Cardiac Emergencies in Dogs

Sarah M. Cavanaugh, DVM, MSc, DACVIM reviews three common causes of canine cardiac emergencies: congestive heart failure, arrhythmias, and pericardial effusion.

Speaker Bio:

Sarah M. Cavanaugh (Scruggs), DVM, MSc, DACVIM (Cardiology), is an assistant professor at Ross University School of Veterinary Medicine. After graduation in 2005, Dr. Cavanaugh completed her internship at the AMC and her residency & MSc at Colorado State University. Prior to joining the faculty at RUSVM in 2017, Dr. Cavanaugh was in private referral practice in Colorado and Florida. Dr. Cavanaugh's current research interests include degenerative mitral valve disease, nutrition & cardiovascular disease, and tablet-based educational tools.

Learning Objectives:

1. Recognize steps in diagnosing and treating congestive heart failure in dogs, factoring in the differences between left and right sided CHF
2. Differentiate between common arrhythmias in dogs, with attention to diagnosis and treatment of each
3. Recall elements of the diagnosis and treatment of pericardial effusion in dogs
4. Manage client expectations regarding cost, prognosis, and outcome

Slide 1

Cardiac Emergencies in Dogs

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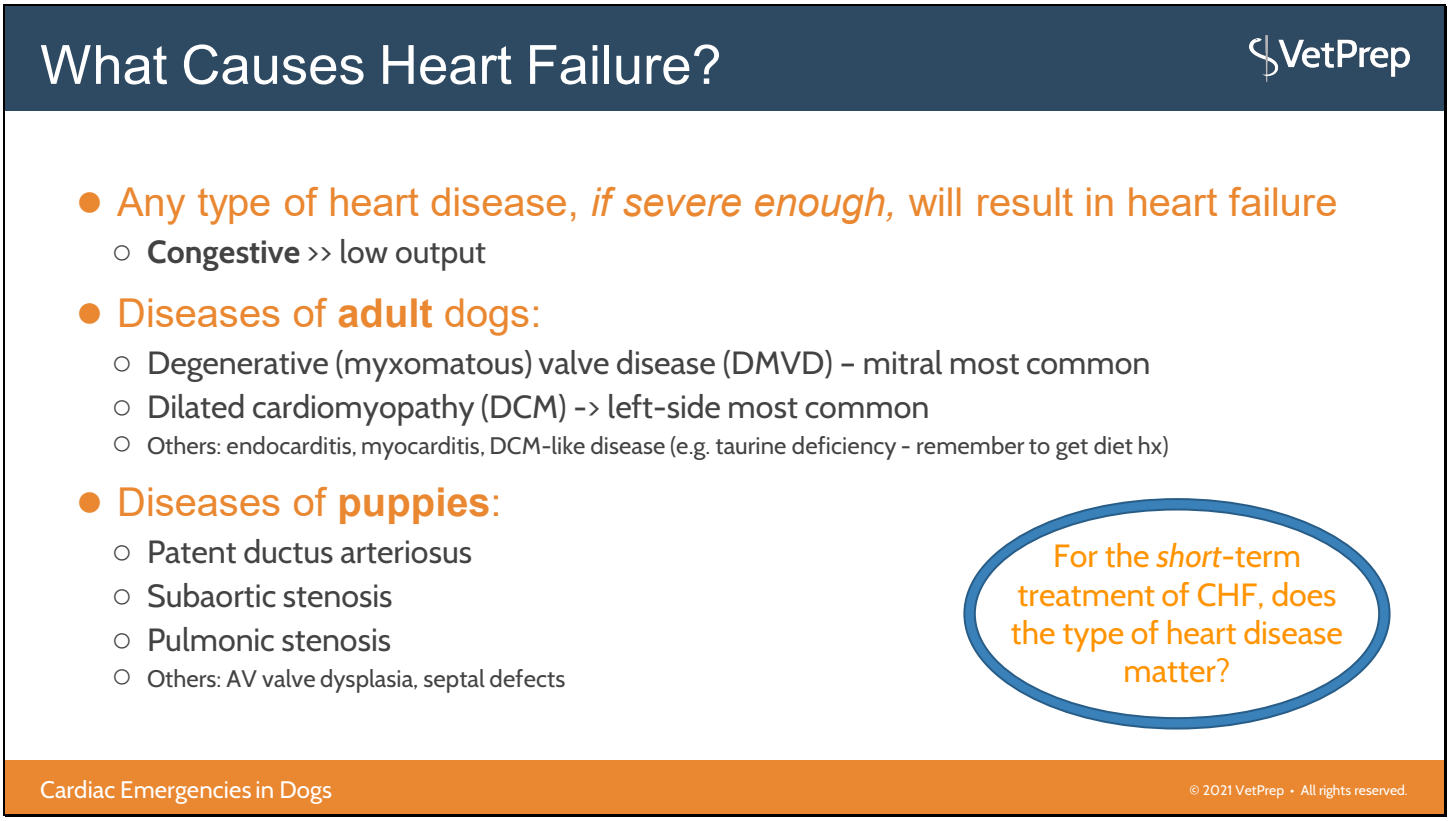
Sarah M. Cavanaugh, DVM, MS, DACVIM (Cardiology)
Ross University School of Veterinary Medicine

Hello. My name is Sarah Kavanaugh. I'm a board certified veterinary cardiologist and assistant professor at Ross University School of Veterinary Medicine. I'm really excited to be with you today to talk about some of the common cardiac emergencies we see in our canine patients. Let's get started.

Slide 2

So by far and away the most common cardiac reason, we're going to have our canine patients presenting to us on an emergency basis. It's going to be because of congestive heart failure. So that's the syndrome we're going to spend the most time talking about today.

Slide 3



From an etiologic standpoint when we think about what causes heart failure, certainly we know that any type of heart disease, if it's severe advanced enough can result in the syndrome of heart failure. And of course for our adult dogs we'd be thinking of acquired heart diseases such as degenerative valve disease, particularly in our smaller older patients and for our larger breed patients we'd be also thinking of potentially DCM. I've listed some other less common diseases as well. But if they become or progress to severe enough state they can certainly result in congestive heart failure as well. When we're seeing a young patient, a puppy or even a one or two year old, then of course we would have on our differential list some of the congenital heart diseases with PDA, subaortic stenosis and pulmonic stenosis being the top three. And even though from a big picture standpoint when we think about the long-term management of patients with cardiac disease, the type of heart disease certainly is important, it does matter. In the short-term emergency treatment of congestive heart failure, it doesn't matter a whole lot. What I mean by that is, because the most common diseases in our canine patients are degenerative mitral valve disease and DCM, and when those patients progress to heart failure we treat them very similarly in the short term. And so, when you're dealing with that emergency patient, you can feel confident and feel comfortable that having a definitive diagnosis of the disease is not critical in that short term. You can still absolutely provide the best possible care and outcome for a patient with heart failure in those initial stages without that echocardiogram or without that definitive diagnosis.

History & Clinical Signs

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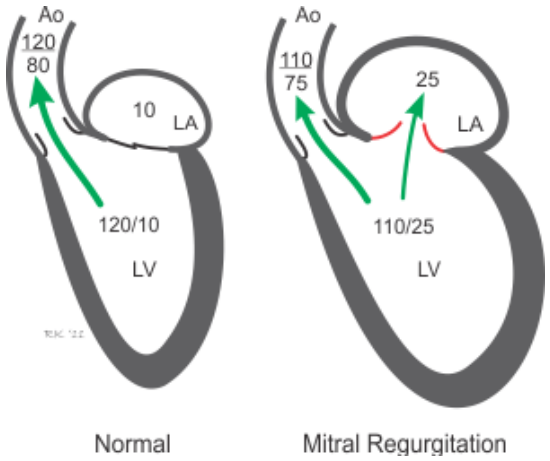
- Left-side
  - High left atrial pressure
    - High **pulmonary venous** pressure
      - **PULMONARY EDEMA**
        - Tachypnea
        - Coughing
        - Dyspnea
        - Syncope
        - Exercise intolerance
        - Secondary: hyporexia, cachexia
- Onset: often acute (days)



Cardiac Emergencies in Dogs

From a history and clinical sign standpoint, I just want to do a brief review of what we can expect or what would be some of the clinical signs or presenting complaints that can occur that we might see in dogs, that could have congestive heart failure. By far and away tachypnea or an increase in respiratory rate is going to be present in pretty much all of our patients that have left sided congestive heart failure. Certainly we can also see coughing in these patients, if they have a lot of pulmonary edema then they're going to become dyspneic in addition to being tachypneic. And what you may not know is that, some patients when they first progress into that heart failure stage, even maybe before they're showing over respiratory signs, they may have a syncopal episode. So good idea to have on your differential less when you think of that clinical sign or symptom of syncope to remember, that congestive heart failure might be the culprit or might be the cause. And then exercise intolerance as well, particularly if it was a patient that was maybe previously or historically more active, that may be something that the client has noticed. Patients that do have congestive heart failure be it left or right sided, as they started to have difficulty breathing, then a lot of times as a result of that, we will see their appetite drop off. And in some patients, particularly those with dilated cardiomyopathy, sometimes they will have a drop in their appetite even before the onset of those respiratory type signs. Cardiac cachexia is very much a possible sequelae of heart failure. And so these patients we might notice as well, or maybe the client has noticed that they've started to maybe lose some of their muscle mass. And that might be part of the presenting complaint as well. But by far and away it's really going to be those respiratory signs with tachypnea, coughing, and dyspnea being at the highest top of the list. Onset, when we think about, is this more likely a respiratory disease patient or is this maybe a heart failure patient, when we're seeing a patient present on an emergency basis with respiratory signs. Looking at the duration of the signs can be really

helpful. So left-sided congestive heart failure, if that's what's going on, they usually-- they are going to have a pretty short duration. So the client's going to have noticed these change or notice these clinical signs usually within a matter of days. This isn't going to be a real prolonged history of tachypnea, coughing or dyspnea.

Slide 5

History & Clinical Signs

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- Right-side
  - High right atrial pressure
    - High **systemic venous** pressure
      - **PERITONEAL and/or PLEURAL EFFUSION**
        - Abdominal distension
        - Tachypnea
        - Dyspnea
        - Cough
        - Secondary: hyporexia, cachexia, diarrhea
      - **ABDOMINAL ORGAN EDEMA**
        - Abdominal distension
        - Cranial organomegaly
- Onset: often subacute (weeks)

Cardiac Emergencies in Dogs

We're thinking about right sided heart disease and therefore if it becomes severe enough, those patients can then develop right-sided congestive heart failure. Now the issue or the alteration in the hydrostatic pressure is on that systemic venous side. In contrast to the pulmonary venous side that we think about with the left-sided heart diseases. So patients that progress from their right-sided heart disease and a right-sided heart failure, their congestion or their fluid buildup for dogs especially is typically going to be in their peritoneal cavity. So they're going to develop abdominal effusion and usually then that's going to start to cause some abdominal distention. Certainly they can have respiratory signs as well too but they may not always. If they do not have concurrent pleural effusion, then they might be breathing pretty well and you might only be appreciating or noticing the abdominal distention. Depending on the severity of their right-side cardiomegaly and again whether or not they have concurrent pleural effusion, they might present with some coughing as well too. Occasionally every once in a while a dog with right sided heart failure will have predominantly pleural effusion and no or minimal peritoneal effusion. But that's really more typical in cats. In dogs with right sided usually they're going to have the big bellies from a lot of effusion. Of course there we're talking about not only abdominal effusion, but some edema or congestion in their abdominal organs too. So as you begin palpating these guys, a lot of time you're going to notice some cranial organomegaly oftentimes associated with enlargement of the liver. The onset of signs of the duration that you're going to be getting from these clients or these pet parents for right-sided heart failure, a lot of times it's more of a subacute onset. So again particularly if their breathing is not affected, if they are dealing mostly with the abdominal manifestation, then it might not become very obvious that there's something wrong to the clients, until there's been—until a couple of weeks have passed and there's a lot of abdominal distention. So a little bit of a difference there. When

we're trying to make the decision between, is this more likely left or right sided, or is it some other cause, some completely different system or disease process that's causing abdominal distention.

Slide 6

Approach

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- Patients suffering from cardiac emergencies are fragile
- The veterinary team needs to be:
  - Calm
  - Gentle
  - Efficient
  - Cohesive

Our approach to these patients, and this isn't really for-- so much for us as a veterinarian, but I like to remind myself because our team, those that are working with us with our own patients that have cardiac emergencies, especially if it's maybe a more junior assistant or technician, they may benefit from understanding that these patients are very fragile. And so I think one of the most important things we can do is to make sure that we're calm. And if you have some time or you have the opportunity with your team, with your emergency team in particular to do some simulations, to do some role playing, to do a practice CHF case if you would. I think that gives the junior staff members really the support that they would need when they do have that first case to feel better and more confident about it. But certainly the more calmness that we can exude, keeping things peaceful and you know we want to be swift, we want to be quick, but we can do all of those things very calmly and also very gently. Especially when we're thinking about handling our patients and handling those clients. So as zen as possible. And again you know certainly just like our patients in heart failure, when we're managing and as you start managing more and more heart failure patients, I mean our response is to have that bump in our sympathetic nervous system. That's completely normal, completely expected, still happens to me. You know and just really taking a moment, taking a breath or two as I'm moving, as I'm talking, as I'm gathering what I need, can really make a huge, huge difference for the outcome of these patients.

Slide 7

Physical Exam

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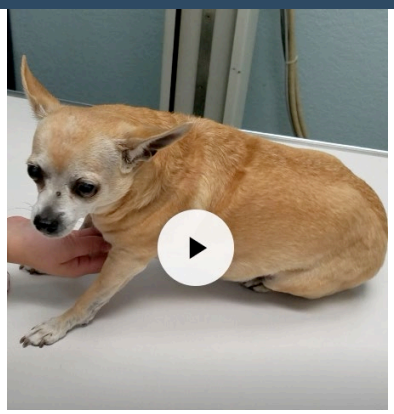

- Left-sided CHF
  - **Tachypnea**, +/- dyspnea
  - **Increased heart rate** (high-normal or tachycardia)
    - Exception: chronic bradyarrhythmia
  - **Abnormal heart sound(s) common**
    - Underlying disease will dictate type
      - E.g. DMVD – left apical systolic
  - Normal lung sounds if early/mild pulmonary edema
  - Increased breath sounds/crackles/wheezes with more significant edema
  - Muscle loss
    - https://wsava.org/wp-content/uploads/2020/01/Muscle-Condition-Score-Chart-for-Dogs.pdf

Cardiac Emergencies in Dogs

What about once we start to really do our exam and a lot we can tell in patients just from our distance examination. So we can assess whether or not they've got tachypnea and dyspnea. And again, as we mentioned if we thinking about patients presenting a left-sided congestive heart failure, they're almost always going to have an increase in the respiratory rate and then if they've got a lot of pulmonary edema they are probably also going to be dyspneic. And this little guy right here to this little chihuahua I'd like to just have you look, not only at his breathing pattern but his mouth too. You can see his mouth throughout a portion of the video here. And I think this is a really nice example of-- sometimes I get the question or even I question when I'm seeing a patient. Are they just panting or are they dyspneic? And you can see he was doing some open mouth activity. Where you might think, well, maybe he's just panting. But when you see that combined with the increased chest excursion, and in him you could even see some abdominal effort or abdominal component. You can feel very confident that no he's probably-- that's probably actually open mouth breathing. He's probably actually doing that because he is having trouble breathing, and he's trying to maximize his respiration by opening his mouth. And so initially he wasn't having dyspnea, he was just having some tachypnea. Thinking about doing radiographs in him but then, now seeing that just in that little bit of extra stress, probably taking away and taking him moving him from his family getting him into the radiology area. That will sometimes cause patients in heart failure to decompensate. And so perfectly fine to abort, and that's exactly what was the decision that was made here. Because he was showing this dyspnea. Not worth taking the chance of restraining him to get those radiographs. We can go ahead and proceed with treating and get him stabilized before doing those additional diagnostics. The other big thing to really keep in mind if you're-- especially if you're unsure if you're dealing with heart failure, is that those patients are almost

always going to also have an increased heart rate. Either up on the normal, high normal range or they might actually be tachycardic. Also abnormal heart sounds are very common. What I mean by that is usually they're going to have a murmur, maybe a gallop, or an arrhythmia. So those, again, for dogs, when they progress to heart failure, almost always you're going to find or you should find on your auscultation murmur, gallop, or arrhythmia. Some patients may be all three. If the pulmonary edema is early or just a mild amount, then your auscultation might be normal. You might just-- you might hear relatively clear lung sounds even if they are mildly tachypneic, but as the pulmonary edema becomes more and more, that's when we're going to start usually hearing some increase or louder breath sounds. or even potentially some inspiratory crackle sounds and even wheezing as well too. Some patients with pulmonary edema they'll start to have some bronchoconstriction or bronchospasm particularly as the edema gets worse and worse. And so that can create some wheezing sounds on our auscultation. As we mentioned cardiac cachexia a possibility. And particularly in our patients that have lived with chronic heart failure most, not most, but many of those patients are going to have muscle loss over time. And I just included there, If you're not familiar with the muscle condition scoring chart there's just a really nice visual representation of that for scoring our patients.

Slide 8

Physical Exam

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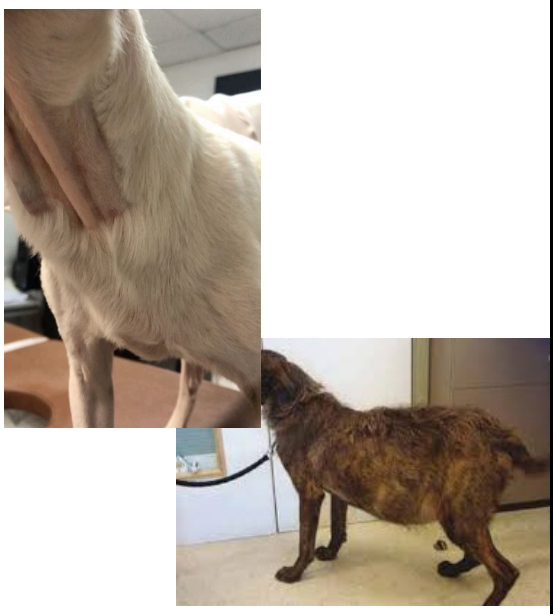
- Right-sided CHF
  - **Increased heart rate** (high-normal or tachycardia)
    - Exception: chronic bradyarrhythmia
  - **Distended jugular veins**, +/- pulsation
  - **Abnormal heart sound(s) common**
    - Underlying disease will dictate type
  - **Abdominal distension** due to peritoneal effusion
  - Tachypnea/dyspnea due to pleural effusion (or severe peritoneal effusion)
    - Muffled or absent lung sounds
  - Muscle loss



Cardiac Emergencies in Dogs

Right-sided heart failure, how is their physical exam different? Some similarities, some differences. Similarity would be the increased heart rate. So heart failure across the board that means those patients have reduced cardiac output and oftentimes are hypoxic either from pulmonary edema or pleural effusion. And so that-- the combination is going to create that sympathetic response. And so the increased heart rate, as long as it's a sinus rhythm or sinus tachycardia for example, remember that's an adaptation, that's compensatory for these guys. So that's why it's happening. And what that also means is that if it's a sinus rhythm, that as we get the heart failure under control. So as we manage these patients in-- especially those that need to be hospitalized. It's so beautiful to see not only their breathing get better, sometimes hopefully within a matter of hours, but also their heart rate decrease as well too. Right-sided heart failure, their jugular veins remember are going to be distended. That's a reflection of that increase in the systemic venous pressure. If they've got a lot of tricuspid regurgitation they'll have some pulsation as well there too. So this guy here-- I do have a special love for chihuahuas. But this guy happens to be a chihuahua too. But I swear that it's not all chihuahuas that presented heart failure. But certainly we do see mitral valve disease and also chronic respiratory disease in lot of these guys. And this particular patient one of his problems causing or contributing to his right-sided heart failure, was actually that he had chronic respiratory disease that led to severe pulmonary hypertension. He also had degenerative tricuspid valve disease and the combination just kind of put his right side over the edge. And you can see there he's got a distended jugular vein, he's also got some pulsation because of the tricuspid regurgitation. Again the most common finding though in dogs with right sided heart failure, is going to be the distended abdomen because of the abdominal organ congestion. But also the peritoneal effusion that is extravasating from the circulatory system from those high hydrostatic

pressures. Keep in mind that if they do have a lot of pleural effusion then-- that they might have respiratory trouble as well too and muffled or absent lung sounds on auscultation.

Thoracic Radiographs

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- NOT in a dyspneic patient
  - Make a presumptive diagnosis based on:
    - History
    - Clinical signs
    - Physical exam findings
  - Initiate emergency treatment
  - Perform diagnositcs once patient is no longer dyspneic

I mentioned this a little bit earlier, again just a little bit of a reminder that thoracic radiographs they give us so much information and especially in a patient that's coming in with respiratory signs. There are our go to. But if the patient is dyspneic we really have to just pump the brakes. And step back and say, the risk of restraining this patient lateral recumbency or a DV or VD radiograph just outweighs the potential benefit. And that is the case sometimes in patients and dogs with acute and more severe left-sided congestive heart failure. The good news is that you can usually-- you will be able, usually using those clues we talked about from the history, clinical signs, and your physical exam findings, to make a presumptive diagnosis. So please feel okay-- when we manage congestive heart failure on an emergency basis, we do not have initial radiographs in lot of those patients. That is completely okay and trust me you'd rather be in that situation than push the patient to get the radiographs and they decompensate. So we're going to usually-- if we're dealing with a dyspneic patient, then we're going to use that other clinical information. And we can initiate our emergency treatment of congestive heart failure once we get them stabilized and they're no longer dyspneic. Then we can do some additional diagnostic testing.

Thoracic Radiographs

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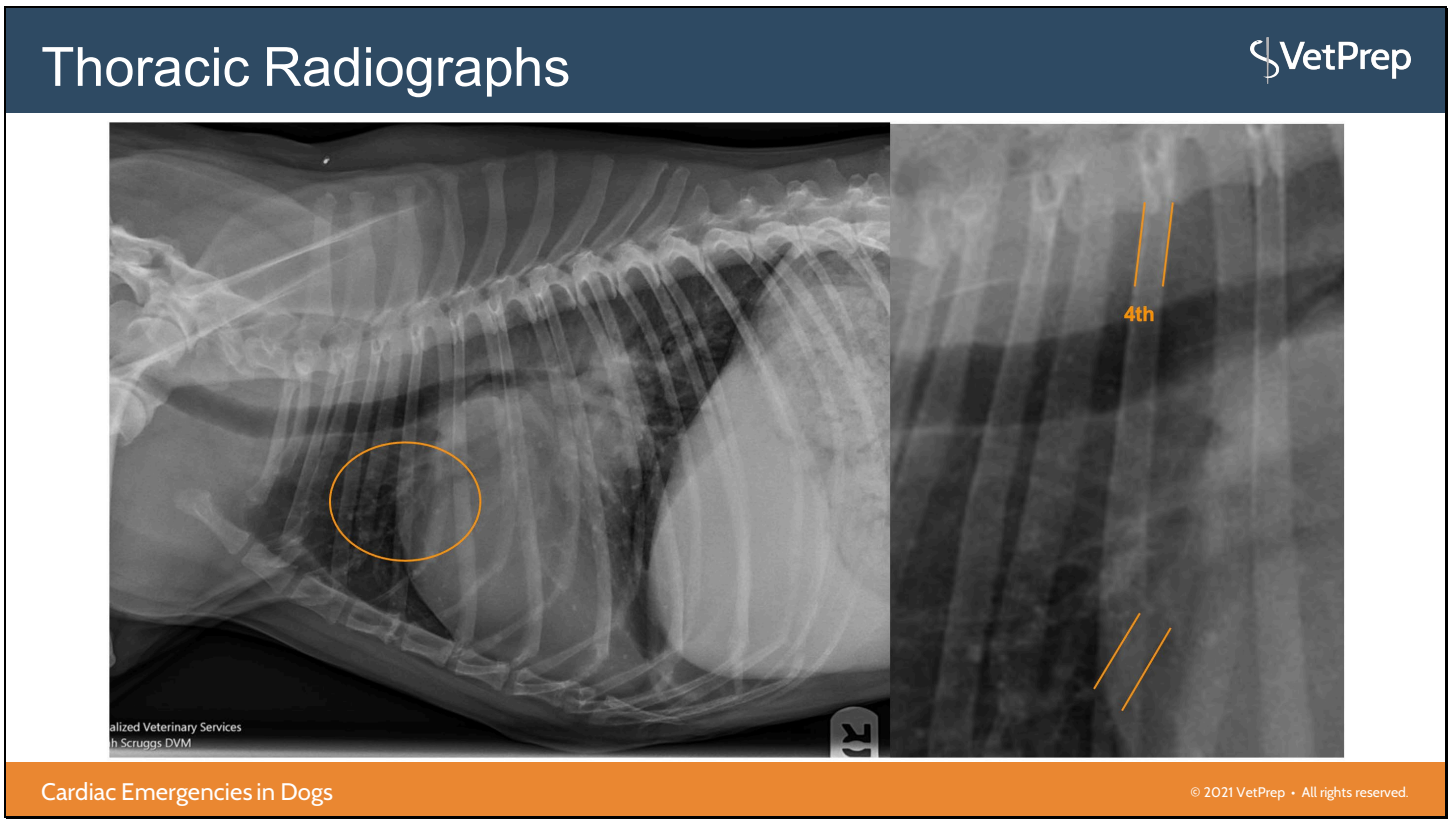
- Criteria for the diagnosis of left-sided CHF*
  1. Left-sided cardiomegaly characterized by **moderate-severe**** left atrial enlargement + #2
     - Tools: vertebral heart size (VHS) & vertebral left atrial size (VLAS)
  2. Interstitial pulmonary infiltrates, +/- alveolar infiltrates
     - Edema typically begins in the perihilar region -> caudo-dorsal & then (if untreated) spreads cranially & ventrally
  3. Enlargement of the cranial and/or caudal pulmonary veins (not always present or visible)

*Criteria are the same regardless of the type of underlying heart disease

**Exception: Chordae tendineae rupture (regurgitation worsens acutely, so atrium has not had time to sufficiently dilate)

I do want to mention as a recap of for criteria, radiographic criteria, for the diagnosis of left-sided congestive heart failure. So thoracic radiographs are still really our main way and best way to diagnose congestive heart failure in dogs when they're stable enough to tolerate those radiographs. So when we see the combination of significant left atrial enlargement and an interstitial pulmonary infiltrates, and for dogs remember the pulmonary edema is usually going to start in that perihilar region. So if you catch it early you're usually just going to see those infiltrates in the perihilar region. We'll look at that in a minute. And then it'll spread. The edema will spread caudodorsally. Now if left untreated the edema will ultimately affect all of those long lobes. But those patients, a lot of times, we don't have the radiographs on those patients because they're too unstable, too dyspneic to get the pre-treatment radiographs. We have third element from the radiograph, so we want to look for our-- look out of the pulmonary vein. So oftentimes patients with congestive heart failure they're going to have notably dilated or enlarged pulmonary veins. Now this-- you don't have to have enlarged or dilated pulmonary veins to make the diagnosis. If you have severe left atrial enlargement and you've got the kind of the typical pulmonary pattern, that's sufficient. But certainly if you also have the finding of enlarged pulmonary veins that's further support.

Slide 11



Those cranial pulmonary veins, we're going to see those best from our lateral view and they're in that-- the orange circle that you see here. Pulmonary vein and artery should be the same size, veins are ventral on the lateral, central on the VD or DV. If you want to be a little bit more objective about the size of the veins, remember that you can compare the width of the veins. So I've got the pulmonary vein here in the second image on your right. outlined with the two orange lines, and then the borders of the proximal fourth rib outlined. And so if we're seeing the width of the vein more than the width of the rib, then that's consistent or evidence of pulmonary venous enlargement or pulmonary venous distention. And this is a patient too that has a lot of severe left atrial enlargement too. So my mouse is kind of tracking or outlining the back of that left atrium. This patient also, as you can see here some fuzziness in that perihilar region. So this patient is at the very beginning of left sided congestive heart failure. They're just starting to have a little bit of pulmonary edema there.

Thoracic Radiographs

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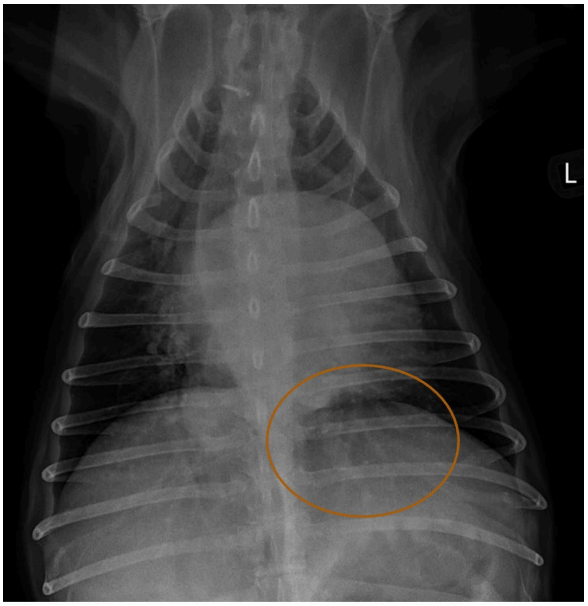

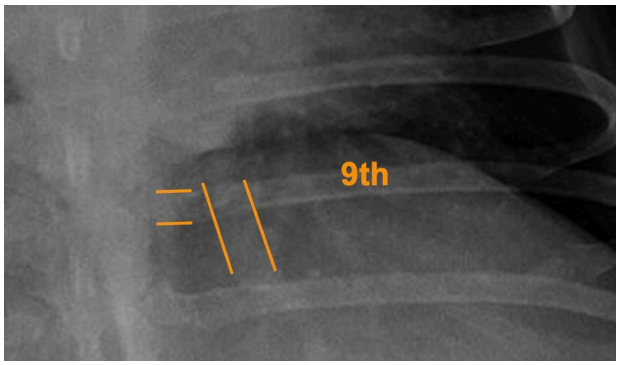

Cardiac Emergencies in Dogs

And for looking at those pulmonary veins remember we're going to get the best look at those from caudally. So caudal to the cardiac silhouette and a lot of times a dorsal ventral projection will allow a little bit more, better visualization those caudal veins. So here on the dorsoventral or ventrodorsal the veins are central. Meaning close near midline. So ventral on the lateral, central on the VD or DV. And here we can compare the width of the rib to the-- excuse me, the width of the pulmonary vein to the width of the ninth rib to get a more objective assessment. And if it's bigger more than the width of that rib, then that is-- that's evidence of those pulmonary veins are big.

Vertebral Left Atrial Size

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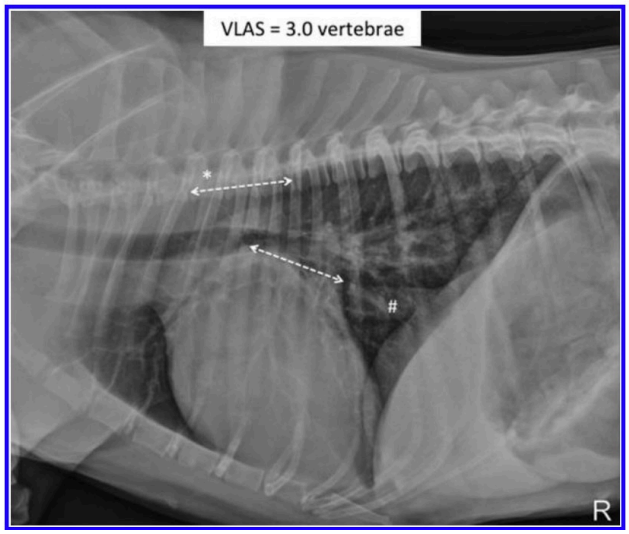

J Am Vet Med Assoc. 2018 Oct 15;253(8):1038-1045. doi: 10.2460/javma.253.8.1038.

Diagnostic value of vertebral left atrial size as determined from thoracic radiographs for assessment of left atrial size in dogs with myxomatous mitral valve disease

Elizabeth L Malcolm, Lance C Visser, Kathryn L Phillips, Lynelle R Johnson

Figure 1—Representative right lateral thoracic radiographic image of a dog with LA enlargement secondary to MMVD that depicts measurement of the VLAS. A digital caliper was used to draw and measure (in arbitrary units) a line from the center of the most ventral aspect of the carina (bifurcation of the left and right mainstem bronchi) to the most caudal aspect of the left atrium where it intersects with the dorsal border of the caudal vena cava (#). Then, a second line that was equal in length to the first was drawn beginning at the cranial edge of T4 (*) and extending caudally just ventral and parallel to the vertebral canal. The VLAS was defined as the length of the second line expressed in vertebral-body units to the nearest 0.1 vertebra. For this dog, the VLAS was 3 vertebrae.

I know most of you are familiar with-- probably learned in school the vertebral heart size developed by Dr. Buchanan a while back. But I also want to mention another measurement that maybe you heard of or maybe not, called the vertebral left atrial size. And I think this is a really helpful assessment in the setting of emergencies and particularly congestive heart failure. Because remember with left sided congestive heart failure, the expectation is that there's a lot of left atrial enlargement. And so this particular assessment focuses just on looking at the left atrial size. So the measurement, or you would do your caliper, draw your line is from the ventral aspect of the carina. And then to the furthest or caudal most point of the left atrium where it intersects with the caudal vena cava. So caudal vena cava is here where this pound sign is. And so that's going to be your end point. And then just as for vertebral heart size you go to the fourth thoracic vertebrae and count the number of vertebrae. So again vertebral heart size tells us about the overall size of the heart. Vertebral left atrial size tells us specifically about the size of the left atrium. And there's a really nice paper, provided that reference there for you as well too. Just to give you an idea, normal dogs, there VLAS usually falls within the range of about 1.8 vertebrae to about 2.3. Dogs that have severe heart disease and particularly those that are in left-sided congestive heart failure have a VLAS in the upper 2's and into the 3s's

Thoracic Radiographs

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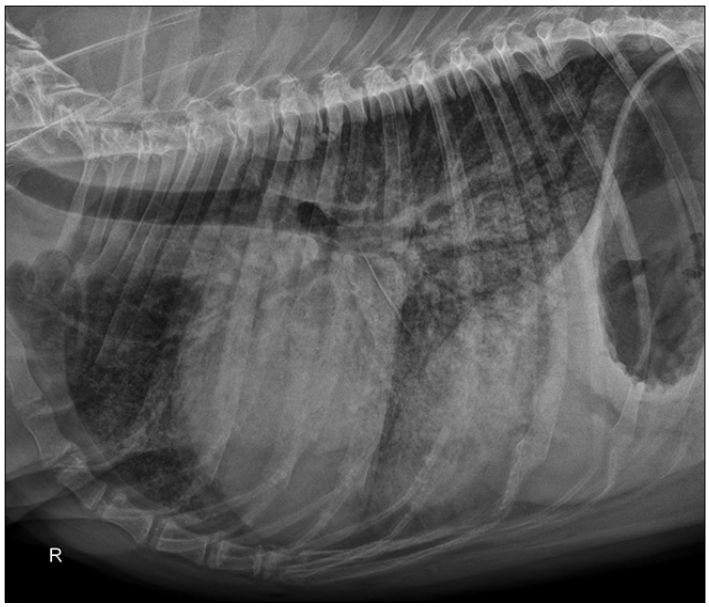

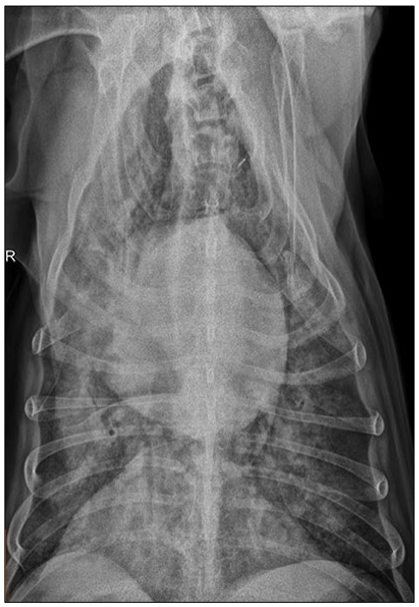

Another example here of thoracic radiographs from a canine patient. This is a canine patient with left sided congestive heart failure and ultimately figured out it was secondary to dilated cardiomyopathy. So much more pulmonary edema in this example here. But again we've got straightening of that caudal cardiac silhouette consistent with the left atrial enlargement. It would be pretty tough to do a VLAS in this patient because the pulmonary infiltrates will sometimes make it too difficult to see those borders.

Treatment

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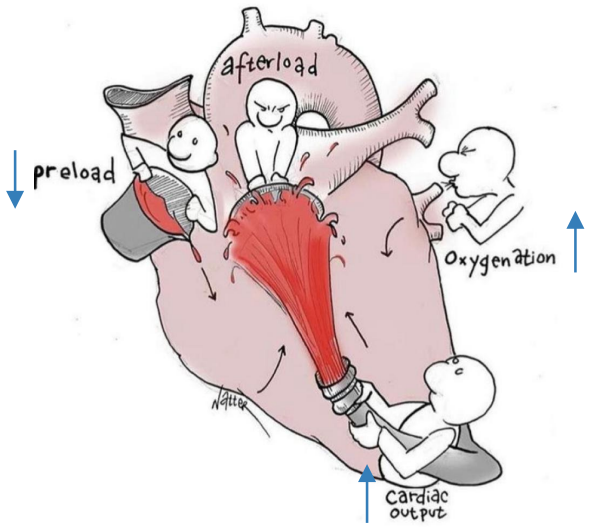
- Goals of emergency therapy:
  - Relieve congestion
  - Improve cardiac output
  - Support the respiratory system



So now we've moved past where we now need to think about how are we going to manage these patients. once we've made either maybe when a patient isn't yet dyspneic, we were able to get thoracic radiographs. So we have a definitive diagnosis that what we're dealing with is left-sided congestive heart failure. or patient's dyspneic, we're in a really critical situation. And from information from our history, clinical signs, and physical exam have pushed us to have CHF as our top differential. And one thing I want to mention, if we were dealing with right-sided congestive heart failure, radiographs are still going to be very helpful. But of course in that scenario instead you'd have significant right-sided cardiomegaly. Remember right-sided heart failure is not going to cause pulmonary edema, right sided heart failure is not going to cause pulmonary edema. But you would see peritoneal effusion, potentially pleural effusion. But it would need to be in conjunction with a lot of right-sided enlargement and then also usually you'll be able to see some enlargement or distension of the caudal vena cava on your radiographs as well. So goals for therapy, patients that are having respiratory troubles are really our first and foremost goal is to relieve their congestion. We got to get them-- we need to get those lungs clear if we're dealing with pulmonary edema. If they're having breathing trouble because it's right sided heart failure and they have pleural effusion then we need to get that pleural fluid out of there as quickly and effectively as possible. So relieve congestion but also improve cardiac output. I love this illustration because it really ties in those physical parameters of preload, afterload, and contractility. But puts them in I think the visual representation that I just think is really helpful. And in addition to improving cardiac output oftentimes these patients are going to need some respiratory support too, most often in the form of supplemental oxygen. So we want to decrease preload. That's going to be our most effective means to relieve the congestion. We want to improve cardiac output, that's going to not only help to

maintain their blood pressure, but the more we have remember going forward, the less there can be backing up. And then oxygenation as well to so supporting their respiratory system.

Treatment

VetPrep

- Provide supplemental oxygen (FiO2 40%) if:
  - Any dyspnea,
  - RR >40 at rest
  - Excessive coughing, or
  - SpO2 <95%
- Every emergency CHF patient needs:
  - Furosemide IV (or IM initially)
  - Minimize stress, handle gently
- Patients with R-sided CHF may need:
  - Thoracocentesis
  - Abdominocentesis

So you might be wondering, if you haven't yet managed as the primary clinician in the congestive heart failure patient. You may be wondering, how do I know which patients need supplemental oxygen versus which don't? Well a lot of times it's obvious. So a patient that's having increased effort or any degree of dyspnea, go ahead and give those patients supplemental oxygen straight away. If I'm concerned that whether it's a cardiac patient, and I've got concern for congestive heart failure, and they're tachypneic at rest, usually going to give those guys some supplemental oxygen as well too. At least as I'm figuring out exactly what's going on and refining the therapeutic plan for them. I also find supplemental oxygen to be helpful in patients with heart failure that are having excessive coughing. Those patients will sometimes decompensate from all the coughing. They literally will, may having so much coughing they can't catch their breath. And so if they're already struggling with oxygenation with gas exchange and now or now because of the coughing they're not allowed to get a break and breathe in between, their hypoxemia is going to be even more severe. So I also like to give those patients some supplemental oxygen. If you're not sure, maybe a patient is not dyspneic. Their effort is normal but their rate is up a little bit and you're just not sure or you're just trying to get something a little more concrete. Then check a pulse ox. That's another really helpful way to make that decision. Do they need some supplemental oxygen or do they not. Remember every emergency CHF patient needs furosemide. That's going to be across the board. And when they're in that emergency or acute situation, we want to quickly, very quickly get that congestion under control. So we're going to give the furosemide to them, the loop diuretic to them parenterally. Either intravenously or initially maybe an intramuscular injection. And then of course we were going to minimize their stress level and handle them as gently as possible. Keep in mind if it's a right-sided congestive heart failure and they're having breathing difficulty, check them.

Listen again maybe if you need to. Because they might have pleural effusion and need to have a thoracocentesis. That's going to be the patients with a lot of pleural effusion. You would still give them furosemide but that-- the furosemide, if it's a lot of effusion it's not going to help that fast enough. So we need to just manually remove that and then also have the furosemide on board. Some of these dogs too, some of you may have already experienced this. Some of them will come in with sometimes a few liters of abdominal effusion. And just the pressure on their diaphragm, they don't have pleural effusion and respiratory pathology. But just the pressure on their diaphragm from the abdominal effusion can sometimes impact their breathing. And so don't hesitate to make them not only breathe better but just more comfortable to get out that peritoneal effusion with abdominocentesis.

Treatment

VetPrep

- Furosemide – loop diuretic
  - 2-4 mg/kg IV or IM boluses
    - Dose & frequency depend on severity of pulmonary edema and response to therapy (see next slide)
      - Severe cases often require 3+ boluses over the first 1-2 hours
    - IM route is a good option for dyspneic patients who may not tolerate IV catheter placement
  - Patients with moderate or severe edema: bolus(es) initially then continuous rate infusion (CRI) 0.5-1 mg/kg/hr (or continue boluses if CRI not available)

So we talked about furosemide and everyone's familiar with furosemide. It's a loop diuretic but I wanted to really focus on now. Because I think a lot of times in that school really but for your own practice it is. But how much furosemide do they need and how do I make that decision. So that's really what I want to focus on. I've given you a dosage range then I mentioned to give you an IV or IM. And then the dose and the frequency, how often we repeat the bolus depends on the severity of pulmonary edema and response to therapy. That's pretty vague and I appreciate that and that's what all the formularies say. So let's get a little bit more specific. So for example, patients with severe pulmonary cardiogenic pulmonary edema, I usually giving them three or more boluses within the first hour or two. Just to kind of put into perspective how much furosemide they might need. And if a patient either based on my-- how severe their respiratory signs are in my auscultation, or maybe there were radiographs taken. If they've got moderate or severe pulmonary edema you can absolutely start at that high end. So start with a 4 mg per kg dose. Again those severe cases, a lot of times I'm giving them three or four mg per kg three or four times in the first hour or two. IM is a really good option for dyspneic patients who you just think they wouldn't even tolerate getting an IV catheter or you're not sure. If you're not sure always err on the side of caution with these dyspneic patients. Quick and easy, tiny needle not much discomfort for them at all. And it's going to start working on that congestion. And then once they're stabilized you can get an IV catheter in them. But trust me or you may already experience some patients come in and just the restraint in handling, that is necessarily to get a good IV catheter is sometimes too much for these guys. So not to worry not to fear, IM is going to work almost as quickly and almost as effectively as giving an IV. We can also offer a continuous rate infusion of furosemide. So patients that have moderate or severe edema are still going to start with a bolus or sometimes a couple of bolus while we're getting

our teams getting a CRI ready and going. Like any CRI that really requires the tech support and the nursing support, and usually need to be in an ICU or 24 hour ICU facility to do that. But more and more general emergency practices have those capabilities. So I find that to be really effective. If you can safely do a CRI of furosemide and you've got a patient with moderate or severe edema I would advocate for doing that. It's going to keep that level of furosemide systemically at a much more constant level, and can be very effective for getting the pulmonary edema cleared up. One thing I want to point out though with the CRI remember these patients don't need fluid. Fluid is going to make these patients worse. And so when we do a CRI of furosemide we're going to use the smallest volume possible. We're going to only put it in enough volume to deliver the drug. We're not trying to give them fluid replacement or fluid support. So it's going to be a really small rate or low rate CRI.

Response to Diuretic Therapy

VetPrep

- Improved respiratory rate (RR) & effort (RE)
  - Mild-moderate cases usually show some improvement within 30 mins of first dose
- Once RR & RE begin to improve: Cautiously taper diuretic dose (usually over 12-24 hrs) to reach maintenance dose (2 mg/kg BID)
- Once RR has normalized (20s-30s at rest): Taper supplemental oxygen over 6-8 hrs

So we've given the furosemide and maybe we've given a couple of doses. Now where do we go from here? And I think until you've managed a few of these cases, it's really impossible to know. So what I hope you can take away from today is that, one of the biggest things that will help you and helps us make that next decision of what am I going to give the next dose of furosemide? And how much am I going to give or I've got him on a CRI when am I going to start to taper this CRI? How much their respiratory rate and respiratory effort if they have dyspnea is improving. What is the expectation? For mild to moderate cases most of those dogs, if we're dealing truly with cardiogenic pulmonary edema, usually you're going to see some improvement in their breathing within about a half an hour of the first dose. Now severe cases sometimes it's longer but most of the patients are going to fall into that mild or moderate range. And so that will hopefully help you, that can help manage your expectations. And as we're starting to see that improvement then we can start spacing out the time between doses. So for example, if you needed to over the first four hours you needed to give a dose every 45 minutes or 60 minutes. But now by hour three or by hour four you've seen a marked improvement in their respiratory rate. Well now you can go to maybe every four hours or even maybe every six hours between dosages. A little bit of it is individualized. So but that's typically what I do if I'm at an hourly dose then as I'm seeing significant improvement then I'm usually going to go to q4 hours. And then if I'm seeing more improvement, q8 hours and then q12 from there. And as you're doing that wean as you're doing that taper things get worse, that's okay. Sometimes you're going to have to go back and give it more frequently or bump up your dose for a couple of boluses. I'll tell you that most dogs with left-sided congestive heart failure are the ones that require hospitalization, it's usually a pretty short hospitalization. Usually a day some patients will need two days. So that's the good news. I also mention

that because as we said sometimes respiratory disease can look similar to CHF. So if you're following these guidelines, you've given a couple of doses or a few doses of furosemide and they're not getting any better, it's time to just take a pause and maybe reconsider that maybe you're actually-- maybe the patient actually has a primary respiratory disease and not congestive heart failure. It shouldn't take hours of furosemide before you're starting to see improvement. Usually again for most cases, we're going to start to see some improvement in terms of their respiratory rate decreasing, and if they had dyspnea their respiratory effort decreasing within about a half an hour. What to do about the-- if they've been on-- they were hospitalized so they've been on supplemental oxygen as well. So a good rule of thumb is I usually don't start to think about tapering their oxygen until the respiratory rate has normalized. They're going to benefit from that supplemental oxygen until most of that pulmonary edema is gone. So once they start getting readings in the 30's for their respiratory rate at rest, then we can start thinking about tapering their supplemental oxygen. Generally can do that over about six to eight hours. And what you're mostly monitoring for as you do that, is their respiratory rate staying stable. If it's starting to tick back up as tapering then it means they need to stay on the oxygen for longer.

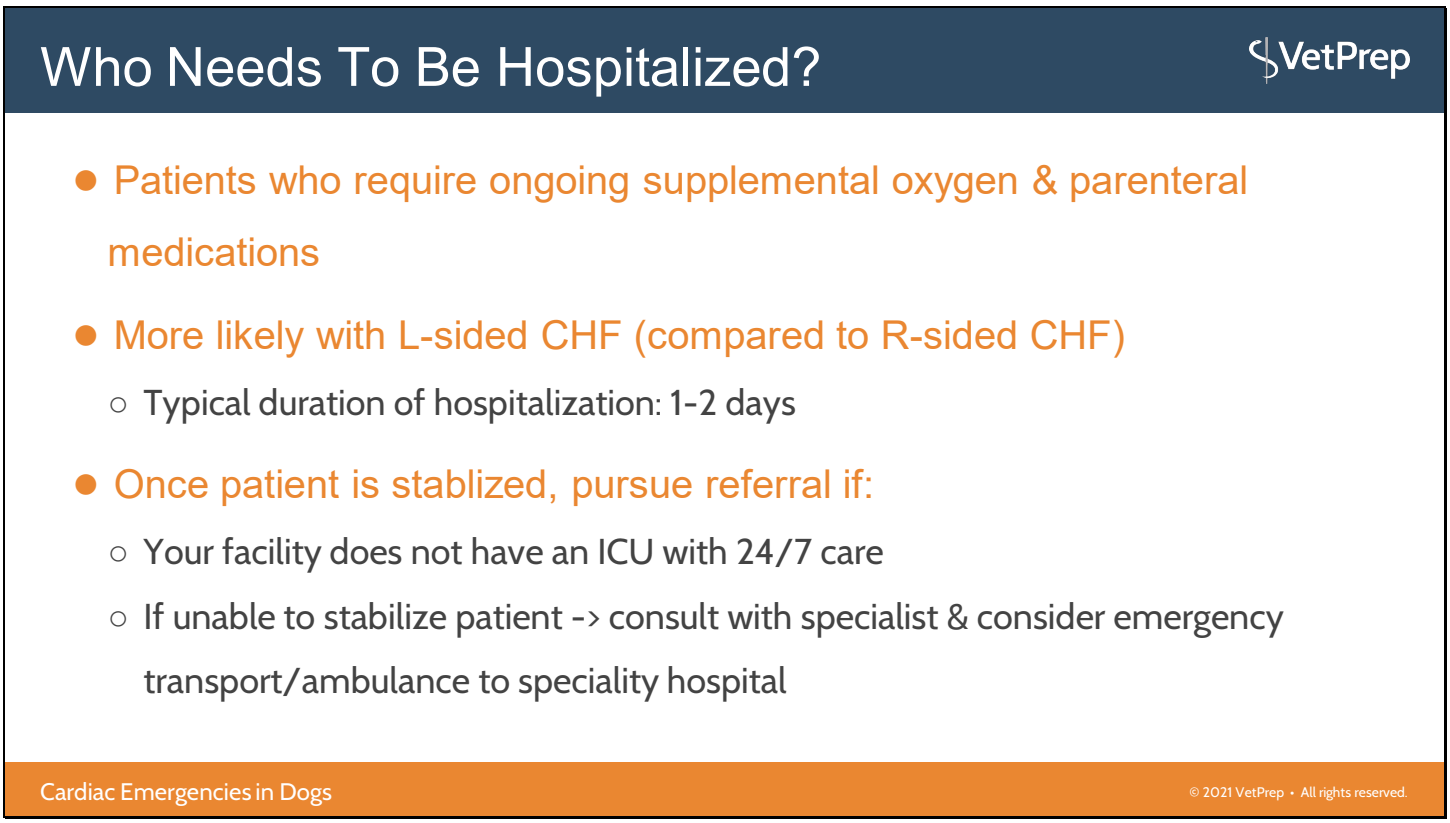
Treatment

VetPrep

- Pimobendan – positive inotrope & vasodilator
  - For patients with known *or suspected* degenerative valve disease, DCM, PDA, valve dysplasia, or septal defect
    - Suspicion based on signalment & murmur characteristics
  - 0.25-0.3 mg/kg PO BID (OK to hide in treat or canned food)
    - If patient will not take voluntarily, crush + H_2O & give as slurry

Most dogs that you're going to be dealing with congestive heart failure an emergency basis, are going to also need pimobendan. So remember this is the inodilator. So it has both positive inotropic properties. And it's a vasodilator, predominantly arterial vasodilator. And it's labeled for patients with degenerative valve disease and also for patients with DCM. But we also use it routinely if a patient-- if we're dealing with a young patient with congenital heart disease that has caused them to go into congestive heart failure. So PDA patients with valvular dysplasia or septal defects. But again most-- in clinical practice most of the patients you're going to be dealing with heart failure it's going to be acquired heart disease. They're going to be adult patients a lot of times senior or even geriatric. And those cases are most often going to have either mitral valve disease or dilated cardiomyopathy. So pimobendan is going to really help from the cardiac output standpoint. So that second goal that we talked about pimobendan does come intravenously, but I know that most general practices and even most emergency practices aren't going to have it. But you're going to have the tablets. And it begins working even orally quite quickly. Within an hour, it starts having those pharmacologic effects from the oral route. So we absolutely use it and it should be used for acute heart failure in dogs, especially if you think it's because they've got degenerative valve disease or dilated cardiomyopathy or one of those congenital heart diseases if you were dealing with a younger patient. Remember those dogs with degenerative mitral valve disease, those guys are always going to have that left apical systolic murmur. A lot of dogs with advanced DCM will also have, usually are going to be a lower grade compared to mitral valve disease but we usually also have a left apical systolic murmur, arrhythmia, gallop those are possibilities as well too. So just looking at the signalment of the patient and then their murmur characteristics will help you know again what's the likely underlying type of heart disease. We don't have to know for sure to be able to treat them and

get them out of heart failure. Can definitely hide pimo in a treat the tablets if you get the standard formulation those are flavored tablets as you probably know. They crush really nicely and so you can hide them in food. or if you've got a patient that just won't take, just is not taking anything orally, you can crush it mix it with some water and syringe it in as a slurry. I would not advocate in a patient that's got significant dyspnea trying to give them anything orally. Focus on your supplemental oxygen, your furosemide, then once they stabilize a bit, then you can get the first dose of pimobendan on board.



And just some clarification on who does need to be hospitalized because again I think this decision really is hard to know until you've experienced the cases. So I think probably obvious indication is that if they need ongoing supplemental oxygen. And we talked about what patients need supplemental oxygen. And they need parental medications. Right? If they need to get the furosemide intravenously or IM because they've got a significant amount edema such that it's really affecting their breathing and then they're going to need to be hospitalized. And this is much more common for those left-sided heart failure patients than it is with right. Because again remember the main reason for the hospitalization is the pulmonary edema and patients with right-sided congestive heart failure don't have pulmonary edema. So a lot of right-sided congestive heart failure patients they're managed straight away as an outpatient. Occasionally a left sided congestive heart failure patients if you catch it they've come in, the client maybe-- it's a client that knows they have heart disease. So maybe they've been-- there they're a little more astute or maybe they've been checking breathing rates at home and you catch it early. Then sometimes those patients don't need to be hospitalized. You can give them a dose of furosemide, their breathing rates okay, they're not dyspneic and then they can go home and get started on oral meds after that initial injection of furosemide. But some left side heart failure patients because that pulmonary edema is going to take some time they may need to be hospitalized, And as I mentioned, usually about one to two days. Once the patient is stabilized you might run into the decision, do I need to refer this patient for more management of their congestive heart failure. If that's the case, that might be the case if your facility, if your hospital or clinic doesn't have an ICU with 24 hour care. But again you still will need to do that initial stabilization. To send them without starting the furosemide and getting their, at least their dyspnea resolved. It's not going to be safe to send those patients on their way. Unless

perhaps you have access to an ambulance or some sort of emergency transport. So if you're ever struggling to stabilize a patient that you believe has congestive heart failure or any other respiratory emergency, then definitely want to get on the phone with a specialist. Get some help, get some guidance, and you might need to consider emergency transport for a select few of patients.

In-Hospital Monitoring

VetPrep

- Respiratory rate & effort
  - q30-60min initially
- Mentation
- Heart rate & rhythm
- Blood pressure
- Urination/hydration/body wt
- Appetite
- Renal values & electrolytes
  - CBC & full chem at some point
- Thoracic radiographs

But again, as I mentioned, most of the time when I see patients that are referred to me, they usually been managed by their primary care vet for their congestive heart failure. And I'm seeing them a few days or a week later and they've been managed really beautifully by either their primary care vet at the general practice or maybe on an emergency basis. In hospital monitoring respiratory rate and effort we've talked about. Listed just some other things too. Just really a checklist for when you're making out your treatment sheet you can make sure you have all these other things on there as well too. At some point because we're dealing with a diuretic medication or sometimes maybe these patients haven't been eating really great recently, they are oftentimes older they may have co-morbidities. So it is nice at some point if finances permit to get a full chemistry and also a CBC as well in these patients. But definitely at a minimum because of the diuretic therapy we need to be keeping an eye on their renal values electrolytes especially that potassium.

Discharge & Follow-Up

VetPrep

- Oral medications at home:
  - Furosemide: 2 mg/kg PO BID
  - Pimobendan: 0.25-3 mg/kg PO BID
  - Once appetite & renal values are normal, add ACEi & spironolactone step-wise (~1 week apart)
  - Diet: https://www.veterinarypracticenews.com/understanding-nutrition-in-dogs-with-degenerative-mitral-valve-disease/
- Schedule consultation with cardiologist
  - Determine/confirm type of underlying heart disease
  - Assess for other heart disease
  - Assess for pulmonary hypertension
  - Rule out secondary myocardial diseases (if indicated)
  - Medication adjustments/therapeutic plan
  - Long-term follow-up
  - *CHF patients who receive collaborative care (GP & cardiologist) have improved survival*

What about discharge? I think this is one of the things that I never really think about. Because again most patients that have acute heart failure that a lot of times we as a cardiologist we don't ever see those patients. So it's important to also just be thinking about I've got them I saved them, that's amazing and wonderful. So now what does going home look like? So I've just listed here what they need to go home on. It's really two main drugs, for both oral furosemide and pimobendan and doses are there for you. We don't need to in a hurry get them on the ACE inhibitor or spironolactone. We do need to get them on those long term big picture. They're going to benefit especially from not only clinical signs but even survival. So it's important that we ultimately will get them on all four but that does not need to happen in the short term. Particularly if they haven't been eating well we're going to delay adding more oral medication. So furosemide, pimo to go home, and then I usually add an ACE inhibitor and then spironolactone last. And I don't start doing that until their appetite is normalized and their renal values of normalized. Diet does become important again, in the short term that doesn't really need to be your focus or your worry. But I do have a reference there that talks you through some of the dietary and nutrition nutritional considerations for these patients. And then ultimately another step an important client communication standpoint, is going to be to-- we've gotten them through the crisis now we do need we need a long term plan. These patients even though most of the time it's a terminal condition, there are some exceptions, if they happen to have an underlying heart disease that's reversible. So for example, if it's a nutritionally related DCM Lyme disease of taurine deficiency perhaps, there are some instances where the problem is actually curable or reversible. But in most instances it's not going to be and these patients are going to need long term care and long term management to live and have a good quality of life with their chronic heart failure. So that's where I think consultation with a cardiologist

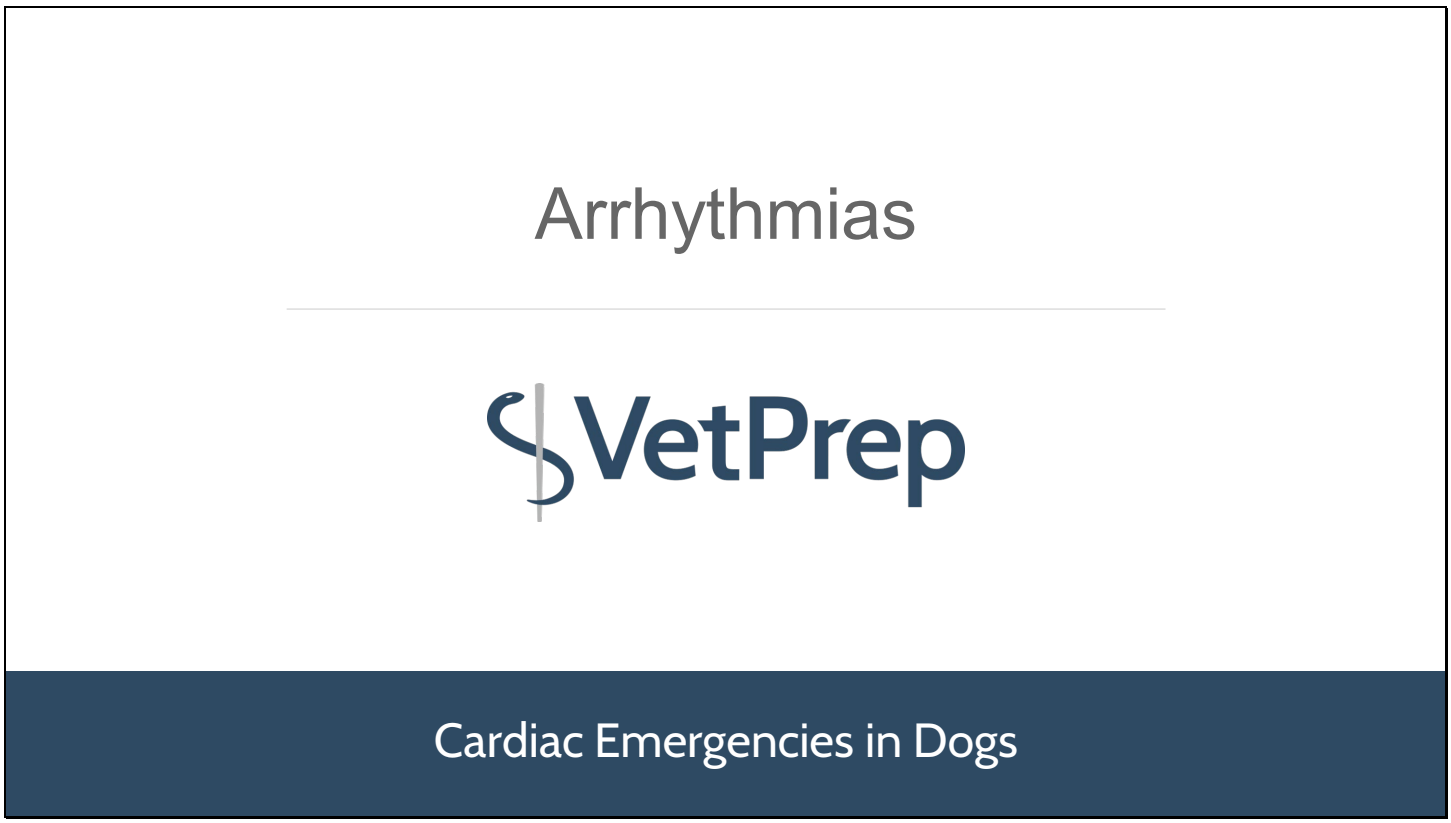
becomes really important. Also with the cardiologist they're going to get that echocardiogram to confirm the underlying heart disease and assess for other heart disease. Also assess for pulmonary hypertension and then the cardiologist, we're going to be able to fine tune tweak medications and make a longer term therapeutic plan. We know that in the short term, the main thing is saving their life and getting that congestion cleared up and getting them feeling better and then we can really help with the bigger picture and longer term plan. And we're always of course, can help too if you're having trouble stabilizing a patient then please pick up the phone and let us help. Because a lot of times we can just think of things or something we've experienced for a more sort of rare case, we can usually help in those situations. There is also some information well known in human cardiology that collaborative care. So patients that have both their primary care veterinarian and are seeing a cardiologist they have improved survival and there's actually some data in dogs as well. So just I think important not only for your knowledge but from a client communication standpoint. If you've got a client that may be is resistant to referral or having trouble understanding referral you know they're going to look at you and think, you've saved his life or you saved her life and why do I need to go anywhere else. And really is just about that long term management, especially as the more advanced stages of heart failure develop and some of those patients will need six, seven, eight different types of medications. And that's where really we can help them continue to have that good quality of life together as a team.

At-Home Monitoring

VetPrep

- Monitoring for progression of CHF:
  - Sleeping/resting respiratory rate (S/RRR), cough, syncope, weakness, lethargy, decreased appetite, abdominal distension
    - S/RRR instructional video: https://youtube.com/watch?v=uEptzj6G-Jk
    - S/RRR >30 should prompt reevaluation (same day)
    - Recurrent/increased cough, syncope, etc. should prompt reevaluation
- Monitoring for adverse effects of medications:
  - Appetite, energy level, vomiting/diarrhea
    - A decrease in appetite and/or energy level should prompt reevaluation
    - The development of vomiting and/or diarrhea should prompt reevaluation

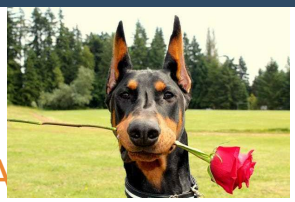
Monitoring at home, really important, just a reminder about patients and patients with heart disease, teaching the clients at discharge to-- how to check their dog's resting and sleeping respiratory rate at home. Got an instructional video link here for you. So you can review if it's been a minute since you've thought about that or if you want to share it with clients as well too. And then of course the other signs and symptoms that we talked about too. So we're doing combination in these patients. And we need really the help of the clients' monitoring for progression of the heart failure, but also now they're on a few new medications. So we also need to monitor for the adverse effects of the medication. And the big thing is going to be GI side effects as well. So if we're seeing any of the recurrence of any of these clinical signs or any new clinical signs develop our clients need to know we need to get them back in and check them. And really usually the same day if any of these signs develop or if they're seeing an increase in that sleeping or resting respiratory rate.



So that takes care of congestive heart failure for the most part now I want to talk a little bit about some of the arrhythmias that we can see in practice. Both in awake patients and then also some anesthetized patients as well too.

Atrial Fibrillation

VetPrep



- AF is common in dogs with severe DCM
- Some small-med breeds with severe DMVD develop A
- Development of AF is usually peracute -> new or recurrent clinical signs -> present to DVM
  - E.g. increased RR, lethargy, hyporexia, owner sees/feels arrhythmia
- Large-giant breeds can develop lone AF
  - No underlying structural heart disease
  - May be asymptomatic or have mild clinical signs (e.g. exercise intolerance)

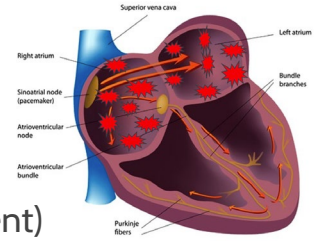
Cardiac Emergencies in Dogs

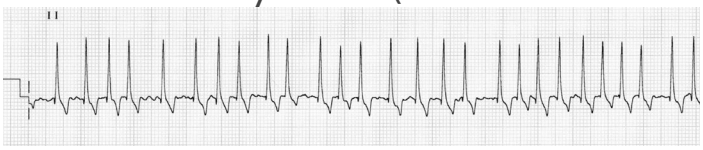
So atrial fibrillation by far and away one of the most common pathologic arrhythmias we're going to see in dogs, particularly we see it quite commonly in dogs that have severe DCM. And some dogs will present in heart failure and may also have atrial fibrillation or another arrhythmia. So I think it's really important to talk about them in unison. Some small and medium breed dogs can also develop atrial fibrillation if they've developed enough atrial dilation or atrial enlargement. Dogs usually are in sinus rhythm one minute and then switch in another second or in AFib. And once dogs go into AFib they usually stay in AFib. So different than people some people they can go in and out of AFib, but for dogs the development of AFib is usually peracute and it's typically going to cause them to have some clinical signs such as-- and then present-- so increase in respiratory rate, lethargy, hyporexia or sometimes even the client may when they're petting them or watching them from across the room may actually be able to see or feel the irregularity. Large and giant breeds can develop lone AFib. So meaning they have no underlying structural heart disease. And those guys can be asymptomatic or they may have more mild clinical signs.

Atrial Fibrillation

VetPrep

- ECG criteria:
  - Irregularly irregular RR intervals
  - Fibrillation waves (variable morphology) instead of P waves
  - QRSs are usually narrow (will be wide with ventricular enlargement)





- Treatment indicated in tachycardic patients:
  - Diltiazem – Ca^{++} ch blocker -> slow AVN conduction -> reduce ventricular rate
    - 1 mg/kg PO TID of standard (prompt-release) formulation
    - Many dogs will need diltiazem + digoxin to control HR – consult w cardiologist

Cardiac Emergencies in Dogs

So I won't spend too much time here but do want to just review those ECG criteria. Remember for AFib, it's usually very obvious or from your auscultation. So this is those sneakers in the dryer, a lot of these patients are also very tachycardic. So when you're hearing an irregularly irregular rhythm particularly then if it's high rate then you definitely want to be thinking of AFib but ultimately the ECG is going to confirm that for us. And if they are tachycardic, and again most are going to be, sometimes in patients with lone AFib they may still have a normal heart rate. But the patients that have DCM or have that mitral valve disease, if they develop AFib they're going to usually be tachycardic. And they're going to need some help controlling their heart rate because of the AFib. So my recommendation for first choice antiarrhythmic when you diagnose AFib in a patient if they're tachycardic is going to be diltiazem. And I recommend a starting dose of 1 mg per kg, PO, it's a TID or Q8 hour of the standard or prompt release formulation. Be a little bit weary out there some human pharmacies will sell extended release. So you just want to make sure you emphasize that it's a standard or prompt release formulation. If you're going to be using at that dose in that frequency. That's always a pretty safe option if you've diagnosed AFib and you know they're tachycardic so you need to start antiarrhythmic diltiazem at that dose is a relatively safe and a good first choice.

Ventricular Premature Complexes & V Tach

VetPrep

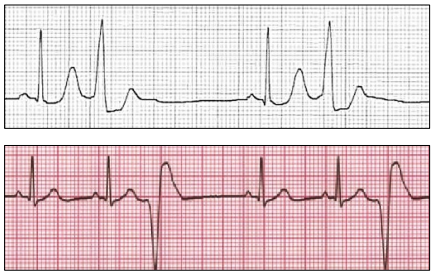
- VPCs are common in dogs with DCM or ARVC
- Some small-med breeds with severe DMVD develop VPCs
- VPCs also common in many non-cardiac conditions
  - GDV, hemoabdomen, sepsis, electrolytes derangements (e.g. hypo- K^+/Mg^+), abdominal tumors/surgery, acidosis, hypoxemia, hypovolemia, anemia, trauma, drugs/toxins
- May be asymptomatic if no V Tach (VT) or if VT is infrequent & VT runs are short

VPCs and V-tach VPCs in particular, you probably have started to see those a lot out there and in especially emergency practice. Certainly our dogs with structural heart disease can have VPCs But keep in mind too a lot of the non-cardiac conditions can cause VPCs as well here. As I've listed so GDV, hemoabdomen. So it's not always going to be from primary heart disease it might be something else. If they're just having VPCs, they're not having ventricular tachycardic, they may be asymptomatic. Or if they're having just very short or infrequent runs of V-tach.

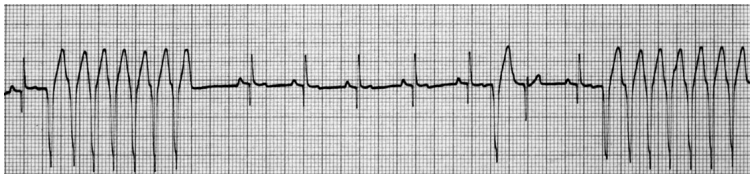
VPCs and VT

VetPrep

- ECG criteria - VPCs
  - Premature QRS that is W I D E (>60 ms)
  - QRS not associated with P wave (ie, AV disassociation)
    - Inconsistent PR intervals



- ECG criteria – VT
  - ≥ 3 consecutive VPCs with an abnormally high rate (ie, tachycardia)

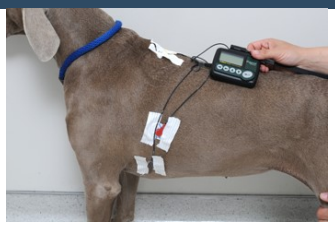


So I've included here the ECG criteria for a little bit of a review for VPC, those premature and wide QRS complexes. And they do not have a P wave that's associated with them. And then if we're seeing three or more consecutive VPCs then that is V-tach.

VPCs and VT

VetPrep

- Indications for anti-arrhythmic therapy
  - VPCs
    - >1000 single VPCs/24hr,
    - Frequent or sustained ventricular bi- or trigeminy,
    - "R-on-T" phenomenon
  - VT
    - Generally, any frequency or duration of VT warrants treatment b/c risk of ventricular fibrillation/sudden death
- Treatment:
  - Lidocaine for frequent or sustained VT: 2 mg/kg IV SLOW over 2-3 mins
  - In asymptomatic patients who have VPCs and/or infrequent VT, best to obtain baseline Holter before tx – consult w cardiologist



Cardiac Emergencies in Dogs

You might be wondering at what point in a patient that has VPCs do I pull the trigger and get him started antiarrhythmic medication. So we do have some criteria there if they've got just VPCs but they've got a lot of them. And what I mean by that is more than 1,000 in a 24 hour period. So usually that's information that we're going to get from a Holter monitor. So a lot of times what we try and do if we know a patient's having VPCs, we're going to need to do some testing to figure out why they're having the VPCS . But then we also will ideally get a baseline Holter monitor or pretreatment Holter monitor in part to assess well how bad is it? How many are they having? But also if we need to start them on therapy that we then have a baseline to compare to see whether or not the medication is really helping. If they're having VT here's a really severe case, you can see really rapid VT in this patient. And then they convert into sinus rhythm. So pretty impressive that's what of course with the patients and sustained VT like that, that's what you hope to see happen as you're giving them the IV antiarrhythmic. But remember with VT that can deteriorate to ventricular fibrillation, and VT can cause sudden death. And of course ventricular fibrillation can cause sudden death. So even if a patient is not maybe in sustained VT but they're having runs of VT we're still going to treat those patients because of those associated risks. And in an emergency situation our go to first line of therapies is going to be lidocaine. So if they're having frequent ventricular tachycardia or sustained VT then we want to get lidocaine on board to them. And it's really important that any time you have to give a patient an intravenous antiarrhythmic, I recommend that always being the doctor, and the patient always having an ECG connected and always being nearby your crash cart with appropriate resuscitation drugs and other therapies. And in general we're always going to give these medications slowly over at least a few minutes with keeping a really close eye on that ECG. As I mentioned if they're not having frequent or sustained VT then we ideally will

try and get that pretreatment Holter monitor. And so that's if you're in a situation where you find VPCs in a patient and they're asymptomatic then that's a good phone call to make if you're not sure. Or maybe they're having a little bit of VT but you just saw it once or twice, you're not sure should I go ahead and start treatment or should I wait. It's a good time to just call and check in with the cardiologist on that if you're ever unsure.

AV Blocks

VetPrep

- Bradyarrhythmias may be due to high vagal tone or due to heart disease
  - To differentiate: perform atropine response test (see next slide) *except in patients with head trauma/increased intracranial pressure (Cushing reflex)*
- First-degree and low-grade 2nd degree are often due to high vagal tone and do not require anti-arrhythmic therapy
- High-grade 2nd degree and 3rd degree ("complete") are often due to AV nodal disease and *do* require anti-arrhythmic therapy
  - ER tx for patients with poor cardiac output: : dopamine, dobutamine, or isoproterenol (or atropine, if partially responsive) may increase the ventricular escape rate
  - Definitive tx: pacemaker

Terms of some of the bradyarrhythmias, so AFib and then VPC and V-tach are kind of the most common tachyarrhythmias you'll see out there in practice, but you may see bradyarrhythmias too. AV blocks are some of the more common ones. First degree and low-grade second degree, those are usually in dogs just from high vagal tone and they don't require antiarrhythmic therapy. High-grade second degree and third degree I'll show you a review of those on the next slide. Those arrhythmias are going to make the patients bradycardic usually cause symptoms and those ultimately are going to need a pacemaker. And I've listed here we've got some drug options in the emergency management while they're waiting to get a pacemaker. Or maybe why the client's waiting to get a-- waiting to make a decision or you're doing some other testing, you can use dopamine as a CRI or dobutamine or isoproterenol in those patients. And that will oftentimes increase their escape rate and help stabilize them until they can get their pacemaker. But the first degree and the low-grade second degree again in those guys it's usually more just about figuring out why they might have high vagal tone.

Atropine Response Test

VetPrep

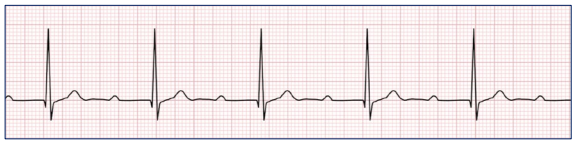
- An atropine response test is used to differentiate bradyarrhythmias caused by *high vagal tone* from bradyarrhythmias caused by heart disease
- Perform & record ECG, administer atropine 0.04 mg/kg IM, wait 20 mins, recheck & record ECG
- Positive response = sinus tachycardia, no pauses, no AV block
  - Bradyarrhythmia is likely due to high vagal tone
- Negative response or partial response = no change in rate/rhythm or rate only mildly increases (e.g. 60bpm to 90 bpm)
  - Bradyarrhythmia is likely due to heart disease

And that leads me to the atropine response test, so this is a way that we can help differentiate. And when we find a bradyarrhythmia in a patient, whether or not it's from high vagal tone from some other condition or maybe even a normal variation say in a very athletic dog or maybe a brachycephalic breed. Or if they've actually got a problem in their conduction system in their heart. So for the atropine response test you're going to get your baseline ECG, you're going to give a dose of atropine. I've listed that dose there, wait about 20 minutes, I like to give it IV and IM. You can give it IV but then you need to check the ECG a little bit sooner. And a positive response if it's from meaning it's high vagal tone it's not a heart problem. Those patients the bradyarrhythmia is going to be abolished and they're going to develop sinus tachycardia. If the bradyarrhythmia does not go away or if it changes but doesn't go away completely, then we'd consider it a negative or a partial response. And that's helpful to know because that tells you it's more likely a conduction system problem and not something else outside of the heart.

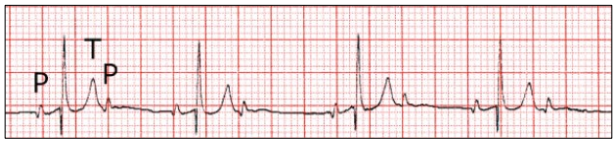
AV Blocks

VetPrep

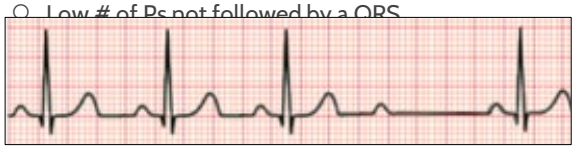
- ECG criteria - 1st degree:
  - PR interval prolonged (>130 ms)
  - PR interval is consistent



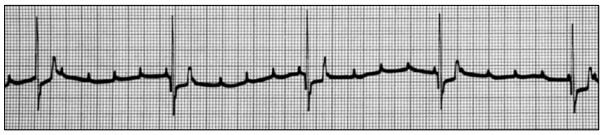
- ECG criteria - high-grade 2nd degree:
  - High # of Ps not followed by a QRS
  - Bradycardia



- ECG criteria - low grade 2nd degree:
  - Low # of Ps not followed by a QRS



- ECG criteria - 3rd degree:
  - All Ps without a QRS, AV disassociation, escape beats
  - Bradycardia



Cardiac Emergencies in Dogs

So review here first degree AV block simply means that we've got prolongation of that PR interval so not actually a block. Names are a little bit misleading. Then we've got second degree, low grade, and high grade. Low grade there's just a few P waves that are not followed by QRS axis, so occasionally that impulse is getting blocked in the AV node. Patients with low-grade second degree AV block, the reason it's usually not too big of a deal is because it's often from high vagal tone and they're going to have a normal heart rate. So they're not going to have any sort of symptoms or issues from it. It's not going to make them feel bad versus high grade second degree AV block. In here or with this arrhythmia a lot of the P waves are being blocked. So there's a high number of P waves that aren't followed by a QRS. So in this example here every other P wave is getting blocked. So there's-- that tells you that it's high grade second degree AV block and these patients they're going to be bradycardic. Their heart rate is going to be low. Third degree AV block nothing's getting through from the sinus node. Instead these patients remember have escape beats and that's what's generating their QRS complexes. And those patients are also going to be bradycardic. So it's these two, the high grade second degree and the third degree that you would want to certainly if they'd had a response to atropine, you can continue giving them some atropine until they can get a pacemaker or you can go ahead and start one of those CRIs to see if you can increase their rate. If they're bradycardic but they're not hypoperfused, so their lactate is OK, their blood pressure is OK, then you can hold and just monitor them really closely until they can get transferred to a cardiologist for the pacemaker.

Anesthetized Patients

VetPrep

- Bradyarrhythmias are more common than tachyarrhythmias & are often drug-related
- Important Qs to ask when a patient becomes bradycardic:
  1. Can anesthestic depth be reduced? (eg, can gas/other be lowered)
     - If yes, lower & reassess HR
  2. Am I doing something to increase vagal tone (eg, pressure on eye or neck)?
     - If yes, temporarily stop & reassess HR
  3. Is the HR stable or decreasing?
     - If HR is continuting to decrease, give atropine or glycopyrrolate
  4. Is the bradycardia causing low blood pressure?
     - If gas/other can't be lowered (or HR doesn't improve), give atropine or glycopyrrolate

Anesthetized patients, let's talk about a couple of important scenarios that you maybe have already encountered in some of your anesthetized patients. So bradyarrhythmias are more common than tachyarrhythmias in our patients under general anesthesia and oftentimes it's the drugs. So any time a patient becomes bradycardic I get a lot of those calls I am called into the OR, sometimes called on the phone and finds me at another practice this is the very same process I would walk through. First is, if they become bradycardic looking at their anesthetic depth because if it's just-- because they're too deep then that's a quick fix and you don't have to give them any new drugs and then deal with that. So checking their anesthetic depth and if it can be reduced then go ahead and do that and then usually within a matter of minutes the heart rate's going to improve if that's all it was. The other thing to remember is ask yourself, am I doing anything to increase vagal tone. So if you're pressing anywhere on their neck around that carotid sinus, eyes, all those things are going to stimulate that vagal response. And so sometimes it's just a matter of repositioning them or stopping what you're doing and being mindful of it The other question is, is the heart rate decreased? So they're bradycardia but is it stable versus are you seeing it go from 100 to 90 to 80. And you're seeing it continue to decrease you might not have time to go through or wait for those responses if you decrease the gas or whatever. So if you can't quickly adjust their depth of anesthesia and you're seeing it gradually decrease then go ahead and give your atropine or your glycopyrrolate. Or if it's that the heart rate is low, you tried doing other things. It's staying low and stable but it's staying low and it's now starting to affect their blood pressure then it's time to go ahead and give atropine or glycopyrrolate.

Anesthetized Patients - Cases

VetPrep

- 14 yr old FS Shih Tzu
- Reason for anesthesia: Extractions/severe periodontal disease
- Anesthetic protocol
  - Pre-anesthetics: methadone
  - Induction: midazolam, alfaxalone
  - Maintenance: isoflurane, fentanyl CRI
  - IV fluids: lactated Ringer's
- ECG prior to induction: NSR
- A few minutes into the procedure…

So a couple of cases I just want to run through, an older Shih Tzu here needed some extractions. I've listed her anesthetic protocol there for you to see. And this is her ECG that develops, so I'll pause it here. So we can see happening. So nice sinuses complexes but then here we got a P wave not followed by a QRS and then it does happen a couple more times we can see. Can see too over here the heart rate when that happened the heart rate dipped a bit but then the heart rate comes back up and it's really infrequent so it kind of self corrects.

Anesthetized Patients - Cases

VetPrep

- ECG diagnosis: second-degree AV block
  - Low-grade or high-grade?
- Cause: most likely cause?
- Treatment: none necessary/monitor

That's one of the most common bradyarrhythmias you're going to see in these patients other than sinus bradycardia is low grade second degree AV block. So if it's just occasionally that those Ps are getting blocked, and the heart rate's not taking a big deep and it's not getting worse you can simply monitor those patients. You don't need to intervene in that scenario. That's most-- that's happening because of one or more of their anesthetic drugs. Again, if you check their anesthetic depth then they're pretty deep and you can lighten them great. But if you can't, that's okay if it's low grade second degree AV block it's perfectly okay as long as that's what it stays as then you don't need to intervene.

Anesthetized Patients - Cases

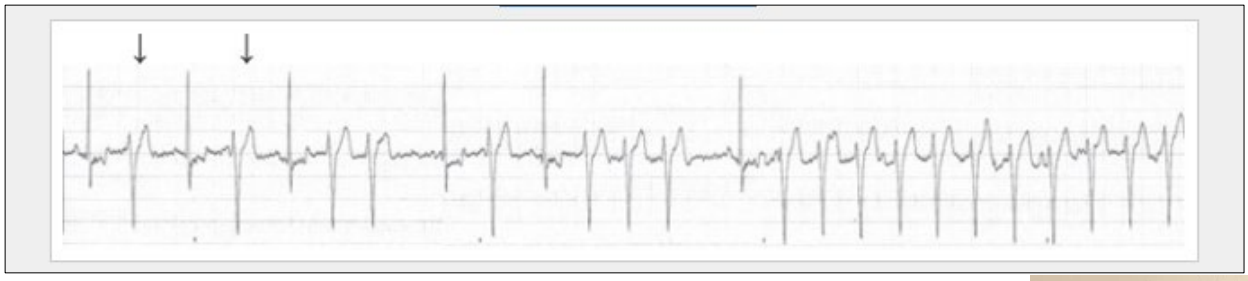
VetPrep

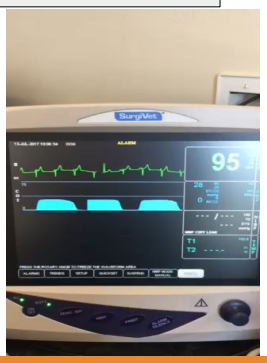
- Patient: 2 yr old FS Rottweiler
- Reason for anesthesia: OHE
- Anesthetic protocol
  - Pre-anesthetics: acepromazine, hydromorphone, glycopyrrolate
  - Induction: propofol, midazolam
  - Maintenance: isoflurane
  - IV fluids: lactated Ringer's
- ECG prior to induction: normal sinus rhythm (NSR)
- Immediately following induction…

Another patient here, so a younger a rottweiler going in for a spay. I've listed her protocol there. Pretty much immediately following induction she developed this arrhythmia here and then here is a video of it.

Anesthetized Patients - Cases

VetPrep





- ECG diagnosis: accelerated idioventricular rhythm (AIVR)
  - Has all the same ECG features as ventricular tachycardia (VT) except it's **not** fast (i.e. not a tachycardia)
  - AIVR rate is usually slightly faster than the sinus rate ...when sinus rate speeds up, AIVR is suppressed

So you can see here, she's got a wide complex rhythm. So they look a lot different these complexes here look a lot different than these more narrow sinus complexes. And then if we look at the video here we see again wide complexes but then it begins to alternate with these more narrow normal looking complexes. And when we check the rate of this ventricular rhythm it's not fast, so it's not a tachycardia. And so this patient actually has what's called accelerated idioventricular rhythm. It looks the same as ventricular tachycardia, it's just not fast. So I like mentioning AIVR because it's a good reminder that we can't always rely on the monitor to tell us the patient's heart rate. The monitors are good but they can sometimes, depending on the sensitivity level, they can over or under-count heartbeats. And so one of my best I think recommendations that I can make is when you've got any patient that's having an arrhythmia under anesthesia or not, verify their heart rate. Actually, you or-- if you're scrubbed in someone else is in the room check their actual heart rate. Because sometimes the monitor is not necessarily right and it's important because IVR doesn't need lidocaine. But if this was ventricular tachycardia the patient would need lidocaine.

Anesthetized Patients - Cases

VetPrep

- AIVR
  - Potential causes: abdominal tumors (e.g. splenic, hepatic), gastric dilatation-volvulus (GDV), hypokalemia, acidosis, anemia, hypoxemia, trauma, sepsis, hypovolemia, drugs/toxins
  - AIVR in small animals is usually *not* due to underlying heart disease
  - Cause in this case: Suspected to be due to propofol b/c all other causes were ruled out
  - Treatment: address underlying cause. Anti-arrhythmic therapy not indicated.

Usually non or extra-cardiac disease that causes AIVR in dogs is generally a stable rhythm so you need to look for these underlying causes and address those. This patient we rule all of those things out, had a complete workup, and based on the timing, how quickly it happened after the propofol and then at about the time that the propofol would be sort of wearing off if it resolved. So that's what we suspected in this case, but we first had to go through and rule out the other conditions first.

Pericardial Effusion and Cardiac Tamponade

VetPrep

Cardiac Emergencies in Dogs

Okay our last and final cardiac emergency we're going to talk about today and a really important one. Not particularly common but life-threatening and pretty straightforward to manage. So I want to be sure we go through it.

Causes

VetPrep

- Acquired most common
  - **Neoplastic**
    - **Hemangiosarcoma**
    - **Chemodectoma**
  - **Idiopathic pericarditis**
  - Toxin/coagulopathy (e.g. rodenticide)
    Trauma
  - Infectious
  - Metabolic
  - CHF (usually small-volume)
- Hemorrhagic/serosanguinous fluid most common

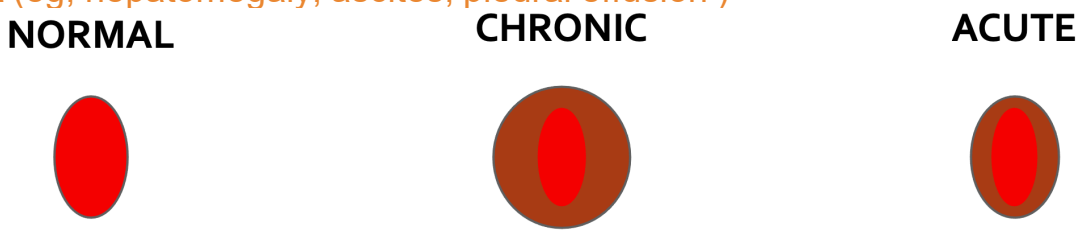
So pericardial effusion, that's the most common pericardial disorder in dogs, and there are a couple of common causes to be aware of. So cancer can certainly cause pericardial effusion but dogs can also get an idiopathic pericarditis that can cause pericardial effusion. I've listed some other causes as well, if they've got a coagulopathy, if they've had trauma, some infectious diseases, sometimes some kind of bizarre metabolic manifestations, and right sided congestive heart failure in some dogs will cause a little bit of pericardial effusion. But it's usually small volume and we don't need to really kind of think about it in the same way of these other causes. The most common fluid type in dogs is going to be a hemorrhagic or serosanguinous fluid. So it can be pretty alarming if you haven't done your first pericardiocentesis yet. A lot of times it comes out looking like blood. And of course you got a needle or catheter close to the heart. So you need to be mindful but I say that so that you're prepared that that's actually the most common type of fluid that we see in dogs with pericardial effusion.

Slide 41

Cardiac Tamponade

VetPrep

- When intrapericardial pressure equals or exceeds intracardiac pressure, diastolic filling decreases (then SV decreases & then CO) = **cardiac tamponade**
- As CO falls, systemic venous pressure rises & congestion and effusion may result (eg, hepatomegaly, ascites, pleural effusion)



Cardiac Emergencies in Dogs

It becomes a life-threatening emergency when there is a change in that pericardial pressure such that it's higher than the intracardiac pressure. So when that happens the heart can no longer fill normally and that's cardiac tamponade. So pericardial effusion if it's not causing cardiac tamponade it's still an urgent situation, but the minute it transitions to causing cardiac tamponade it becomes a life threatening emergency and it's very quick and relatively straightforward to resolve it. But you got to know that that's what you're dealing with. So what I want to point out here a reminder about the pericardium is that, it does have some ability to stretch and so if a fluid or effusion is happening slowly it can get to be a large volume without causing tamponade or it can take much more volume before it will cause tamponade. Because that pericardium has stretched and that's allowed for a period of time some normalization of that entire pericardial pressure. Versus a patient that has an acute effusion there's not been time for that pericardium to stretch and even a small amount of fluid can cause tamponade.

History & Clinical Signs

VetPrep

- Dogs with acute PE commonly present on an emergency basis for collapse or weakness
  - Vomiting w/n 24-48 hrs of presentation is also commonly reported
- Dogs with chronic PE may present for vague signs such as lethargy, exercise intolerance, decreased appetite, *abdominal distension*
  - Peritoneal effusion is usually ascites due to systemic venous congestion secondary to pericardial effusion, but other causes (eg, hemoabdomen) must also be considered
- Respiratory signs such as coughing, excessive panting, tachypnea, & dyspnea are also possible

If dogs have acutely developed effusion in their pericardium such as a tumor that's bled, for example they're usually going to present on an emergency basis for collapse or weakness oftentimes with also recent history of some vomiting. If it's been more chronic the volume of fluid has been slowly building up, slowly accumulating then they will usually present with some more vague signs such as lethargy, maybe some exercise in tolerance, decreased appetite and very commonly they're going to have abdominal distention because of that back up, that the fluid in the pericardium is preventing the heart from filling well. That means that everything is getting backed up on that systemic side. And so those patients can then start to push out fluid into their peritoneal space and sometimes into their pleural space as well too. Some other signs there are two respiratory signs are possible, especially if they do have pleural effusion or if they've got a lot of pericardial effusion. And it's caused their pericardial sac to be very big and press on surrounding structures.

Physical Exam

VetPrep

- Heart sounds are decreased in intensity ("muffled") or absent if there is a large volume of pericardial effusion
  - Dogs with acute PE may only have a small volume of fluid; therefore, heart sounds may be only mildly decreased (or may sound normal)
  - Lung sounds may be decreased if moderate or severe *pleural* effusion is also present
- High-normal heart rate (or tachycardia) & weak femoral pulses are common
- Jugular venous distension, hepatomegaly & peritoneal effusion are common
  - Must rule out cardiac tamponade in any dog with unexplained peritoneal effusion
- Tachypnea, dyspnea, excessive panting may be present
- If tamponade is profound, arterial hypotension & hypoperfusion will be severe
  - Weakness (reluctance to stand/walk), depressed or dull mentation, comatose, cardiopulmonary arrest

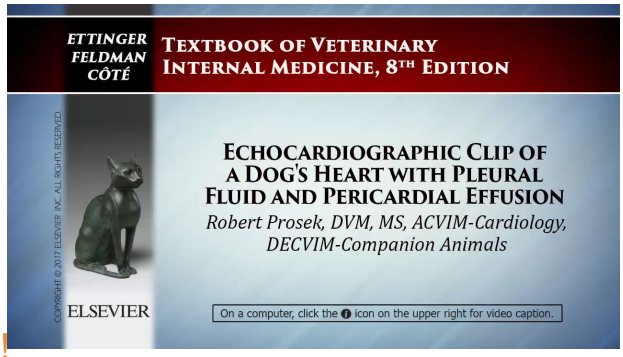
Cardiac Emergencies in Dogs

Remember that the big kind of key here on physical exam is decreased intensity of their heart sounds or kind of muffledness to their heart sounds. If they've got a lot of pericardial effusion you might not be able to hear their heart at all. Patients with pericardial effusion are also particularly cardiac tamponade are usually going to have a high normal heart rate and may have weak femoral pulses. You may also find some distension of their jugular veins, hepatomegaly, and the peritoneal effusion is for those reasons that we just talked about. If their tamponade is profound these guys can present down and out because of hypertension and hypoperfusion. So usually from history and then our physical exam findings we can have a strong index of suspicion that we're dealing with pericardial effusion. And then we can-- we're usually most often if you've got access to ultrasound, which a lot of practices do. We can do a quick ultrasound scan to confirm that is in fact what's going on. You maybe know how to identify the effusion but you might be wondering, well, how do I know if they've got tamponade. Well one, remember clinically they're going to have increased heart rate, decreased femoral pulses, their blood pressure may be low, usually their lactate is going to be high So you can get any of those clues on physical exam combined with decreased or absent heart sounds should tell you this is a patient that has pericardial effusion like this, tamponade.

Diagnostics

VetPrep

- PE is usually strongly suspected based on history & physical exam findings
- Thoracic FAST confirms the diagnosis
  - Right atrial wall collapsing = cardiac tamponade



- If cardiac tamponade is present, proceed to emergency pericardiocentesis – do not delay!
- Some patients with cardiac tamponade may "look pretty good" (especially labs, pits, etc.) – do not be fooled…cardiac tamponade is a life-threatening condition and, therefore, is *always an emergency*

But we can also confirm it by looking with ultrasound at the wall of their right atrium. So that's what my mouse is surrounding here. So the left atrium is over here on your left side. And you can see here. That's how the atrium should look. But you can hopefully see this sort of flailing or flapping wall. That's the ultrasound confirmation of cardiac tamponade. So if a patient either based on your physical exam and other diagnostics, and certainly if you're able to look with ultrasound, has tamponade they need to get an immediate emergency pericardiocentesis. Or you need to perform an emergency pericardiocentesis right away. It's completely live threatening these guys can go in a matter of seconds to minutes from walking around and looking pretty good to arresting. So I can't emphasize that enough.

Slide 45

Treatment

VetPrep

- Pericardiocentesis
  - Place an IV catheter & collect samples for laboratory testing
  - Connect & monitor ECG
  - Aseptically prepare centesis site (usually right side @ 4-5 ICS)
  - Administer local anesthetic (eg, lidocaine), +/- sedative (eg, butorphanol)
  - 14-16G 6in (large dogs) over-the-needle catheter inserted (cranial to rib) through the parietal pericardium (may get a flash or may need to aspirate w/ syringe)
  - Catheter is advanced off the needle further into the pericardial cavity, stylet removed, extension tubing (w/ 3-way stopcock & syringe) connected to catheter
  - Place small sample into a no-additive tube & observe for clotting
  - Remove as much pericardial fluid as possible & collect samples for analysis +/- culture (EDTA & no-additive tubes)
  - https://www.youtube.com/watch?v=gVy7uCKFB7Y (UMN- Dr. L. Powell, DACVECC)
- IV fluid therapy & monitoring (arrhythmias, recurrent PE)

Cardiac Emergencies in Dogs

And I also like to talk about this disease because in that school it's not routinely taught how to do a pericardiocentesis. And so I've outlined that procedure for you. But also here Dr. Powell from University of Minnesota - it's a really, really practical and excellent in my opinion video that walks you through. And she's actually going to begin with a cadaver. She'll walk you through those different steps. And then I have the steps written out here. So I hope you will take an opportunity. You know this is a procedure that because of its emergency nature really does need to be performed on the front line. And so it's important to have some comfort level and certainly watching how to do it and being comfortable with those steps is the first step in doing that. During an ECG we want to make sure they're hooked up to a-- during the procedure we want to monitor them for arrhythmias, we're usually also going to start some IV fluid therapy as well.

Next Steps

VetPrep

- Coagulation profile – ideally, performed prior to pericardiocentesis to r/o coagulopathy, but this is not always possible (dependent on patient stability)
- Minimum database (CBC, complete biochemical profile, UA)
- Thoracic radiographs - ideally, performed after effusions have been removed so lungs can be thoroughly evaluated for metastatic disease
- Fluid analysis & cytology of effusion(s)
  - + culture if an infectious cause is suspected
- Echocardiogram – primarily to look for a tumor
- Staging with abdominal ultrasound or CT (in cases with a suspected or confirmed neoplastic etiology)

Next steps. So again, you've saved them, wonderful you've got them tapped. They're feeling much better. I mean these dogs usually just go from feeling pretty terrible to sometimes, I mean, I've literally had them come in on a gurney and 15, 20 minutes later they're chowing down and wagging their tail, I mean it's incredible. But we do need to take some additional steps once we get them through that crisis and do some diagnostics to figure out, OK, what's causing the pericardial effusion. Remember in dogs we said that cancer is the most common cause. But the second most common cause is idiopathic pericarditis and that actually has a good prognosis. So we want to definitely get a minimum database on these patients. And again this is going to be post stabilization, thoracic radiographs, we want to get that fluid analyzed. And we're going to need to get them an echocardiogram as well to look for a tumor and then if we're suspicious of is that they do have a tumor then we're going to need to do some staging to look for metastasis or a primary tumor.

Client Communication

VetPrep

- Prognosis dependent on cause of PE
  - Poor for hemangiosarcoma (MST with chemo & SX = 6.3 months)
  - Fair for chemodectoma (MST with pericardiectomy = 730 days)
  - Good for idiopathic pericarditis (1/2 of cases resolve after pericardiocentesis)
- Many of the initial diagnostics can be done at GP
  - Results will dictate referral (e.g. cardiologist vs. oncologist)
- PE can recur at any time, so at-home monitoring is crucial

Prognosis here because this so often is an emergency situation. So I think having a handle on the client communication is key. First and foremost, if, they've got to get the pericardiocentesis, that's step one. I mean if the clients want to continue. Right but a lot of clients are going to need some more information about what this could be in the prognosis before they make that decision. And so again I want you to just have the most important points. And I try and encourage clients if they can, if it's within their financial means to go ahead and let-- we've got to get them through that emergency crisis. Let's get this pericardiocentesis we don't have much time let's get that done, not very expensive won't take very long at all. And then once we know that we've got them out of the emergency then we can have a little bit more time take a breath and talk about some of the prognosis and kind of where we go from here. And if you're going to need to talk about really these three possibilities. Because on an emergency situation you're may not be able to necessarily with the diagnostics you have available know for sure. So it's good to just talk about these three hemangiosarcoma, chemodectoma, and then idiopathic. And you can see here the range in prognosis is pretty big. So poor for hemangiosarcoma, so median survival time with chemotherapy and surgical removal of that tumor remembers is only about six months. Versus pericarditis half of those cases resolved just after the pericardiocentesis and the other half are usually cured with surgery to remove the pericardium. So I think it's important to give a representative or have a representative discussion with the clients. It's not always cancer. So I just want to mention that and highlight that. And as an emergency doctor on the frontline it's impossible to have all of the answers. But I find that at least if we can be complete in the possibilities and tell them worst case, best case, that's going to be able to really help most clients make the decision that's best for their pet and for their family. Most of those initial diagnostics of these guys needs of the clients decide to proceed can be done at your

practice, the thoracic radiographs, the blood work, et cetera. And who knows you might from those-- you might find pulmonary metastasis. And in that point you're going to know they really are going to need as a next step if they want to continue to see an oncologist. Or maybe you nothing is showing up at his cancer. And so next step is really for them to get that echocardiogram to see if we're dealing with a cardiac tumor. So those initial results from testing will help you decide or know which cardio-- or which specialist they should head to. Pulmonary, or excuse me pericardial effusion, just remember that whether it's from a tumor or it's idiopathic, it can recur at any time. And that's really important client communication as well too. I definitely have had clients that because of the fear-- if their dog got very, very sick from the pericardial effusion, because the fear of them getting that sick again was just too much for them to bear, definitely some clients may elect to euthanasia at that time. So it's important because it can really-- if they don't know they might just think, oh they're fixed. And not realize we have taken care of it for right now but the effusion can reoccur. That can influence the family's decision so important to mention.

I really thank you. It's been a pleasure and I hope to see you in my other lecture on heart murmurs. Have a wonderful rest of your day. Thank you so much.

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