go into what makes the *best* approach for each individual situation (Figure 21.2) and our job as the veterinarian is to obtain and provide information that is helpful in guiding an owner's decision about which of the three treatment approaches is best for them and their pet.

There are a couple of examples that I think illustrate these points reasonably well. The first is a highly anxious and aggressive large breed dog who presents with severe peripheral lymphadenopathy consistent with lymphoma. While the approach associated with the longest survival time would be a chemotherapy protocol (i.e. CHOP), the repeated visits to the veterinarian may not be best for the dog, a pet-centric factor. Similarly, for a patient presenting with spontaneous hemoabdomen, if a family would not take their pet to surgery, staging to look for the presence of a mass and metastasis will not lead to another valid treatment option. However, if knowing there is a mass present would help a family make a decision about euthanasia, then now that becomes a helpful test. The ability to communicate how the results of a test will drive a treatment decision is a crucial aspect of shared decision-making.

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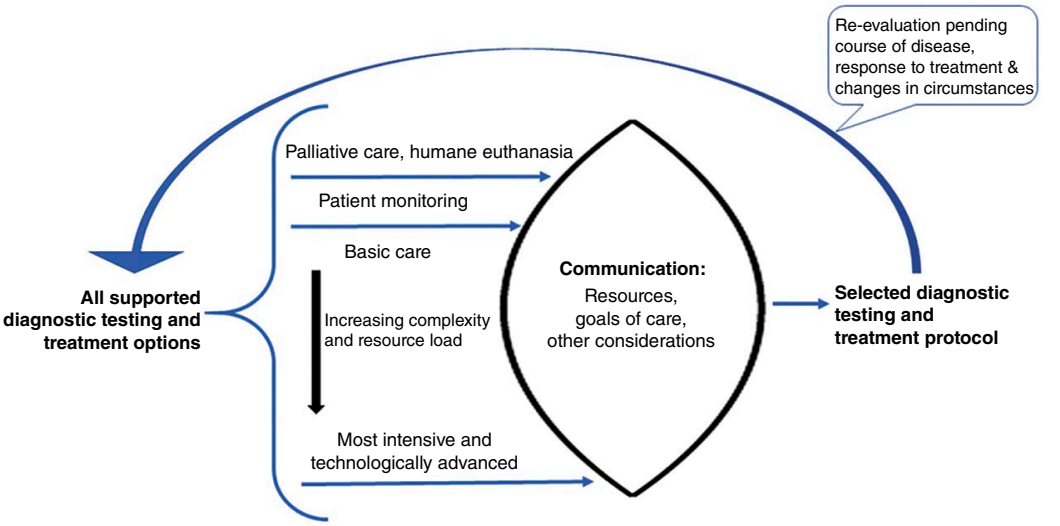


FIGURE 21.1 A spectrum of care ranging from palliation/hospice and euthanasia up through advanced therapy has to be interpreted in light of the owner, pet, and veterinary resources to yield the optimal diagnostic and therapeutic plan for each individual patient. *Source*: Brown et al. (2021)/American Veterinary Medical Association.

Pet factors	Owner/family factors	Veterinary factors
Medical condition	Financial resources	Level of experience
Cognitive function	Time/logistical constraints	Access to advanced diagnostics/therapeutics
Behavior at veterinarian	Culture/values	Previous cases
Quality of life	Past experience with other pets	Outcome/prognosis

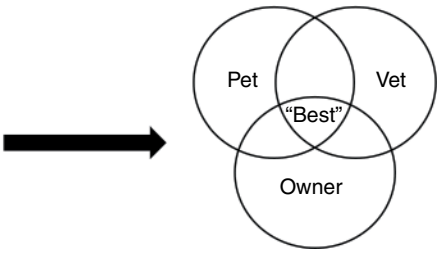


FIGURE 21.2 The "best" diagnostic and/or treatment approach is one that appropriately balances owner, pet, and veterinary factors. *Source*: Brown et al. (2023)/American Veterinary Medical Association.

WHAT ARE THE OWNERS' GOALS OF CARE?

Having an honest conversation about a client's goals of care for their pet can be emotional and intimidating, especially for the newer doctor. While there are tools available to help guide this discussion (Goldberg 2019, Lummis et al. 2020), I will take you through some of the simple steps I use when approaching this conversation. The first is to make sure the family understands what disease process is occurring. If further testing is required to differentiate between two diseases, make sure they understand how the results of those tests might influence treatment approaches as discussed above. Second is to outline the spectrum of care in terms of definitive and palliative options as well as the likelihood of success and outcome for each approach to the best of your ability. Finally, ask to make sure the family understands what you have just said, because that opens the floor for questions. Ideally, at the end of this conversation, the family should feel like they have all of the information needed to decide about either further diagnostic testing or which treatment option to pursue.

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HOW TO BEST PROVIDE PALLIATIVE/HOSPICE CARE

A more formal definition for palliative care is treatments aimed at relieving symptoms and optimizing quality of life for patients with terminal disease, while hospice care is geared more toward the last stages of life when aggressive treatment is not being pursued. My personal concept of these approaches is to relieve the pain, stress, and discomfort of a disease without actually directly treating the disease itself, which is closer to hospice rather than palliation. While I will focus on the tools we have for symptom relief, I think it is also important to acknowledge that there are other aspects of palliative/hospice care, as shown in Figure 21.3.

In developing an appropriate palliative/hospice care plan, it's important to have a list of potential symptoms detrimental to a pet's quality of life along with the medical and therapeutic interventions you have to combat them, as shown in Table 21.1.

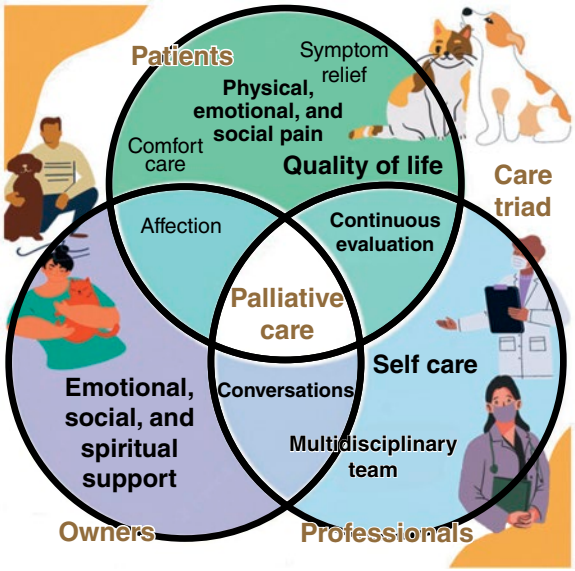


FIGURE 21.3 Effective palliative/hospice care encompasses multiple non-medical concepts beyond symptom relief. *Source*: Paz et al. (2024)/John Wiley & Sons/CC BY 4.0.

Table 21.1 Palliative/hospice medications available to treat common clinical signs.

Symptom	Medical intervention
Pain	NSAIDs, opioids, acupuncture, amantadine, gabapentin
Vomiting	Antiemetics, antacids
Decreased appetite	Capromorelin, mirtazapine, palatable foods, corticosteroids
Cough	Antitussives (hydrocodone, dextromethorphan)
Dyspnea	At home oxygen, palliative thoracocentesis, bronchodilators
Agitation	Trazodone, SSRIs, gabapentin

NSAID, nonsteroidal anti-inflammatory; SSRI, selective serotonin reuptake inhibitor.
Source: Paz et al. 2024/John Wiley & Sons/CC BY 4.0.

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HOW TO DISCUSS/RECOMMEND EUTHANASIA

Introducing the concept of euthanasia as a treatment option into the conversation can sometimes be a daunting but necessary task. For disease conditions where euthanasia is a reasonable option, I will directly bring it up at the end of my list of treatment options, followed a statement such as "I don't want you to feel like euthanasia is the only option here, but I bring it up so that you know that it's okay to consider for this situation." In this manner, I have directly given the family permission to consider euthanasia in a nonjudgmental way. While there are multiple quality of life assessment scores available for owners to guide euthanasia decision-making, I use two simple questions for families in considering this issue. The first question is, how bad is their current quality of life? The second is, can we improve that quality of life and for how long? If I feel like I do not have the ability to appropriately relieve a pet's suffering, then I will tell them that. If I think I can help relieve some (or all) of their clinical signs using either definitive treatment or palliative/hospice care, then it's up to the family to decide if that tradeoff is worth it.

Sadly, we are frequently required to rapidly provide owners with terrible news about a beloved pet that can be shocking and difficult to process. It is crucial to allow owners the necessary time to grasp the medical and emotional complexities of their pet's disease process. As long as their pet's medical condition allows it, providing the pet with symptomatic relief to provide some quality time with the family at home to say goodbye can be a valuable option. It can also allow them time to arrange for an at-home euthanasia if that aligns with their goals of care. In these situations, I always go through the signs of disease progression so that owners do not inordinately delay the euthanasia process.

It can be challenging to navigate family dynamics when different owners have different approaches to end-of-life care or when owners disagree with your euthanasia recommendation. I wish I had a perfect strategy for handling these situations, but each one is unique in its own way. Ultimately, everyone involved has a different journey toward the passing of their pet, and they have to feel comfortable walking down that path. I feel my job as the veterinarian is to provide guidance and advocate for the pet when I feel like they are suffering (i.e. owner wants to take home a dyspneic animal). However, it is up to the family members to work out any disagreements among themselves and make the best decision possible. If you have a social worker on staff, they can also be invaluable in helping families discuss the complex emotional and familial issues surrounding euthanasia.

CONCLUSIONS/SUMMARY

Client communication, especially surrounding quality and end of life, is a skill borne out of the innate compassion that drove us all to enter veterinary medicine in the first place. Sadly, the more situations you encounter, the more tools you will develop for handling each unique one. Some of the key points from this chapter are as follows:

- Understanding the owners' goals of care and what treatments they would/would not pursue is critical in guiding what diagnostic testing is helpful.
- There are pet, owner, and medical factors that guide these decisions, and it's important to weigh all of them appropriately.
- For owners who decline "definitive" treatment for their pet's disease, consider hospice care depending on the symptoms present.
- When appropriate, euthanasia should be brought up alongside other treatment options to give an owner permission to consider it as a pathway forward.
- We (including us as veterinarians) have a different journey to take toward the passing of our patient/pet. The goal for us as a vet is to provide guidance and support along that pathway while advocating for our patient as necessary.

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Cardiac Emergencies

CHAPTER 22

CRITICAL QUESTIONS

1. How much furosemide should I give?
2. How do I support cardiac function?
3. What if they aren't getting better?
4. What if they've been in heart failure before?
5. Which feline arterial thromboembolism can do okay?
6. Why is there an arrhythmia?
7. Should I treat that arrhythmia?
8. How do I approach pericardial effusion?

INTRODUCTION

Cardiac emergencies can be stressful because they can present in extremis and also have the potential to deteriorate despite appropriate management. There are also a couple of pitfalls, especially with arrhythmia management, where we have the potential to actually make things worse rather than better. The goal of this section is to review the common cardiac conditions that present through the emergency room with a particular focus on congestive heart failure (CHF). Hopefully, at the end of this chapter, you'll have a firm framework in your head as to the initial management for these diseases as well as some fallback plans for when the initial approach isn't working.

INITIAL CHF MANAGEMENT

We talked extensively about the diagnosis of CHF in the chapter on dyspnea and so we won't repeat that discussion here. For the patient in CHF, there are roughly three simple treatment goals: alleviate congestion through diuretics, improve cardiac function, and manage hypoxemia with sedation/oxygen therapy. There is a potential fourth goal of arrhythmia management that we'll discuss later on as well.

Our first medication goal is to begin relieving/reducing cardiogenic pulmonary edema through the use of diuretics. Traditionally, this involves the use of either intravenous (IV) or intramuscular bolus doses of furosemide with transition to a continuous rate infusion if there is no persistent response (Keene et al. 2019). Interestingly, no difference in outcome was seen comparing bolus and CRI doses of furosemide (Filipejová et al. 2016, Ohad et al. 2018), though in healthy dogs the duration of diuresis was longer with a CRI (Harada et al. 2015). Torsemide can also be considered as a first-line agent in stable patients but has not been evaluated in patients requiring hospitalization (Besche et al. 2020). If any cardiogenic pleural (+/− peritoneal) effusion is identified, centesis will also help alleviate a reasonable degree of dyspnea.

The next medication goal for dogs is to support cardiac function through the use of an inodilator such as pimobendan, or rarely, dobutamine (Keene et al. 2019). While the use of dobutamine for patients is reasonable with signs of low output/forward flow heart failure (hypotension, cold extremities, poor perfusion parameters), it's rare to have that degree of systolic dysfunction (Goutal et al. 2010). While IV pimobendan is not commercially available in the

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United States, it can be given rectally with reasonable bioequivalence (Her et al. 2020), and a liquid version may soon become available that may improve administration as well (Kuhlmann and Markert 2025).

The use of pimobendan in cats is an interesting debate because, despite a fairly good safety profile, it was traditionally considered contraindicated in most non-dilated cardiomyopathy (DCM) cardiac diseases due to concerns about exacerbating any outflow tract obstructions (MacGregor et al. 2011). While some initial studies indicated safety and efficacy for hypertrophic cardiomyopathy (HCM) cats with and without obstructive pathology (Reina-Doreste et al. 2014, Ward et al. 2019), the largest trial showed no therapeutic benefit (Schober et al. 2021), and current consensus guidelines only provide a low level of evidence for its use in cats without left ventricular outflow obstruction (Luis Fuentes et al. 2020).

Our final goal is to maximize oxygenation through a combination of oxygen support and sedation to reduce the work of breathing as necessary, which was discussed in Chapter 15.

WHAT IF IT'S NOT WORKING?

CRITICAL QUESTIONS

1. Is there refractory pulmonary edema?
2. Are they hypoxemic?
3. Is there a left atrial rupture or ruptured chordae tendinea?

While the majority of patients will respond to the initial treatment, there is a percentage who will remain dyspneic +/− hypoxemic despite those therapies. The first question to consider is whether or not there is still evidence of pulmonary edema on lung ultrasound. If the answer is yes and you have maximized your diuretic dose, there is a reasonable chance they have a flail leaflet/ruptured chordae tendinea. Anecdotally, I have found these patients more difficult to control and their presence has been shown to be a negative prognostic indicator (Sargent et al. 2015) though some patients with certain physical exam and echocardiographic parameters can have prolonged survival (Serres et al. 2007). If there is no pulmonary edema and they are still dyspneic, you can consider concurrent pulmonary hypertension, especially if they have a known previous history of CHF (Borgarelli et al. 2015). Finally, if your patient in CHF abruptly deteriorates, you should also consider the possibility of a left atrial rupture, as shown in Figure 22.1.

Now, what are our additional treatment options for the pet with ongoing dyspnea or hypoxemia due to refractory cardiogenic pulmonary edema? From a procedural standpoint, as we discussed in the dyspnea chapter, high-flow nasal oxygen therapy can be considered for

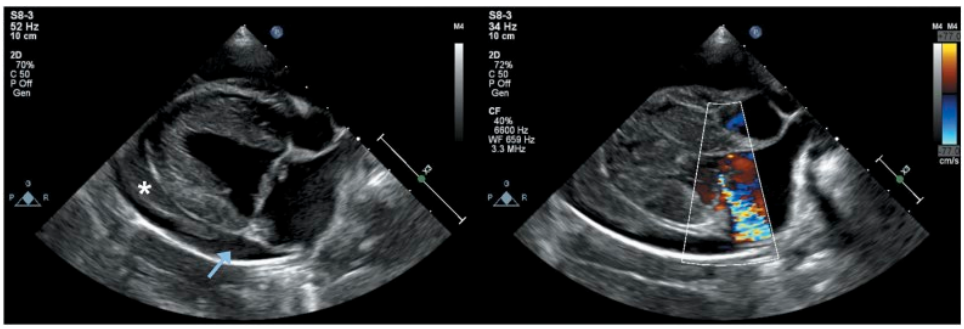


FIGURE 22.1 A left atrial rupture is a common cause for the peracute decline of an otherwise "stable" CHF patient. Intrapericardial blood clot (*), pericardial effusion secondary to left atrial rupture (→). *Source*: Czech et al. (2024)/Elsevier/CC BY 4.0.

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these patients (Whitney and Keir 2023, Adamik et al. 2025). Interestingly, the use of continuous positive airway pressure (CPAP) has also been described for these patients and led to decreased dyspnea and diuretic requirements (Prete et al. 2023). Finally, mechanical ventilation is an option with an acceptable success rate, though the expense and complication rate make it an infrequently used treatment modality (Edwards et al. 2014, Oppenheimer et al. 2022). From a medication standpoint for patients on maximum-dose diuretics with refractory pulmonary edema, an afterload reducer such as sodium nitroprusside or nitroglycerin can be considered (Greer et al. 2004, Achiel et al. 2020), though careful blood pressure monitoring is imperative. Empirically starting sildenafil for potential pulmonary hypertension in a patient with ongoing recent CHF is likely not something you should consider as an emergency clinician. Finally, for patients with a left atrial rupture, pericardiocentesis can be considered, though it is frequently unrewarding due to the presence of pericardial thrombus (Sugiura et al. 2022). Dobutamine can also be considered with or without pericardiocentesis (Isayama et al. 2022).

There is one additional treatment for feline CHF that we'll briefly talk about here (more in a later section) and that is starting anticoagulants/antiplatelet agents to prevent arterial thromboembolism (ATE). Interestingly, there is little direct evidence to support that these medications reduce the risk of a CHF cat developing an ATE (Rishniw 2024), and cats presenting with pleural effusion maybe at lower ATE risk (Busato et al. 2022). However, since the physiologic rationale remains sound with a reasonable risk profile, the use of clopidogrel is still recommended for cats in CHF (Luis Fuentes et al. 2020, Shaverdian and Li 2023).

CONCLUSIONS AND CLIENT COMMUNICATION

I've included my therapeutic flowchart for canine CHF in Figure 22.2, and for cats I would omit pimobendan and start clopidogrel.

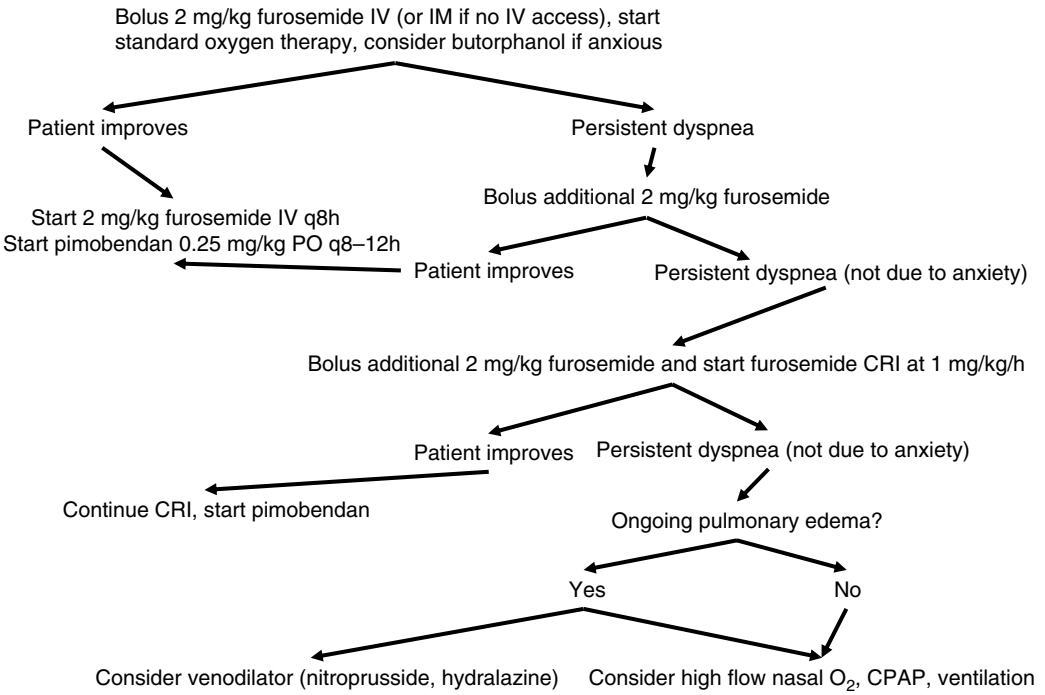


FIGURE 22.2 Basic flowchart for initial management of canine CHF. Courtesy of the author.

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The final part in this section is an outline of my initial client communication about treatment and prognosis for CHF:

- Your pet has evidence of CHF, which represents the end stage of a cardiac disease. In this condition, there is a fluid buildup in the lungs or chest that causes difficulty breathing and oxygenating.
- This is a condition managed through a combination of medications to support heart function and relieve the fluid buildup that they will have to go on for the rest of their lives.
- While these patients can do well, they benefit from frequent follow-up and intense monitoring.
- For dogs with degenerative mitral valve disease (DMVD) and CHF, the median survival time (MST) is roughly 12 months (Brložnik et al. 2023). Hypochloremia is likely a negative prognostic indicator (Roche-Catholy et al. 2021).
- For dogs with DCM and CHF, the MST is less than 6 months with a 34% 1-year survival rate. The presence of atrial fibrillation and right sided CHF are negative prognostic indicators (Friederich et al. 2020, Martin et al. 2010, Borgarelli et al. 2006).
- For cats with hypertrophic cardiomyopathy and CHF, the MST is 6–12 months with the presence of an ATE, larger left atrial size, pleural effusion, age, and right ventricular enlargement as negative prognostic indicators (Rush et al. 2002, Payne et al. 2010, Tantisuwat et al. 2018).

MANAGING THE CHRONIC CHF PATIENT

Patients presenting with signs of recurrent CHF are challenging because you have to weigh both the pros/cons of repeated hospital admission versus outpatient therapy and the risks/benefits of medication adjustments. The hospitalization recommendation should be made based off whether the pet requires oxygen (unless the owner has at-home oxygen) and the ability to receive oral medications along with a consideration for their goals of care.

The presence of either cardiogenic pulmonary edema or pleural effusion indicates that their current medication regimen is ineffective and, so you either have to adjust the diuretic dose/frequency (most common) or one of the other medications, such as pimobendan (Beaumier et al. 2018). We had discussed earlier the use of torsemide (as opposed to furosemide) as a first-line agent for CHF. It may be a reasonable option for pets with advanced heart failure as well, since its increased potency and half-life will decrease the dose required and increase the dosing interval. This may lead to improved owner compliance in patients with potential furosemide resistance (Oyama et al. 2011, Chetboul et al. 2017). Subcutaneous furosemide is also an option for these patients as they may have decreased oral absorption with advanced cardiac disease (Lombardo et al. 2025).

One of the big concerns with increasing diuretic doses is the onset of renal insufficiency. While uncommon, starting (or dose increases of) an ACE inhibitor (ACEi) in patients with CHF can lead to progressive renal dysfunction, especially in dogs with preexisting azotemia (Lee et al. 2025). Interestingly, in both dogs and cats, the majority of acute kidney injury/progression occurs in the hospital setting and has not necessarily been shown to impact survival (Giorgi et al. 2022, Rogg et al. 2025).

FELINE ARTERIAL THROMBOEMBOLISM (FATE)

CRITICAL QUESTIONS

1. Is it actually an ATE?
2. What is the likelihood of successful treatment?
3. What drugs should I start?

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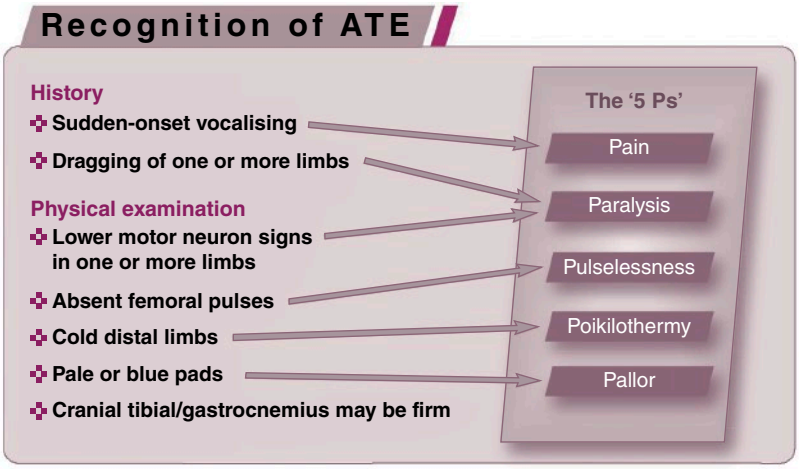


FIGURE 22.3 Prompt recognition of feline ATE is crucial to begin appropriate treatment. *Source*: Fuentes (2012)/Sage Publication.

Feline arterial thromboembolism or ATE is a reasonably common presenting diagnosis through the emergency service due to the acuity of its onset and visible signs of distress and pain it produces. Given its relatively high mortality rate, appropriate diagnosis and management of this condition are crucial for owner education and setting reasonable expectations.

The first question is to make sure the cat actually does have an ATE rather than an orthopedic or neurologic condition. While you should immediately suspect this condition in any cat with an acute onset of difficulty walking, other signs on physical exam consistent with an ATE are shown in Figure 22.3.

For patients with diagnostic equipoise, differential blood glucose and lactate levels can be helpful in clinching the diagnosis where a glucose difference of greater than 41 mg/dl and lactate difference greater than 2.2 mmol/l were more or less pathognomonic for ATE (Yee et al. 2024).

Your initial stabilization should involve analgesics and treatment for heart failure (if indicated by your physical and ultrasound exam) as roughly 40–50% of cats with ATE will have concurrent CHF (Schoeman 1999, Smith et al. 2003, Moses et al. 2024). After those are administered, you have to talk to the family about whether or not they wish to proceed with further treatment. Overall, the euthanasia and mortality rate for feline ATE is high with roughly 65–73% of cats succumbing within 7 days (Smith et al. 2003, Borgeat et al. 2014). However, I think it is important to provide owners with appropriate statistics and information regarding prognosis and outcome to help them make the best decision possible. The first point to address is the presence/absence of CHF. If owners are unwilling or unable to treat CHF, then managing the two diseases concurrently is futile. For owners who wish to treat their cat, they should be aware of the following negative prognostic indicators: concurrent CHF, temperature less than 96.2 °F, higher lactate in the affected limb, paraplegia, and bilateral pelvic limb involvement (Smith et al. 2003, Borgeat et al. 2014, Guillaumin et al. 2022, Guillaumin 2024). For owners who elect to treat, short-term survival is between 30–40% for bilateral ATE and for unilateral perhaps up to 50–60% (Borgeat et al. 2014).

I've included a summary of some of the FATE prognostic factors in Table 22.1.

Besides analgesia and treatment for CHF (if applicable), the mainstay of treatment for ATE is starting antithrombotic medications. Originally both aspirin and warfarin were used for this purpose (Smith et al. 2003) until clopidogrel was demonstrated to be a superior agent (Hogan et al. 2015). While there was initial enthusiasm around rivaroxaban (a direct oral anti-Xa inhibitor), the largest trial to date showed no significant difference when compared to clopidogrel

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Table 22.1 Summary prognostic factors for FATE.

Prognostic factors	Reported survival
Unilateral versus bilateral	42–68% (unilateral) versus 19–35 (bilateral)
Motor function present versus absent	65% (present) versus 27% (absent)
Normothermic versus hypothermic	45% versus 55%
CHF absent versus present	83% (absent) versus 26% (present)

Source: Adapted from Smith et al. (2003) and Borgeat et al. (2014).

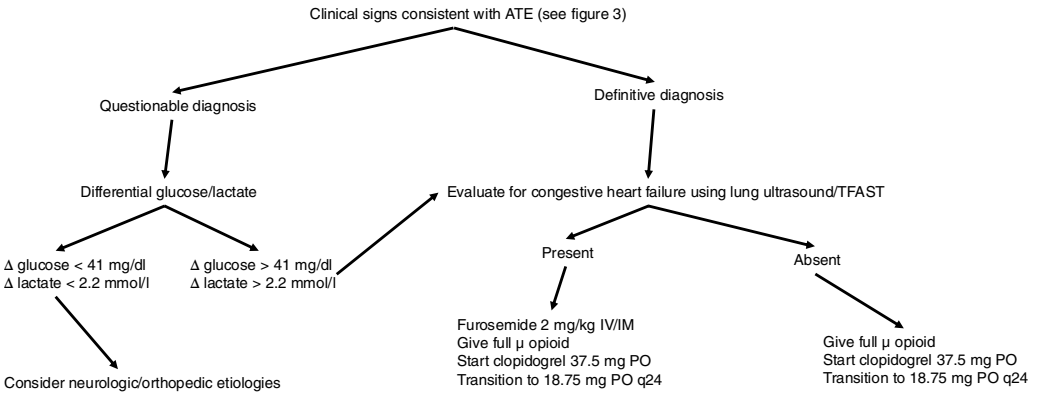


FIGURE 22.4 Initial diagnostic and therapeutic approach to feline ATE. Courtesy of the author.

(Brainard et al. 2024). Dual therapy with BOTH clopidogrel and rivaroxaban has been shown to be safe but has not been shown to be superior to either agent independently (Lo et al. 2022). The time to recurrence for clopidogrel was 443–663 days, depending on the study and was 513 days for rivaroxaban (Hogan et al. 2015, Brainard et al. 2024). There is no substantial evidence that the use of thrombolytics improves outcomes in feline ATE (Hannabuss 2019, Sharp et al. 2022, Guillaumin 2024).

CONCLUSIONS

I've put my own flowchart for initial diagnosis and treatment of ATE in Figure 22.4. I think when it comes to prognosis, IF a cat can survive the first 48 hours and show some form of improvement AND the owner is willing to manage the ATE (and CHF if present), these cats can have a reasonable long-term prognosis. However, cats exhibiting certain negative prognostic indicators (hypothermia, higher lactate, etc.) are much less likely to show improvement or survive that time period.

ARRHYTHMIA MANAGEMENT

CRITICAL QUESTIONS

1. What arrhythmia is present?
2. Is the origin of the arrhythmia primary cardiac or extra cardiac?
3. Would the patient benefit from treatment?
4. What medications are indicated?

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ARRHYTHMIA IDENTIFICATION

We'll cover identification of the most common arrhythmias seen in emergency practice through a stepwise approach with a primary goal of identifying rhythms that require treatment.

The first question to consider is whether or not the heart rate (either on ECG or auscultation) is too fast or too slow AND whether or not it is inappropriate for the patient's condition. For example, in a shocky patient, we would expect to be tachycardic, this is not a pathologic response. The second question is to identify whether or not there are p-waves present as this establishes whether or not you have a functioning sinoatrial (SA) node. Third, evaluate if the p-waves (if present) are all associated with a QRS complex, as this can help identify the function of the atrioventricular (AV) node. Finally, evaluate whether the QRS complex has normal (narrow) or abnormal (wide) morphology as this can help differentiate supraventricular versus ventricular origin. Summary flowcharts for brady and tachyarrhythmias are shown in Figure 22.5a,b.

Ventricular fibrillation does not necessarily fit this diagnostic pathway but is generally fair straightforward to recognize (Figure 22.6).

ARRHYTHMIA ETIOLOGY

The causes of arrhythmias can be categorized into three broad groups: structural cardiac disease, primary conduction abnormalities (with normal cardiac structure), and extra cardiac causes.

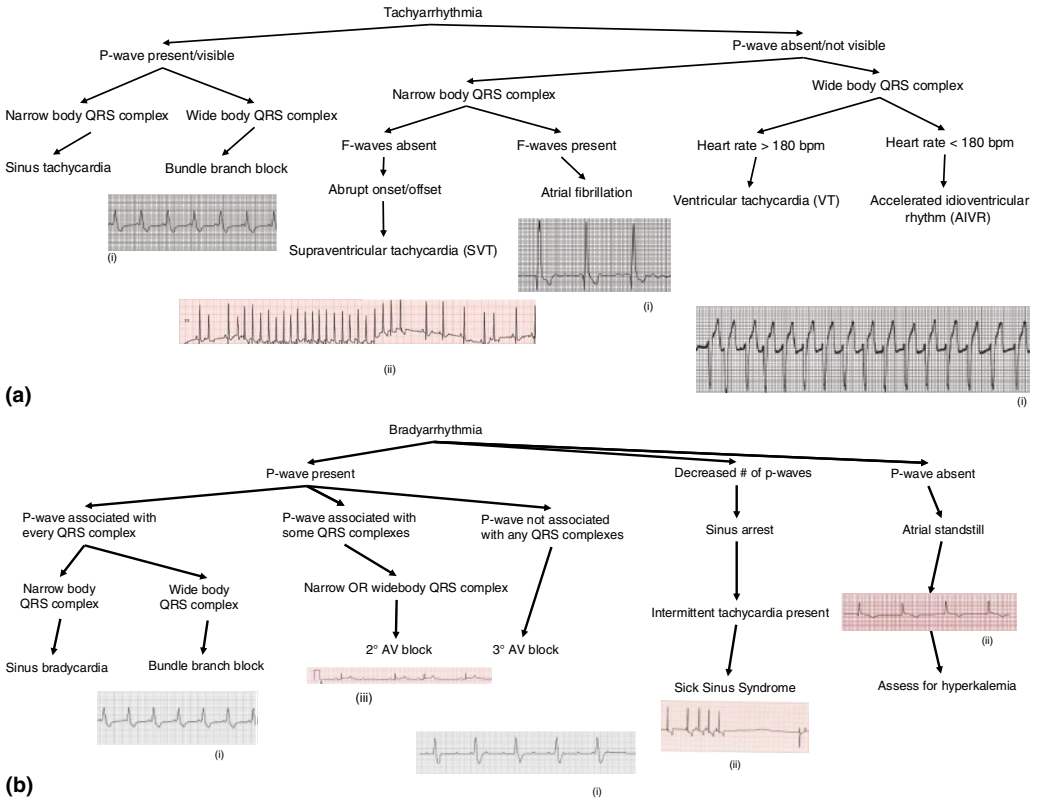


FIGURE 22.5 (a) Diagnostic approach to common tachyarrhythmias. ECG sources: (i) Mellema and Kohen (2012), used with permission. (ii) Oyama et al. (2013), used with permission. (b) Diagnostic approach to common bradyarrhythmias. ECG sources: (i) Mellema and Kohen (2012). (ii) Oyama et al. (2013). (iii) Goldberger et al. (2023). All used with permission.

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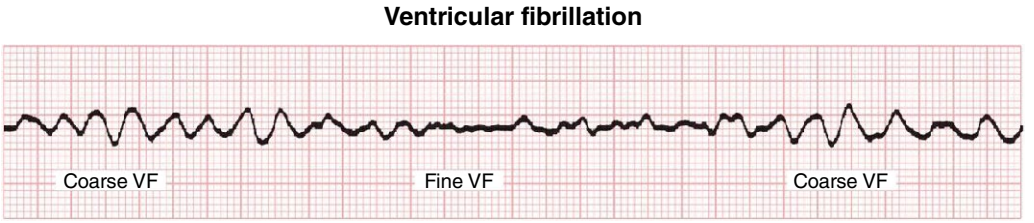


FIGURE 22.6 Ventricular fibrillation is a shockable arrest rhythm characterized by chaotic electrical activity. *Source*: From Goldberger et al. (2023). Used with permission under STM license.

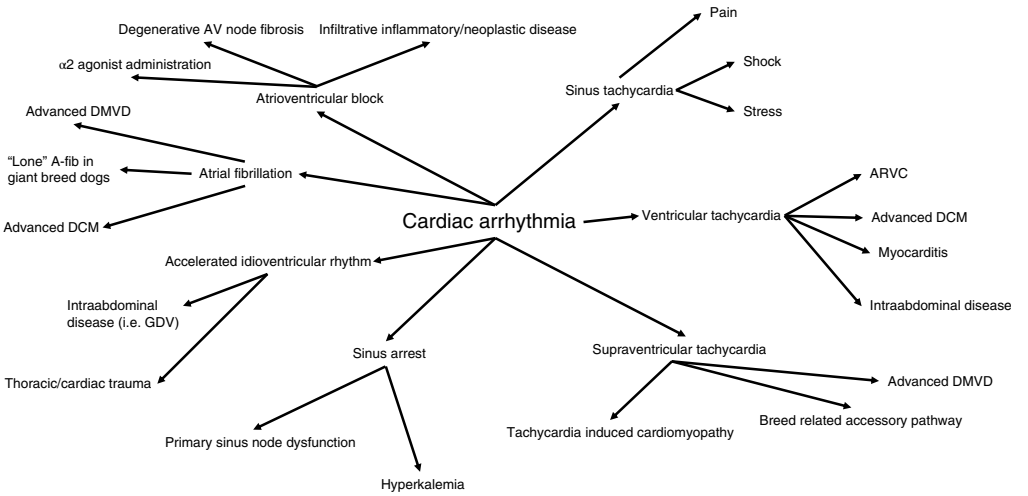


FIGURE 22.7 Common cardiac and systemic causes for select cardiac arrhythmia. Courtesy of the author.

The goal of a diagnostic workup (point of care labs, echocardiography, and potentially abdominal imaging) for these cases is to appropriately place a patient in the appropriate category to guide treatment. Figure 22.7 shows common diseases for each of these categories as well as the arrhythmias they are typically associated with.

With the exception of hyperkalemia-induced changes, feline cardiac arrhythmias are fairly uncommon and the vast majority of them do not require treatment (Szlosek et al. 2024). Those that *would* benefit from treatment are generally associated with more advanced cardiac disease (Côté 2010, Cofaru et al. 2024).

SHOULD I TREAT THE ARRHYTHMIA AND WITH WHAT?

Regardless of underlying etiology, the decisions to treat should involve an assessment of the likelihood of conversion to a fatal arrhythmia, the degree of cardiovascular compromise attributable to the arrhythmia, and concurrent diseases present. In this section, we'll go through the treatment options and decision-making for the two most common arrhythmias seen through the emergency room in dogs: atrial fibrillation and ventricular tachycardia.

As shown above, the most common causes for atrial fibrillation are advanced cardiac disease, usually either DMVD or DCM, due to the progressive left atrial enlargement seen with those conditions with the exception of "lone" atrial fibrillation breeds (Arcuri et al. 2024). As many dogs with

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atrial fibrillation will also have concurrent CHF and potentially right-sided cardiac disease (Ward et al. 2019), it is important to adequately evaluate and treat for those conditions as well. While we know that managing the heart rate below 125 bpm has a survival benefit in dogs with atrial fibrillation (Pedro et al. 2023), the exact timing of when to initiate therapy (i.e. before or after management of CHF) is not 100% clear (Gelzer et al. 2009, Pedro et al. 2020). Patients who are experiencing syncopal episodes might be more likely to benefit from earlier therapy as they are more likely to experience sudden cardiac death (Borgeat et al. 2021). Treatment options include diltiazem, digoxin, and amiodarone with the combination of diltiazem and digoxin potentially leading to the strongest rate control (Romito et al. 2025). For patients considered cardiovascularly stable and in CHF, it is likely reasonable to omit initial antiarrhythmic therapy for the first 24 hours or so. For patients either deemed unstable or not in CHF, consider oral diltiazem +/− digoxin with parenteral diltiazem as an option if they cannot take oral medications.

Ventricular arrhythmias can sometimes require a bit more of an aggressive approach because of their higher risk for conversion to a fatal rhythm (Santilli et al. 2021), even though bradyarrhythmias are more commonly associated with syncope (Perego et al. 2020). Traditionally, treatment has been recommended for ventricular arrhythmias with a rate higher than 150–180 bpm, sustained runs (>30 seconds), multiform complexes, or if R-on-T phenomenon is present (DeFrancesco 2013). However, clinical context is important when making a decision about whether or not to institute treatment because many patients with ventricular tachyarrhythmias will have systemic, not primary cardiac disease (Figure 22.7). For example, while ventricular arrhythmias in postoperative hemoabdomen patients are a negative prognostic indicator, the benefit and appropriate duration of therapy are unknown (Michael et al. 2023). In patients with ARVC, if they are symptomatic (i.e. syncopal or exercise intolerant) or have structural cardiac changes, then treatment is recommended (Cunningham and Dos Santos 2022). For patients with ventricular arrhythmias requiring emergent intervention, lidocaine is generally the first line agent followed by procainamide (Chandler et al. 2006) and amiodarone (Pedro et al. 2012, Levy et al. 2016) if that fails to convert the rhythm, as shown in Figure 22.8.

For patients requiring greater than two boluses of lidocaine, I will also consider a dose of magnesium (Schoeller et al. 2020). In dogs requiring ongoing oral antiarrhythmic therapy, sotalol (+/− mexiletine) is the first-line agent for patients without ventricular dysfunction or CHF where one might consider amiodarone as a first-line agent (Visser et al. 2018, Romito et al. 2024).

PERICARDIAL EFFUSION

CRITICAL QUESTIONS

1. What is the likelihood of a nonneoplastic etiology?
2. What are the risks of pericardiocentesis?
3. How do we reduce the risk of re-effusion?

INTRODUCTION

Similar to a hemoabdomen and ATE, pericardial effusion (PCE) is a common presenting complaint because of how abrupt its signs can appear. You should have a high clinical suspicion for PCE in any older dog presenting with collapse, weakness/lethargy, and in many cases, vomiting (Johnson et al. 2004, Fahey et al. 2017). In addition, if you ever see gallbladder wall edema on POCUS, always make sure to evaluate the heart to ensure the patient doesn't have PCE (Lisciandro et al. 2021). The goal of

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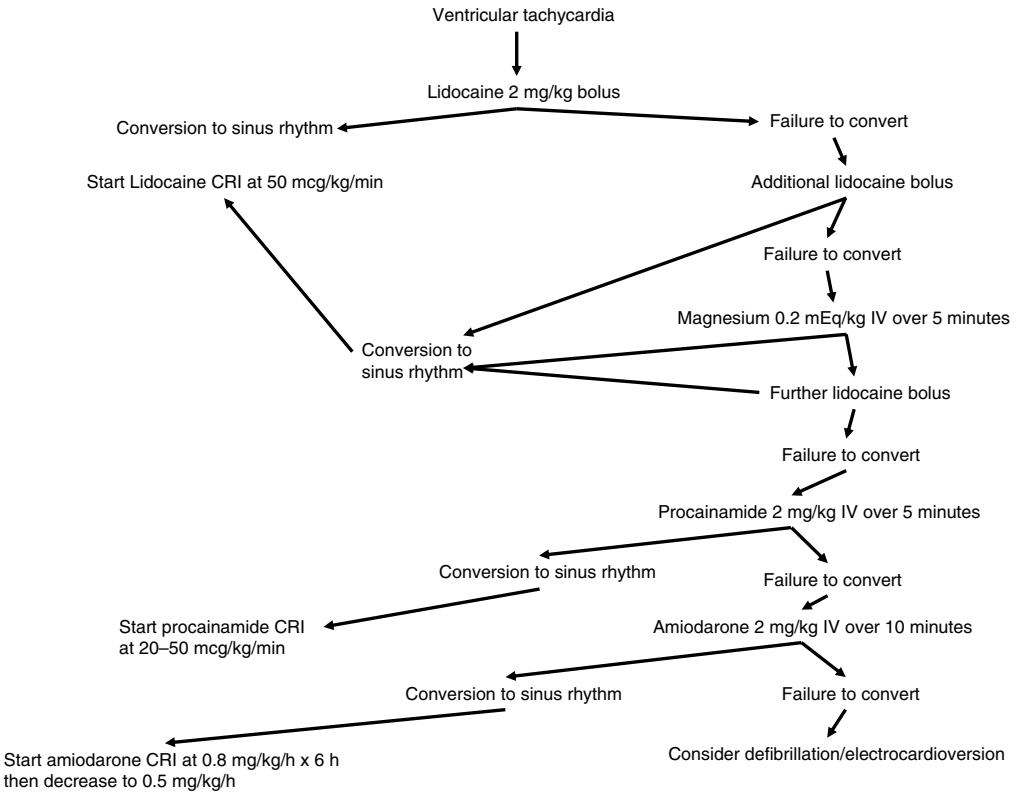


FIGURE 22.8 Author's initial parenteral management for canine ventricular tachycardia. Courtesy of the author.

this section is to review the diagnostic workup for PCE as well as the prognosis associated with some common underlying diseases. While we won't cover the procedure of pericardiocentesis itself, we will discuss some of the common complications (and how to fix them) so you can provide informed consent to the family prior to performing the procedure. As 75% of cats with PCE are in CHF (Hall et al. 2007), which we have previously covered and don't typically require pericardiocentesis and so we won't go into much greater detail.

DIAGNOSTIC WORKUP FOR PCE

At its core, the goal of the diagnostic workup in dogs is to differentiate between cardiac mass(es), disease of the pericardium itself (neoplastic, inflammatory, or infectious), and idiopathic PCE (Linney 2023). While left atrial rupture in dogs is also a cause for PCE, we covered that topic earlier in this chapter. Given the vastly different treatments and prognosis between several of these diseases, obtaining a diagnosis can be helpful in providing an owner with the most accurate picture of their dog's disease.

The first question to answer is whether or not there is a cardiac mass present, usually through the use of an echocardiogram. While echocardiography is extremely specific for the presence of a cardiac mass, a "negative" echo in a patient with a high suspicion of neoplasia should be taken with a grain of salt (Macdonald et al. 2009). In the cited study, roughly 62% of dogs presenting with PCE had a cardiac mass with most right atrial masses representing hemangiosarcoma. Interestingly, while traditionally most heart base tumors were thought to be chemodectomas (and that was the

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plurality here), there was also a diverse array of other neoplasia present as well. The use of CT or MRI has not been shown to improve diagnostic accuracy compared to echocardiography (Boddy et al. 2011, Scollan et al. 2015).

For patients without a visible cardiac mass, the next diagnostic goal is differentiating infectious, inflammatory, idiopathic, and neoplastic diseases of the pericardium. Ideally, cytology would be the diagnostic test of choice, but it is only diagnostic in a minority of cases. Its diagnostic yield can be increased if the effusion PCV is less than 10% (Cagle et al. 2014), and in the future, flow cytometry may be able to help differentiate neoplastic versus nonneoplastic effusions (Sini et al. 2024). Unfortunately, in the absence of other contextual clues such as signalment, travel history, and concurrent diseases, a definitive diagnosis for the cause of PCE with no visible mass can be challenging for the emergency clinician. Given the overlap in patient signalment between patients with neoplastic and nonneoplastic effusion, it can be difficult to provide a prognosis for dogs with no visible mass as many of those dogs will re-effuse (Johnson et al. 2004). While surgery can lead to prolonged survival times in dogs with idiopathic PCE (Scheuermann et al. 2021), it is important for owners to realize that preoperative differentiation of idiopathic PCE and mesothelioma is challenging (Stepien et al. 2000).

PERICARDIOCENTESIS

While the procedure of pericardiocentesis is well described elsewhere (Daly 2012), I do think it is worthwhile to go through some common complications both for your own mental preparation and to discuss with the owner. The most common adverse event seen tends to be (ventricular) arrhythmias (Humm et al. 2009), and while you can give lidocaine to convert the rhythm, I have also found that simply retracting the catheter slightly away from the heart is also helpful. And while the short-term mortality of this procedure is low (I think less than 25% as cited in Humm et al. 2009), I do always warn owners about that risk, either due to rapid fluid re-accumulation or cardiac arrhythmia. While the placement of pericardial catheters has been described both for single and serial drainage, their superiority to needle pericardiocentesis has not been fully demonstrated (Cook et al. 2019, Cook et al. 2021). As with any "high stress" procedure, it is always important to have a checklist, as shown in Table 22.2, and a mental image of the procedure in your mind.

Table 22.2 Pericardiocentesis checklist.

Sedation: butorphanol and consider local lidocaine block
Pericardiocentesis catheter: I will usually use an 18 gauge long IV catheter
Drainage set
Antiarrhythmic dose of lidocaine
ECG monitoring

Post procedure your patient's vital parameters and shock indicators should improve and stabilize (Moumadah et al. 2025). For patients without left atrial rupture, I will usually give a small fluid bolus as well. If the patient fails to stabilize or regresses, you should reassess for recurrent PCE.

LONGER TERM MANAGEMENT OPTIONS AND PROGNOSIS

The following is more for discussion with an owner because in all likelihood, you will not be responsible for initiating the majority of these treatments. For dogs with suspected right atrial hemangiosarcoma, chemotherapy with doxorubicin can lead to MST of roughly 3–5 months (Ghaffari et al. 2014, Mullin et al. 2016) and surgery (if amenable) did not seem to improve that MST (Weiss et al. 2005). For dogs managed without chemotherapy, the MST is generally less than 1 month (Johnson et al. 2004, Weisse et al. 2005, MacDonald et al. 2009).

For patients with heart base tumors (usually chemodectomas), pericardiectomy alone can yield an average survival time of roughly 18–24 months (Vicari et al. 2001, Ehrhart et al. 2002). While use of toceranib alone has been postulated to yield similar results (Coto et al. 2021), less than 50% of the dogs in that study had PCE make generalizability difficult.

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Prolonged survival times can be seen with pericardiectomy (and repeated pericardiocentesis) for idiopathic PCE (Scheuermann et al. 2021). However, the difficulty in preoperatively differentiating that disease from mesothelioma should be a conversation with the owner as MST for that cancer managed surgically is roughly 9–10 months (Case et al. 2013).

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Reproductive Emergencies

CHAPTER 23

CRITICAL QUESTIONS

1. Is it actually a dystocia?
2. Does it need a cesarean section?
3. What causes postpartum discharge/illness?
4. What drugs can I use peripartum?
5. When does the pyometra patient need surgery?

INTRODUCTION

Since the incidence of reproductive emergencies is relatively low as the majority of patients are either spayed or neutered, many clinicians are uncomfortable managing these types of cases. The goal of this chapter is to review decision-making around some of the more common cases that will present through the emergency room.

DEFINING A DYSTOCIA

Understanding risk factors for dystocia allows a prompter patient identification and phone triage for higher risk patients. In dogs, these include smaller litter size, small/toy breed dogs, older age of the dog, and brachycephalic breeds (Münnich and Küchenmeister 2009, Cornelius et al. 2019, O'Neill et al. 2019).

Similarly, risk factors for dystocia in cats include singleton pregnancy, certain breeds (British Shorthair, Oriental breeds, Birman, Ragdoll, Abyssinian), and obese queens (Holst 2022, Sendag et al. 2025).

For any potential dystocia, the first challenge is actually identifying whether or not the patient even HAS an emergency! The stages of canine and feline labor are displayed in Table 23.1, which forms the basis of your history questions to determine which stage of labor the patient is actually in.

Table 23.1 Standard stages of labor and duration for canine and feline patients.

Clinical stage	Clinical signs	Expected duration (canine)	Expected duration (feline)
Pregnancy/ prepartum	None, progressive abdominal distention	Average 65 days gestation	Average 65 days gestation
Stage I of labor	Restlessness, panting, nesting	Usually 6–12 hours, can be up to 24 hours	Typically less than 2 hours
Stage II of labor	Clear vaginal discharge, visible abdominal contractions, puppy birth	First puppy after 2–4 hours, total duration 12–24 hours	First kitten within 1–2 hours, usually all kittens within 6 hours (every 30 minutes), though maybe prolonged up to 48 hours
Stage III of labor	Passing of placenta	Should occur within 15 minutes of puppy birth	Usually simultaneous to kitten birth

Source: Adapted from Gendler et al. (2007) and Holst (2022).

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Based on those answers and further historical questions, you can determine whether or not the patient is actually experiencing true dystocia if they meet the criteria listed as follows:

Canine

- Persistent abdominal contractions for >30 minutes without delivery
- Intermittent straining for >4 hours without delivery
- Green vaginal discharge
- Greater than 2 hours between delivery of puppies
- Foul smelling or hemorrhagic vaginal discharge
- Visible puppy in birth canal

Feline

- Intermittent straining for >2 hours without delivery of a kitten
- Hemorrhagic vaginal discharge
- Persistent abdominal straining for greater than 5 minutes or kitten stuck >5 minutes
- Abnormal vaginal discharge (hemorrhage, purulent)
- Greater than 90 minutes between kittens (5% can go longer)

Finally, you have to try and diagnose the etiology or source of dystocia as that will also help guide the decision whether or not to pursue surgical management. Common causes of dystocia include fetal malposition, feto-maternal mismatch, and either primary or secondary uterine inertia (Münnich and Küchenmeister 2009, Bailin et al. 2022).

DIAGNOSTIC WORKUP FOR DYSTOCIA

Your initial workup for a dystocia patient should include an evaluation of fetal health via ultrasound (to obtain a fetal heart rate), an assessment of fetal size, and an evaluation of maternal health.

Point of care ultrasound is critical because it allows assessment of the fetal heart rate (Fulton 2021), which correlates with the degree of fetal distress. Values for normal and distressed fetal heart rates are shown in Table 23.2.

Radiographs can be helpful in determining the number of fetuses present as well as the likelihood of an obstructive type dystocia not amenable to medical management, as shown in Figure 23.1.

Finally, an assessment of maternal health using lab work (especially an ionized calcium level) may be helpful in determining correctable factors to increase the chances of success for medical management (Frehner et al. 2018). Furthermore, if surgical management is indicated, evaluation of lab work will also help guide preoperative stabilization and anesthetic optimization.

MEDICAL VERSUS SURGICAL DECISION-MAKING

The decision to pursue medical versus surgical management is multifactorial, encompassing medical, patient, and owner factors. Disease factors, such as the presence or absence of fetal obstruction may make medical management contraindicated while owner factors, such as finances and

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Table 23.2 Fetal heart rate ranges for both canine and feline patients.

Fetal heart rate (canine)	Fetal heart rate (feline)	Degree of fetal distress
<150 beats per minute	<160 beats per minute	Critical
150–180 beats per minute	<180 beats per minute	Moderate to severe
>180 beats per minute	>200 beats per minute	Normal

Source: Adapted from Pretzer (2008) and Smith (2012).

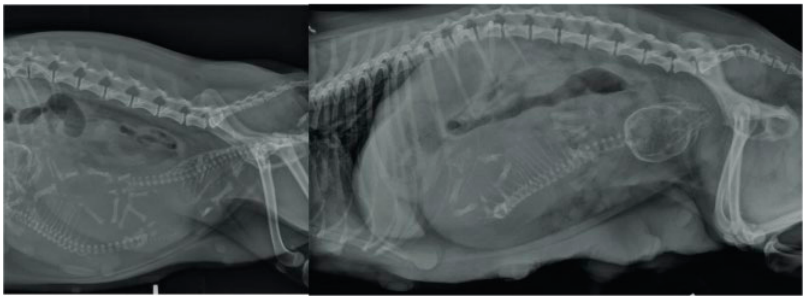


FIGURE 23.1 Radiographic diagnosis of obstructive dystocia (fetal malposition shown here) is crucial to maximizing the success and minimizing the risk of medical management for dystocia. iCa, ionized calcium. *Source*: Runcan (2019) with permission of John Wiley & Sons.

prioritization of maternal/fetal health may drive earlier (or delayed) surgical intervention. While we'll focus mostly on the medical factors here, it is important to communicate with the owner about their goals and priorities as it may trigger a decision to pursue cesarean section earlier to maximize the chances of fetal survival.

Classifying patients with dystocia can be helpful in terms of risk stratification for the chances of success with medical management. Emergent surgical management should be recommended for any patient with obstructive dystocia or any signs of fetal distress based on fetal heart rate (Traas 2008b, Smith 2012). While medical management can be considered for patients in early dystocia with no evidence of fetal distress and a small litter size (Pretzer 2008), prolonged stage II labor is associated with increased fetal mortality (Darvelid and Linde-Forsberg 1994, Münnich and Küchenmeister 2009).

NONSURGICAL MANAGEMENT FOR DYSTOCIA

Initial medical management for dystocia involves oxytocin and intravenous calcium (Runcan and Coutinho da Silva 2018) with the dose of oxytocin dependent on the size of the dogs. The use of calcium in cats is potentially problematic, and so in the rare feline dystocia case, the author typically uses oxytocin only (Smith 2012). Owners should understand that the success rate for medical management is roughly 50% and is also breed dependent, where brachycephalic breeds are more likely to fail (O'Neil et al. 2019, Münnich and Küchenmeister 2009). Success rates for medical management in cats are even lower at around 17–30% (Bailin et al. 2022, Sendag et al. 2025). Criteria for failure of medical management are generally considered lack of fetal production or evidence of progressive fetal distress after 2–3 doses of oxytocin at 30-minute intervals. (Pretzer 2008, Smith 2012, Runcan and Coutinho da Silva 2018). I've included my own approach to dystocia medical management in Figure 23.2.

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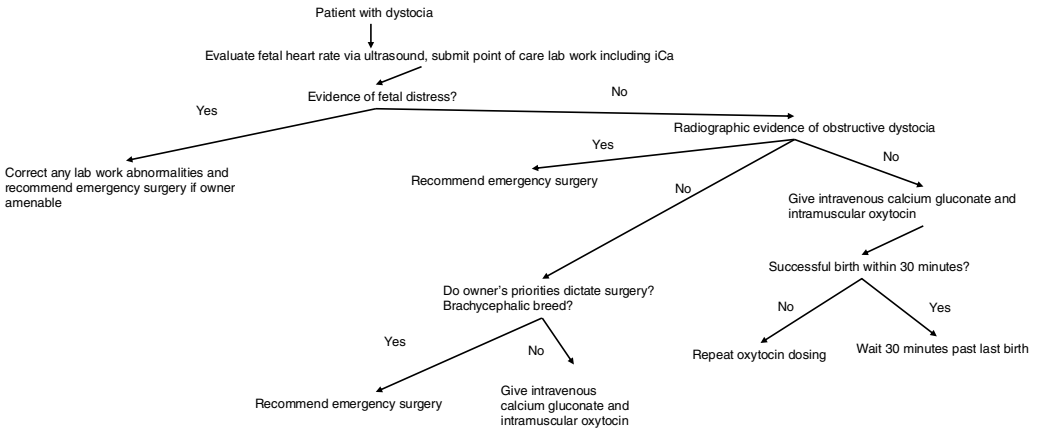


FIGURE 23.2 The author's diagnostic and therapeutic approach to dystocia. Courtesy of the author.

NEONATAL RESUSCITATION

Regardless of the treatment approach undertaken, fetal resuscitation is a key part of dystocia management because the trauma of prolonged birth can compromise an already fragile neonatal physiology. The key points are tactile stimulation, warmth, provision of oxygen, and clearing the airway with a summary flowchart for interventions shown in Figure 23.3.

POSTPARTUM ILLNESS

The most common postpartum presenting complaints are likely hypocalcemia, endometritis, and mastitis (Orfanou et al. 2010). For patients with eclampsia, it is important to be able to differentiate seizure activity from muscle fasciculations. In the latter scenario, the patient is conscious, whereas in the former, they are not. Eclampsia will typically occur within 4 weeks of parturition and is most common in small-breed dogs with larger litter sizes (Drobatz and Casey 2000). Initial management involves a slow intravenous infusion of calcium gluconate (I will usually dilute it 1:3 and give over 15 minutes) with concurrent ECG monitoring, followed by oral calcium supplementation (Davidson 2012). Early full or partial weaning is also recommended to prevent a recurrence of clinical signs (Davidson and Cain 2023).

Postpartum vaginal discharge combined with systemic illness and fever should raise concern for metritis, which typically occurs within 7 days of whelping (Lection et al. 2021). Always consider imaging to evaluate for the presence of a retained fetus that may require surgical management (Lopate 2012). Initial stabilization typically involves fluid therapy and generally an antibiotic such as potentiated penicillin, though in refractory cases, ovariohysterectomy can be considered for source control (Gonzales 2018).

While mastitis is uncommon, it does have the potential to cause systemic illness and may require surgical debridement (Gonzales 2018). While milder cases may respond to oral antibiotics (usually either cephalosporins or potentiated penicillins), more severe cases may require hospitalization for intravenous fluid and antibiotic therapy (Wiebe and Howard 2009). Analgesia through short-term NSAIDs +/− opioids is likely safe (Ferrari et al. 2022) for lactating animals.

PERIPARTUM PHARMACOLOGY

While current drug formularies (i.e. Plumbs') do contain a section on reproductive safety, the majority of the entries are unfortunately lacking in data and recommend weighing the risks

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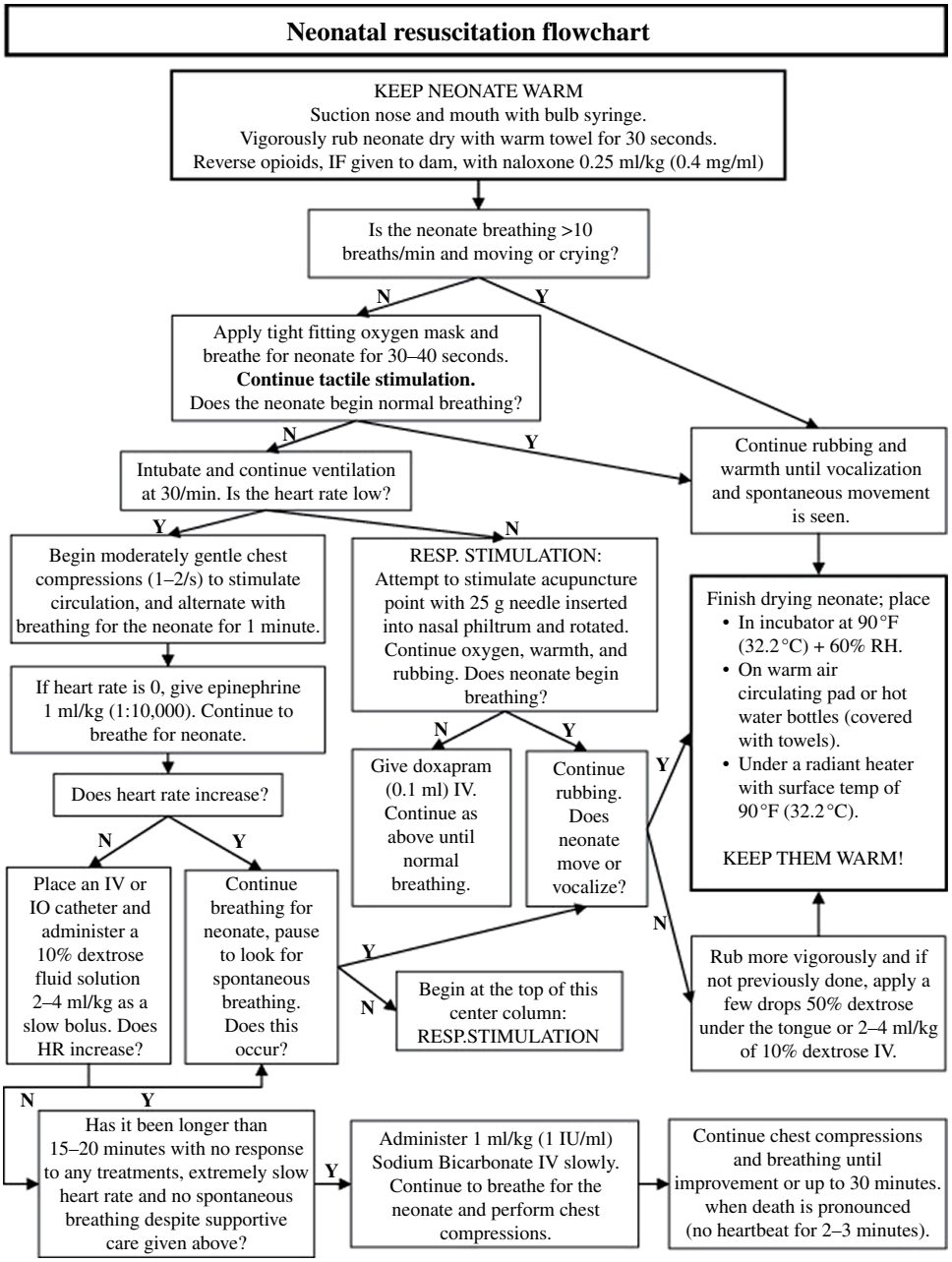


FIGURE 23.3 Initial neonatal stabilization flowchart. *Source*: Traas, 2008a with permission of Elsevier. I will typically NOT empirically give naloxone and have found intubation challenging.

and benefits for the individual patient. The most common drugs we have to consider are antibiotics and pain medications/sedation, though a more complete list is in the Wiebe and Howard (2009) article as well as a recent publication in the anesthesia literature (Navarro-Altuna et al. 2025). Some of the more commonly used antibiotics and sedation medications are shown in Table 23.3.

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Table 23.3 Common antibiotics and sedatives/analgesics with risk profile. Furthermore, you should generally avoid benzodiazepines (can cause profound sedation) and medium to high dose acepromazine (due to hypotension).

Medication	Risk level	Comment
Amoxicillin +/– clavulanic acid	Low	Okay for pregnant/lactating animals
Cephalosporins	Low	Okay for pregnant/lactating animals
Doxycycline	High	Avoid
NSAIDs	Medium to high	Single dose only
Opioids	Medium	Low doses, mostly examined in surgical patients
Dexmedetomidine	Low to medium	Minimal placental transfer (Groppetti et al. 2019)
Ketamine	High	Worsened neonatal survival at C-section
Propofol	Low to medium	Minimal placental transfer (Groppetti et al. 2019)

Source: Adapted from Wiebe and Howard (2009) and Navarro-Altuna et al. (2025).

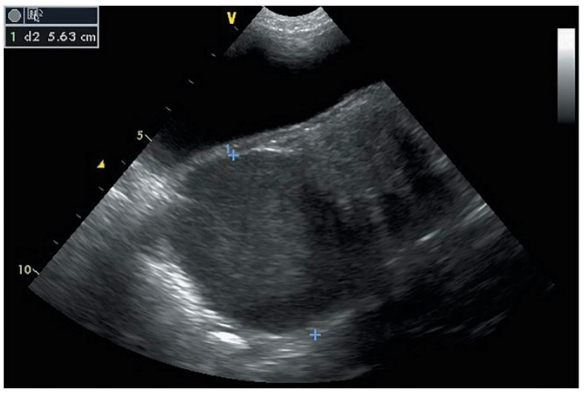


FIGURE 23.4 Ultrasonographic image of a (stump) pyometraillustrating the "multiple bladder" sign typically seen with this disease. *Source*: Hagman (2023) with permission of Elsevier.

AN APPROACH TO PYOMETRA

While typically pyometra will occur 1–4 months post estrus in dogs (Smith 2006), you should consider it as a differential diagnosis in any sick, intact female patient, especially those with vaginal discharge. Point of care ultrasound is very helpful in diagnosing this condition through visualization of multiple distended uterine loops (as shown in Figure 23.4), even in patients without vaginal discharge (Fulton 2021).

Initial stabilization involves fluid therapy and antibiotics, as we've discussed in previous chapters. Antibiotic selection should be targeted against *Escherichia coli* as that is the most common organism isolated (Ylhäinen et al. 2025), though interestingly, the actual agent used has not been shown to impact outcome, and some clinicians will forgo postoperative antibiotics (Turkki et al. 2023, Bertero et al. 2024). My personal practice is ampicillin/sulbactam with transition to oral amoxicillin-clavulanate for 7 days postoperatively. There is an ongoing clinical trial (Ylhäinen et al. 2023) examining whether or not *any* postoperative antibiotic therapy is beneficial in pyometra patients. For further discussion on risk factors for multidrug-resistant infections, see Chapter 6.

The question of surgical timing is always a challenging one. Delays of over 24 hours have not necessarily been shown to worsen outcome (McCobb et al. 2022), and indeed, anecdotally I have seen dogs go longer than that with antibiotics alone. Patients that would benefit from emergency surgery

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would be those with leukopenia, azotemia, peritoneal effusion (indicative of potential rupture/peritonitis), hypothermia, and evidence of sepsis (shock, hyperlactatemia) as that population is already severely compromised from their disease. (Jitpean et al. 2014, Donati et al. 2022, Pailler et al. 2022a). Provided they survive the perioperative period, the long-term prognosis in these patients is excellent (Pailler et al. 2022b).

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Cardiopulmonary-cerebral Resuscitation

CHAPTER 24

CRITICAL QUESTIONS

1. Is there a reversible cause for this arrest?
2. Is there a shockable rhythm?
3. What caused the traumatic arrest?
4. When should we terminate resuscitative efforts?
5. What happens if we're successful?
6. What is the prognosis for survival to discharge with CPCR?

INTRODUCTION

The goal of this chapter is not to provide a step-by-step description of the procedure and medications for basic and advanced life support. The RECOVER guidelines were recently updated (Hopper et al. 2024, Wolf et al. 2024, Brainard et al. 2024, Burkitt-Creedon et al. 2024), and they provide a far superior review on the provision of cardiopulmonary-cerebral resuscitation (CPCR) than I ever could. Instead, I hope to help you think critically about the causes of cardiac arrest and what you can do BESIDES chest compressions (among other things) that may benefit your patient. We'll also talk about what happens when you successfully achieve return of spontaneous circulation (ROSC) and what that means in terms of treatment and prognosis.

PRE-ARREST FACTORS

Some of the most important aspects of cardiac arrest management occur BEFORE an arrest even happens. The first is the discussion with the owner around resuscitation code status. While ideally this conversation is had before any event occurs, veterinarians infrequently discuss both cost and survival statistics associated with CPCR (Gane et al. 2024). Unfortunately, at most emergency hospitals (including my own), a resuscitation decision is made at admission and then rarely revisited once a diagnosis or treatment plan is made. Furthermore, in certain cases, such as sedation-related arrests, surveys have shown many veterinarians will occasionally override a do not resuscitate order in cases where the cause for arrest is deemed easily reversible (Cabral et al. 2019). I think these studies highlight the importance of a collaborative discussion with an owner BEFORE a crisis occurs and reevaluating their decision-making as further information becomes available.

Secondly, the role of preparation for CPCR cannot be overstated. Beyond knowledge of and adherence to the RECOVER guidelines, appropriate staff training, a well-stocked crash cart, and adequate qualified personnel are important pre-arrest determinants of success (McMichael et al. 2012, Hoehne et al. 2025).

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MEDICAL CAUSES FOR CARDIAC ARREST

The following list provides some of the common overall causes for cardiac arrest with the most common etiologies being hypotension (i.e. shock) and respiratory disease/hypoxemia (Hoehne et al. 2023):

1. Cardiovascular
   - **a.** Heart disease including arrhythmia, myocardial infarction, myocarditis, cardiac chamber thrombus, acquired, or congenital structural heart disease.
   - **b.** Blood disorders including anemia, hypovolemia, hemorrhage.
   - **c.** Vascular disorders including clinical signs of poor perfusion suspected to be secondary to septic shock and severe persistent hypotension despite adequate fluid resuscitation.
   - **d.** Pericardial effusion.
2. Respiratory
   - **a.** Pulmonary parenchymal disease including pneumonia, noncardiogenic pulmonary edema, cardiogenic pulmonary edema, lung lobe torsion, pulmonary hemorrhage, pulmonary hypertension.
   - **b.** Upper airway obstruction.
3. Neurologic including intracranial disease such as hemorrhage, meningitis, seizures, or tumor, cervical myelopathy, and neuromuscular disease such as tetanus.
4. Vagal-tone related suspected based on knowledge of underlying disease process, activity during and preceding arrest, and/or bradycardic arrest.
5. Anesthesia-related
   - **a.** Simple including patients undergoing an elective procedure or those that experienced an anesthetic overdose.
   - **b.** Complex cases in which the animal has a severe underlying disease and was anesthetized or recently extubated at the time of cardiopulmonary arrest (CPA).
6. Electrolyte/metabolic disturbances including hyperkalemia, hyper/hypoglycemia, ketoacidosis, uremia.
7. Severe systemic disease including acute necrotizing pancreatitis, kidney disease, disseminated neoplasia, sepsis, multiorgan dysfunction syndrome (MODS).
8. Multiple trauma including animals that are hit by car or suffer bite wounds to multiple body parts/cavities.
9. Multiple causes that include one or more of any of the above categories.
10. Cause of arrest is unknown.

This list is important because having it in your head can help you focus on certain aspects of the signalment and history to guide a fast physical and ultrasound exam that may expand your treatment options beyond chest compressions and drugs. For example, evaluating the bladder of the arrested young male cat for urethral obstruction may allow you to more rapidly manage his hyperkalemia via catheterization or cystocentesis. Identifying a *reversible* cause for arrest not only allows you a better chance for a successful resuscitation but also a better overall prognosis (Waldrop et al. 2004). Besides anesthetic/sedation induced cardiac arrest, which tends to be more reversible and survivable (Hofmeister et al. 2009), other *potentially* reversible medical causes (adapted from human medicine) for cardiac arrest would be hypoglycemia, upper airway obstruction, hyperkalemia, spontaneous pneumothorax, and pericardial effusion (Dewolf et al. 2022).

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One other classic (though uncommon) reversible cause for cardiac arrest is a shockable arrhythmia, such as pulseless ventricular tachycardia and ventricular fibrillation. Shockable rhythms can either occur as the initial presenting rhythm or develop through the course of CPCR, which is why it is important to have intermittent ECG/rhythm checks. While the presence of a shockable rhythm provides you with another treatment avenue (defibrillation), it is questionably associated with a higher likelihood of ROSC and survival to discharge rates remain similar to those without a shockable rhythm (Hoehne et al. 2019b, Vega Suarez et al. 2023).

EVALUATING THE TRAUMATIC ARREST

As we saw above, not all cardiac arrests are the same, and you should always be on the lookout for a potential reversible cause. In cases where the source of the arrest is trauma, potentially reversible factors include upper airway obstruction/hypoxia, tension pneumothorax, pericardial effusion/tamponade, and hemorrhage (Smith et al. 2015). While intubation is generally sufficient to relieve most upper airway obstructions and provide oxygenation, the other listed causes require different procedural interventions. A tension pneumothorax can be relieved using the "finger thoracostomy" technique described in Chapter 10 (see Figure 10.20). For patients with cardiac tamponade and/or intrathoracic hemorrhage then an emergent thoracotomy is indicated. While there are several sources describing the procedure itself (Wierenga 2012) and several experimental and laboratory studies showing it is more effective than closed chest resuscitation, there are multiple logistical challenges associated with this approach, especially if ROSC is obtained (i.e. need to manage a post thoracotomy AND post cardiac arrest patient). Penetrating trauma (i.e. single injury) patients, where this would be most effective are uncommon, and blunt trauma patients (more common) typically have polytrauma where the additional surgical injury from a thoracotomy is insurmountable. In my opinion, there is little clinical evidence to support the superiority of open chest CPCR to justify the trauma and cost associated with the procedure. In human medicine, survival rates for blunt trauma managed through open chest CPCR are incredibly low, and it is not recommended in the majority of cases (Endo et al. 2017, Tesoriero et al. 2024). Similarly, while the concept of damage control surgery for abdominal hemorrhage has been discussed for trauma patients (Devey 2013, Peterson et al. 2015), it has only been demonstrated in canine patients with non-traumatic hemoabdomen secondary to hepatic neoplasia (Evans et al. 2019).

PROGNOSTIC FACTORS ASSOCIATED WITH ACHIEVING ROSC

One challenging decision is when to stop resuscitative efforts. Frequently, owners will elect to stop efforts when informed about the grave short- and long-term prognosis, but many owners would like the veterinarian to inform them when it is medically unreasonable to continue (Bertram et al. 2024). I've included some of the prognostic factors associated with achieving ROSC in Table 24.1, acknowledging that the range for duration of cardiopulmonary resuscitation (CPR) with patient survival can be quite lengthy (i.e. there are even some, though few, survivors of prolonged CPR).

A small study also showed that higher initial $EtCO_2$ and a value greater than 18 mmHg during CPCR was helpful in differentiating patients likely to achieve ROSC (Hogen et al. 2018). My own approach is to strongly recommend cessation of resuscitation after roughly 12–15 minutes of arrest time without ROSC, except for patients experiencing sedation/anesthesia related arrests.

POST ARREST CARE

Probably the most challenging part of CPCR actually occurs AFTER successful resuscitation, and many clinicians understandably feel uncomfortable with this aspect of care. Current recommendations for post-arrest care were not updated in the most recent edition of RECOVER, though the

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Table 24.1 Factors associated with increased chance of ROSC for select studies published after the implementation of the RECOVER guidelines.

Study	Percentage of ROSC	Prognostic factors	Comments
Hoehne et al. (2023)	60% achieved initial ROSC, 40% achieved sustained ROSC ≥ 20 minutes	Positive: feline species, higher PvO_2, higher base excess	Sustained ROSC was more likely in cats, higher PvO_2, low/normal potassium level, higher base excess
Dazio et al. (2023)	27% ROSC rate	Surgical patients, anesthetic arrests, witnessed arrest, arrhythmia induced, toxin induced	Multivariate analysis showed increased ROSC with surgical/anesthesia arrests and arrhythmias
Vega Suarez et al. (2023)	27% initial ROSC rate, 13% sustained ROSC	No difference seen between shockable/nonshockable rhythms	—
McIntyre et al. (2014)	58% initial ROSC, 32% sustained ROSC	Positive: IV inplace, palpable pulses. Negative: delay to initiation of CPR, longer duration of CPR, neurologic cause of arrest	—
Kawase et al. (2018)	31% ROSC rate	Positive: inhospital arrest, shorter duration of CPR	—
Hoehne et al. (2019a, b)	46% initial ROSC rate, 28% sustained ROSC	Positive: presence of a criticalist, feline species, IVC in place, palpable pulses during CPR, witnessed arrest, dogs with shockable rhythm, overdose/toxicity induced arrest, $EtCO_2$ greater than 16.5 Negative: overnight arrest, heavier patients, increased team size, unknown cause of arrest	For sustained ROSC, positive factors were IVC in place, presence of pulses, witnessed arrest. Negative factors were increased team size, overnight arrest

previous guidelines (Smarick et al. 2012) do provide somewhat of an outline for care, as shown in Figure 24.1.

In the short term, clinical goals are to reverse/treat any identifiable underlying causes as discussed above, maintain cardiovascular stability (heart rate, heart rhythm, and blood pressure), improve acid–base status, and initiate neuroprotective strategies to maximize the chances of neurologic recovery.

As with any critically ill patient, the first step is to begin assembling a list of problems or physiologic abnormalities your patient has. Point of care lab work post ROSC is helpful in identifying potassium and glucose abnormalities that may benefit from treatment, though most commonly you'll see acid–base derangements secondary to the cardiac arrest event itself (Hoene et al. 2022). It is critical to assess cardiac function both structurally and functionally through a combination of ECG, point of care echocardiography (to assess cardiac contractility), and blood pressure. Depending on your findings, you can consider antiarrhythmic agents, vasoactive agents, or fluid therapy. For example, a patient with decreased cardiac contractility might benefit from a continuous rate infusion of epinephrine, while one that is hypercontractile probably needs fluid volume. The next step is to make sure you are optimizing oxygenation and ventilation as these will help correct the patient's shock state and acid/base abnormalities along with improving cerebral perfusion (Smarick et al. 2012). For patients who experienced prolonged arrest or whose cause of arrest was not quickly reversible, I would strongly recommend keeping them on a ventilator to reduce the work of breathing and better manipulate their cardiovascular, oxygenation,

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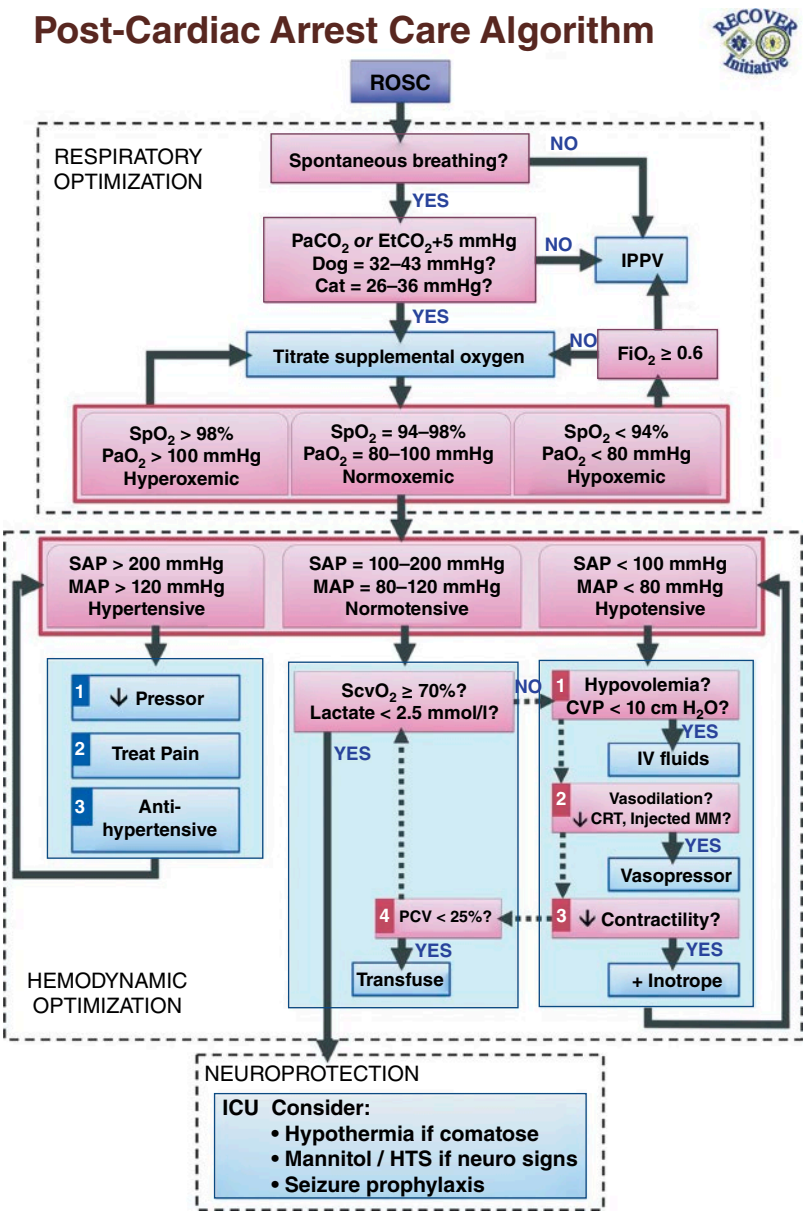


FIGURE 24.1 Post cardiac arrest algorithm based on the RECOVER initiative. *Source*: Fletcher et al. (2012) with permission of John Wiley & Sons.

and ventilation parameters. Continuous monitoring of cardiac rhythm and oxygenation (SpO_2) along with frequent rechecks of acid–base status and blood pressure will allow you to adjust your treatment plan accordingly. Patients with prolonged arrest or who remain comatose may have increased intracranial pressure and likely benefit from hyperosmolar therapy and maintenance of normothermia for neuroprotection. The role for therapeutic hypothermia is not clear in veterinary medicine and was not updated in the most recent RECOVER guidelines. What is likely more important for neuroprotection is the prevention of hyperthermia, a slow rewarming rate (do not actively warm), and aggressive seizure management (Boller and Fletcher 2020).

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Table 24.2 Factors associated with increased chance of survival to discharge for select studies published after the implementation of the RECOVER guidelines.

Study	Number of cases	Survival to discharge percentage (%)	Prognostic factors
Hoehne et al. (2022)	121	11.5	Positive: higher pH intra-CPCR and post ROSC, lower potassium and chloride levels post ROSC, feline species
Hoehne et al. (2023)	709	2.5	None evaluated
Dazio et al. (2023)	272	2.9	None able to be identified
Vega Suarez et al. (2023)	627	2.4	None able to be identified
McIntyre et al. (2014)	151	5.3	Shorter duration of CPCR, presence of IV catheter prior to arrest,
Kawase et al. (2018)	141	3.5	None able to be identified
Hoehne et al. (2019a, b)	219	9.6	Positive: cats, patients under general anesthesia, fewer people involved in resuscitation

PROGNOSIS FOR SURVIVAL TO DISCHARGE

For patients you've achieved ROSC for, it is also important to talk to owners about the chances their pet survives to discharge, as the overall survival rate for these patients remains low. I've included survival statistics and prognostic indicators for studies published after the implementation of the RECOVER guidelines in Table 24.2.

As shown above, since the overall survival is so low, identifying patients who have a reasonable chance of survival while not subjecting the others to undue trauma and discomfort is critical.

CONCLUSIONS AND CLIENT COMMUNICATION

While cardiac arrest is common, it remains a challenging condition to successfully manage. Beyond basic life support/advanced life support (BLS/ALS) RECOVER guidelines, it is important to evaluate for quickly and easily reversible conditions as those patients likely have the greatest chance of a successful outcome. When speaking to the families of the patients, understanding how the survival statistics pertain to their particular pet is critical in helping their decision-making as many patients are uninformed as to the likelihood of success (Oberholtzer and Hofmeister 2020). Involving owners in the CPCR process (Fejfar et al. 2018, Gradilla et al. 2022) may also help with decision-making, especially when deciding to stop resuscitative efforts. I will frequently bring families back when they do not want to stop CPR so that they can see and witness that process and how invasive our interventions are. Emotionally, this can also help families feel like they were present with their pet until the end, which may help with their grieving process.

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