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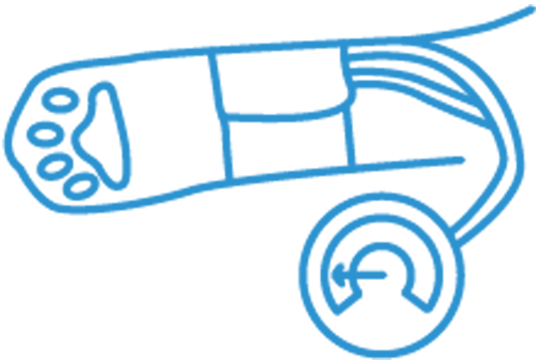

Feline Hypotension

DRIP 3

September 14, 2021

Instructor: Dr. Christopher G. Byers, DVM, DACVECC, DACVIM

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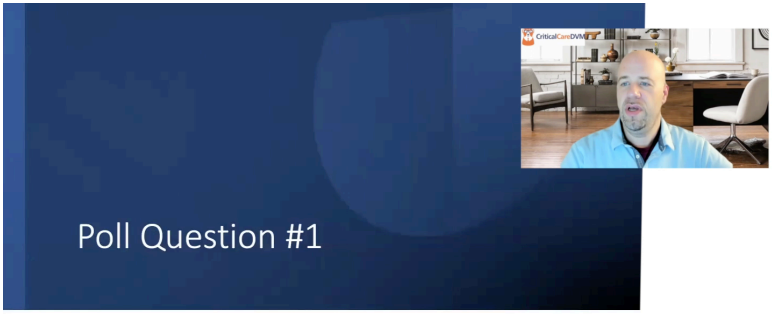
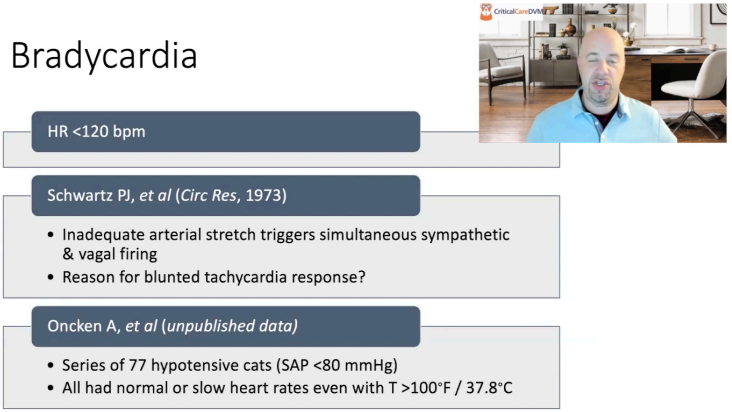


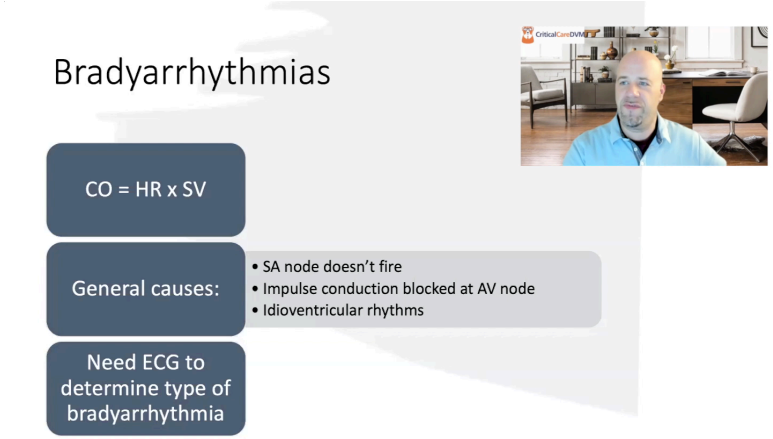
65% of you said true and 35% of you said false. And I'm glad I asked this question because the answer is actually "false". And this is one of the things that make cats special. They truly do adhere to the statement that cats are not small dogs. So when you and I are presented, for example, in hypovolemic shock, or a dog is presented in hypovolemic shock, our sympathetic system fires strongly. And that's why we have initially a compensatory tachycardia.

That doesn't often happen in our feline friends because we know that while their sympathetic trunk is firing, their sympathetic system is just fine. They have concurrent firing of their parasympathetic system. So the vagal tone, it offsets the sympathetic tone. So a cat in shock often is presented with a normal heart rate, or potentially even a slow one. So bradycardia is very common. It is not common for compensatory tachycardia to manifest in our feline patients.

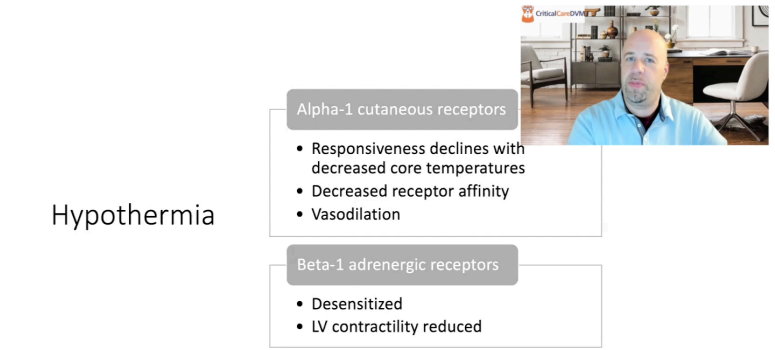


So how do we define bradycardia in our feline friends? Traditionally, less than 120 beats per minute. So Achero's heart rate of 118 beats per minute technically classifies as bradycardia. And again, we know from research way back in the late 1900s-- there it is-- that we have concurrent or simultaneous sympathetic and vagal firing. We also know that Dr. Andrea Anqua, another colleague in the critical care college, had a series of almost 80 cats, all hypertensive, that never, ever were tachycardiac, regardless of their shock state.

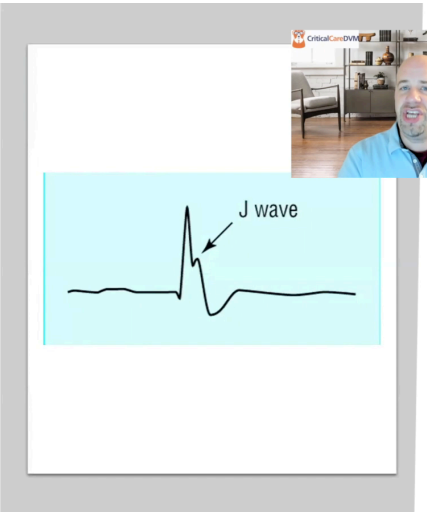
So don't let a lack of tachycardia make you feel or make you believe that your cat isn't in shock. It probably is.



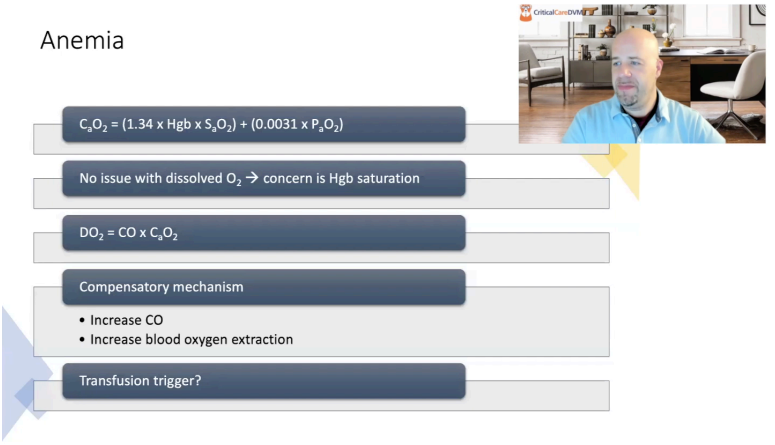
Bradyarrhythmias also contribute potentially to altered cardiac output. So remember cardiac output is heart rate times stroke volume. So maybe the sinoatrial node isn't functioning properly, or impulses are getting blocked as they go through the atrioventricular node, or the patient develops ventricular rhythms. Obviously we need some advanced monitoring in the form of electrocardiography, EKG, ECG to figure that out. And that's straightforward enough.



I really want you to appreciate the impact of hypothermia. We need to keep these patients warm, and we'll review how to do that. But the fact is, a cold body temperature really angers the body. So we have Alpha 1 receptors in the skin that when they're not being stimulated, they lose function, resulting in vasodilation. So your systemic vascular resistance is affected. And the Beta 1 adrenergic receptors in the heart are also unhappy when they're cold. They become desensitized. And so when catecholamines bind to them, they don't do as much, because they're not as sensitive. And so that left ventricle, for example, doesn't contract with as much force. So hypothermia has a negative inotropic effect.



It can also affect rhythms. And so this is called a J wave, or an Osborn wave. And if you see this as you're monitoring your patient, whether they are being monitored in your ICU or monitored under anesthesia, if you see a J wave or this Osborn wave you need to have a big red flag. Go up and say, my patient is cold, I need to check their temperature to confirm that. Other dysrhythmias associated with severe hypothermia are Atrial Fibrillation or AF, and Ventricular Fibrillation or VF.

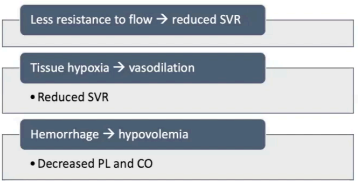


Now, anemia is something to consider. The top equation is called your oxygen content equation. So you can see that the content of oxygen in the blood is influenced majorly by hemoglobin, and very minorly by the amount of oxygen dissolved in the blood. OK. So really when we're talking about anemia from the standpoint of affecting hypertension, not really care about the amount of oxygen dissolved in the blood because of how minor it is. We want to focus on hemoglobin saturation. But I'm always asked, well, when do I transfuse?

There is no specific hematocrit or packed cell volume at which you should absolutely transfuse. It's honestly very different for each patient. So in general, I look for clinical signs. I'm looking for the patient to tell me that they need blood. Lethargy, tachycardia, tachypnea. And there is a concept called Early Goal Directed Therapy that says really strive to keep the packed cell volume greater than 25%, but that's fallen out of favor.

But I want to mention it in case it's terminology with which you're familiar.

Now initially with anemia, you'll get an increase in cardiac output. The body is going to try to take as much oxygen from that hemoglobin as possible to have it consumed by the body's tissues.



Anemia

But with time, that tissue bed, all of the tissue beds are going to become hypoxic. And so those vascular beds are going to vasodilate. Systemic vascular resistance is going to drop. When blood is anemic, it loses its laminar flow. And when you lose laminar flow, there's less resistance, which means the systemic vascular resistance is also low. It's obvious to say that hemorrhage, that type of cause of anemia, can lead to hypovolemia, which is, in a sense, going to lead to a reduction in preload, and ultimately, a reduction in cardiac output.

Low iCa^{2+}

MLCK induces vascular smooth muscle contraction

- MLCK activated by Ca^{2+}

Excitation-Contraction coupling

Common causes of low iCa^{2+}

- CKD
- Acute pancreatitis
- Eclampsia

We always want to make sure that our hypertensive patients have adequate calcium. Low ionized calcium specifically can lead to hypotension, because we need to activate Myosin Light-Chain Kinase or MLCK. How is MLCK activated? From calcium. OK. So if we don't have enough calcium, that excitation coupling that we all learned about probably in first or second year of veterinary school doesn't happen, or it happens less efficiently. And so if you are presented with a cat with chronic kidney disease, acute pancreatitis, or puerperal tetany, eclampsia, that is sick and hypertensive, you need to check their calcium and address hypocalcemia if documented.

Acidosis is never a good thing. The heart doesn't like it, specifically with severe acidosis. So pH is less than 7.2. With acidosis, catecholamine receptors throughout the body become less sensitive. They are down-regulated. There are post-transcriptional defects that happen. So the catecholamines, practically speaking, don't do their job or don't do them as well as they could, if acidemia wasn't present. And peripheral arteriolar vascular beds dilate, negatively affecting systemic vascular resistance.