



Anemia in Cats and Dogs

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version 1

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An Approach to Anemia in Cats & Dogs

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Disclosures

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Hi, everybody. My name is Chris Byers. Let's dive right in. These are my disclosures, they haven't changed, but it's always fair to share them.



What are we going to talk about tonight, specifically as it relates to anemia?

Well, we're going to briefly review erythropoiesis. Then we'll spend some time talking about regenerative and non-regenerative anemia, the various differentials, and how we can utilize red blood cell indices to give us a little more information. We'll spend a little time reviewing some of the diagnostic tests one may need to consider for anemic patients, and then we'll spend a good amount of time going through several cases of anemia. It should be a action packed 45, 50 minutes. But of course, you guys know I love questions.

Erythropoiesis	Prorubric
	Degenerating
Rubriblast	
Prorubricyte	Rubricyte
Rubricyte	Rubribla
Metarubricyte	Diffusely basophilic enthrocyte
Reticulocyte (polychromatophil)	10 µm
Erythrocyte	

mage Credit: Dr. RA Bergman

When we talk about erythropoiesis, we have a lot of cells that we've probably forgotten how to identify from vet school. Let's briefly refresh our collective memories. The most immature is called a rubriblast. Sometimes in various texts, it will be called a proerythroblast or a hernocytoblast, or a myeloblast. This is a stem cell of the erythroid series that's characterized by this very large, rounded nucleus with basophilic cytoplasm.

As we mature, the next step is a prorubricyte or a basophilic erythroblast. That's the other name. This develops from the rubriblast, it's smaller than that stem cell, and the nucleus has chromatin that's coarser. We still have a basophilic cytoplasm that's chocked full of RNA. And the basophilia actually obscures a lot of the hemoglobin content within this type of cell. And then as we take the next step, these prorubricytes will undergo mitotic division to give rise to the next cell, which is a rubricyte, also called a polychromatophilic erythroblast.

This is the product of mitotic division. The nuclear chromatin is more compact, the cell starts to get smaller. Then we have our metarubricytes or normoblast. More mitotic division has happened, the nucleus becomes smaller and pyknotic, the cytoplasm becomes pretty distinctly acidophilic, and that's because the hemoglobin content within that cytoplasm just continues to increase. And then we have our reticulocytes that everybody is very comfortable identifying, and of course, our mature erythrocytes that are non-nucleated with that central pallor.

Cobalamin & Folate



Enzymes required for DNA synthesis, hematopoiesis, & neuron myelination

Methionine synthase & methylmalonyl CoA mutase → cobalamin in a cofactor
Thymidylate synthase → folate required for adequate activity

Cobalamin or folate deficiency induces megaloblastic anemia in humans

Defective DNA synthesis, ineffective hematopoiesis, erythrocyte precursor maturation arrest
Stanley *et al* (*J Vet Intern Med*, 2019) showed association is not routinely present in dogs

Macrocytosis is due to asynchronous development of RBC cytoplasma & nucleus

Increased MCV and RDW may precede anemia

Now, you may be familiar with some internal medicine specialists or some critical care specialists recommending cobalamin supplementation or folate supplementation for some of your anemic patients with whom you've partnered with the specialist. And I want to make sure that everybody is on the same page about why we consider cobalamin supplementation or Folate supplementation for some of these anemic patients. It's because both vitamin B12, cobalamin and vitamin B9 folate are required in some pretty essential reactions.

For example, cobalamin is a cofactor for two specific enzymes. And Folate is required for another enzyme to work. Essentially, they are required to contribute to adequate DNA synthesis and adequate hematopoiesis, and even myelination. In people, in you and me, when we have cobalamin and/or folate deficiencies, we often will develop something called a megaloblastic anemia.

Essentially, this happens because these enzyme systems don't function properly because there's not enough of the necessary cobalamin or the necessary Folate. They develop a macrocytosis, the erythrocytes become bigger. And that's because there is desynchroning between nuclear development and cytoplasmic development. And that macrocytosis and that resultant difference in red cell distribution width or RDW often precedes the anemia. This is just an important point to always make sure you're looking at red blood cell indices, not just red blood cell count, hemoglobin, or hematocrit on your complete blood count readout. We'll talk more about that as we go through our time together.



And you know, I like to start at the beginning. And in this case, the beginning is simply defining what anemia is. The most basic definition is there's a reduction in red blood cell mass. You and I are used to documenting anemia through measuring packed cell volume or looking at hemoglobin or looking at hematocrit on complete blood cell counts.

But we need to always dig a little bit deeper when we're talking about our patients' anemia. And the first step after confirming a patient has anemia is determining whether or not we have a regenerative response or a nonregenerative response.



Poll Question #1



TRUE OR FALSE:

Hemorrhage is always associated with a regenerative anemia

This is a true or false question. Hemorrhage is always associated with a regenerative anemia.