Thyroid Diseases in Pets I:

A Special Interview with Dr. Jean Dodds

By Dr. Karen Becker

KB: Dr. Karen Becker

JD: Dr. Jean Dodds

KB: Hi, this is Dr. Karen Becker. Today I have a very special guest, Dr. Jean Dodds. She doesn't need much introduction because she really is the world's foremost expert on thyroid diseases in pets. Jean, thank you so much for joining me. I really appreciate you being here.

JD: Thank you, Karen.

KB: Question number one (we'll just get started right away), what are the classical clinical signs of canine hypothyroidism and when do they occur, Jean?

JD: That's a good question. Classically, what we look for is a fat, lazy animal with some chronic skin disease that doesn't like the cold. The problem is that those classical signs don't show up until at least 70 percent of the thyroid gland has been damaged or impaired.

KB: And if we are going to include our feline friends in this particular talk, let's contrast that to cats with the symptoms of feline hyperthyroidism. What are the classical signs of feline hyperthyroidism?

JD: Feline hyperthyroidism is a little more dramatic to see because it occurs in older cats primarily where they get ravenously hungry, they lose weight, they howl all the time and pace around. I mean, they act like they're wired. They don't become a sociable, companion animal like they had been earlier in life.

KB: Are basal thyroid levels the same for animals over the age and breed spectrum? What differentiations do you see between different ages and breeds when it comes to identifying thyroid disease?

JD: Well, part of the problem… And Karen, I must address this for a second. This is a big frustration for me. After 20 years of focusing on the thyroid gland as a major master gland affecting the blood and the immune system, the issues that I had studied for so many years, what happens is all of the laboratory reference ranges are the same. There's a reference range for every dog – no matter how old or what breed –and every cat. Now, most cats are roughly the same size, except for wild cats. But dogs vary – from the tiny toy teacup poodle to an English mastiff or a Saint Bernard – and obviously, they don't have the same metabolism.

The problem for us, as clinical veterinarians, is: how do we diagnose the disease in the dog earlier before the end stage signs and 70 percent of the gland has been damaged or more than that? And if we do that, how do we determine what the optimum or minimum ranges should be for a toy breed that's much more active and has a rapid metabolism versus a large, giant breed that's much more sedate. I mean, would you want a Newfoundland with the energy of a Yorkie? Oh, my god.

KB: That's very, very true. Jean, why is it that most traditional testing labs are lumping all dogs into the category of "dog." How did this ever get started?

JD: I guess we don't know that. I think maybe veterinary medicine… First of all, we adapted assays that were used in humans…

KB: Yes.

JD: Because, as you know, veterinary medicine is sort of an orphan specialty in a sense that we don't have reagents that necessarily are specific for the dog, the cat, the horse, or whatever. We adapt what we can from the human reagents. That's problem number one.

Problem number two is how we identify normal animals. Other than the fact that they look healthy and they have a physical exam, we put them in the "normal animal" category. But we know already from published science back in the '70s, Karen, which we've forgotten about, that age affects thyroid levels as well. Because young animals are growing and they have a more active metabolic need. Old animals are not growing so therefore, their metabolic demands for the thyroid are going to be lessened. Even then, in the '70s, we knew there was an age and breed (breed type not necessarily breed specific) difference.

And there were people from Cornell University and other veterinary school who were starting to look at not just the thyroid but in other parameters – blood serum chemistry parameters and complete blood count (CBC) parameters – and seeing that they were different.

KB: Yes.

JD: And we know that. Puppies have different blood counts than adult dogs do. We know that.

KB: Jean, how does one truly, effectively screen for thyroid dysfunction then?

JD: Well, we have to get away from the following: veterinarians will do a proper physical exam – by the way, I love my profession as you know – and they'll do what's called a comprehensive wellness profile if the animal's not coming to them with an obvious illness. That will usually include a complete blood count and blood serum chemistry (hopefully, a comprehensive chemistry, the one that includes an amylase and lipase as well to look at the pancreas), and will add a urinalysis and a total thyroxine (T4).

Oh, my god, if we could just get rid of that total T4, we probably will be a lot better off. We don't want to ignore the thyroid. Never. But a total T4 can be totally misleading. Total and totally misleading. After all, it's affected by non-thyroidal illness; it's affected by certain drugs like steroids at high doses, even monitored doses; and it convulses like phenobarbital and sulfonamides – sulfonamide antibiotics, sulfonamide anticonvulsants like Zonisamide, and sulfonamide non-steroidal anti-inflammatories like Parecoxib.

And also too much iodine. We have this whole issue now about too little iodine promoting thyroid dysfunction, and too much iodine causing hyperthyroidism in older cats and causing thyroiditis or predisposing to thyroiditis in the dog.

We have this problem with doing a total T4. Veterinarians rely upon that. Eight times out of ten, the laboratory result will come back to them, whether they do it in-house or outhouse (I don't mean that derogatorily), with a total T4 that's low. The print out from the laboratory or the machine will say, "Please consider adding on a free T4," to make more definition of what's going on.

We're assuming the animals are hypothyroid. Some veterinarians treat them based on just this total T4 when it may or may not be appropriate. So they add on a free T4, that looks better. But what if they have autoimmune thyroid disease?

KB: Right.

JD: They're not measuring the autoantibody. They're still not getting an accurate diagnosis.

KB: Really, what test should be included in the complete thyroid panel for dogs?

JD: Yes. We should measure a complete thyroid antibody profile. Even at the beginning, up front, the veterinarian needs to explain carefully to the client, "It will be more expensive, but what need to rule this in or out, up front, so we know what we can do with the case from then on." Maybe the veterinarian will say, "Well, the dog doesn't look hypothyroid. It's not overweight. It doesn't have bad skin." Well, maybe it is overweight. Maybe the client is not feeding the dog too much and yet, it's an easy keeper.

KB: Yup.

JD: It's gaining weight and gaining weight. We're skeptical, of course. We say, "Obviously, the dog's getting food from someone else in the household," and yet they're not. That could be one scenario.

The other scenario would be a sudden change in behavior. The animal who is very passive, happy, and outgoing becomes withdrawn, submissive, phobic suddenly to noises or objects they don't see, suddenly take a dislike to a particular person that visits who they've known before, or, God forbid, can become unprovokingly aggressive, and just attack erratically out of the blue.

KB: Really, I'm so glad you bring up this point. In my practice, because my clients are very well-educated, they're nourishing their dogs exceptionally well. I've got dogs on species-appropriate, fresh, non-GMO, and organic diet, so they don't exhibit those traditional physiologic symptoms of hypothyroidism. Many times, the only aspect that tips us off that there could be a metabolic disorder like hypothyroidism, is a very subtle change in personality.

That's a really important point for my audience to hear: if you're nourishing your pets very, very well, sometimes, the only symptom of hypothyroidism could be a very minor personality change. That's a really important point to make.

JD: Absolutely. Or intermittent bowel issues with gas. It passes and they forget about it. Or chronic urinary tract infections. I mean, chronic ear [infections], we deal for thyroid disease and a variety of other issues environmentally. You're going to have chronic urinary tract infections, but nothing else is wrong with the animal. It's competing. It's doing agility. It's wonderful. It's not lethargic yet. But I agree with you, Karen. The most dangerous situation and the clinical sign that we need to watch for is a change in behavior of an animal that has an established home relationship – family relationship or working relationship.

KB: Right.

JD: People see it.

KB: Jean, what test should be included when we talk about a complete thyroid profile or a complete thyroid panel? Let's talk about what clients need to be asking for. Another way of saying this is what is your lab testing is for compared to the regular veterinarian who's doing, let's say, just T4?

JD: Okay. The complete profile should include both the total and the free T4, the free T4 being a very tiny fraction of the total and being the biologically important functional component. It's the free T4 and its sister, the free triiodothyronine (T3), that circulate in the blood, unbound to proteins, that tell the pituitary glands sensor whether or not to stimulate the thyroid gland by putting out thyroid stimulating hormone (TSH). Okay, so you have total T4, free T4, total T3, and free T3.

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Now, that's the next problem. Many experts in our profession believe that it's useless to measure the total T3 and the free T3. I disagree with that completely. You need those in the complete profile because if you have an animal that's ill, and the T4, free T4, T3, and free T3 are all low, it's more likely that that animal has primarily a non-thyroidal illness.

The T3 and free T3 are the markers that tell you that this is likely non-thyroidal. It could be both, but it's more likely non-thyroidal. Why? Because the last thing that seems to go when the thyroid is wiped out is the free fraction of T3. So it can be normal. When the total T3 is low, the total T4 is low or suboptimal, and the free T4 is low.

Finally, we must have a thyroid antibody test. The best one to do for the initial screening is thyroglobulin autoantibody test. Now, you'll notice I didn't mention TSH.

KB: Right.

JD: I didn't mention TSH deliberately because when we know about the canine… By the way, it's species-specific. The human TSH antibody cannot be used to accurately measure the analyte in the dog. Okay, so we have a canine-specific TSH assay. And 70 percent of the time on average, it will predict the presence of primary hypothyroidism, but 30 percent of the time it won't. It will be discordant, either false-positive high of false-negative low. So, how can we rely on a diagnostic test with only 70 percent predictability? It should be a minimum of 90 percent.

KB: Right.

JD: And the same assay in people is 95 percent predictive.

KB: Right.

JD: We only have five percent chance of being wrong with the TSH. It's obviously a better test in people than it is in the dog. Why is that? Because the dog has an active thyroid regulatory pathway through growth hormone, which we're not measuring. If I could do anything, I guess it's two things: I'd get rid of the total T4 only on [inaudible 12:08] and I would get rid of canine TSH except in the cat.

Curiously enough, in older cats that that have their thyroid gland inactivated or ablated because of hyperthyroidism, two to five months later on average (Dr. Mark Peterson and I have been doing a lot of this work), they become hypothyroid because the thyroid gland isn't there anymore. When they become hypothyroid, these older cats that have been, say, radioactive iodine (I-131)-zapped, we have to treat them with thyroid hormone. How does the client recognize that?

All of a sudden, their cat that is hyperthyroid and reactive becomes a blob with sticky uppy hair, doesn't look at anybody, and loses around. Blob. They think, "Oh my gosh, my old cat has really, finally reached the end of his or her life." In fact, they are hypothyroid.

How do we monitor the hypothyroidism in these cats with TSH? It's the weirdest thing. TSH in older cats is very helpful in monitoring whether we need to treat the doses of added T4 and T3 in many cases to keep these cats back to a total metabolic balance, and they become cats again.

KB: Yeah.

JD: People are so excited. They become the cats they were before they were hyperthyroid. It's just amazing. It's amazing that we can do that, and we are doing it exogenously by balancing T4 with a smidge of T3. I mean, it's sort of like… What is it? Is it scientific? Yes. Is it medically sound? Who knows?

KB: Right.

JD: It works so I guess we have to say yes.

KB: Exactly.

JD: These cats get better. There's an entire chat group on Yahoo that talks about how this is done and how we monitor these cats now. It's terrific.

KB: Jean, in that scenario where the kitty was initially hyperthyroid, went through treatment, becomes hypothyroid, and then goes to the rebalancing act of putting back in just the perfect amount of adequate thyroid hormones to bring the kitty back to life…

JD: Right.

KB: Let's talk about one step before that. How do we test? How do we appropriately test thyroid function in adult and older cats?

JD: Okay. In an older cat, we do the same four tests we do in the dog: T4, free T4, T3, and free T3. But instead of doing the thyroglobulin autoantibody, because it's not relevant for the cat, we substitute with TSH.

KB: And along that same vein, when kitties…

JD: Could I say one other thing about cats until you say any over that?

KB: Yes.

JD: I want to give credit to Dr. Jennifer Wakeling who had first alerted the veterinary profession when she was in England and then at Vancouver about the importance of using TSH as an assay in cats with chronic renal failure, because the hyperthyroidism was being missed if the non-thyroidal illness, the renal failure, was pushing the total T4 into the upper limit of the range. We weren't diagnosing these cases until Dr. Wakeling said, "Wait a minute, that's a non-thyroidal problem that's making the total T4 look normal or upper normal. Let's look at other assays."

KB: Wow.

JD: She was the one who found that TSH was the most predictive whether the chronic renal failure cats also had hyperthyroidism.

KB: Wow.

JD: Because when you suppress the thyroid, as you know, Karen, you also worsen renal tissue profusion.

KB: Sure.

JD: So you have to be very careful. Some cats actually do better being slightly hyperthyroid when they have renal disease. You keep their kidneys working better.

KB: Yes.

JD: I want to give her credit for that.

KB: Excellent point to make. Additionally, all of us kind of know you as, I don't know, say, "exclusively dog" in terms of thyroid. The thyroid queen of dogs. Do you feel, Jean, like you have… Are you doing more and more thyroid testing on cats?

JD: We're doing more and more thyroid testing in cats, particularly because of the older cat situation. As I mentioned to you, Dr. Mark Peterson and I have been struggling along with Dr. Rhett Nichols about what to do with this, and then we hit on the fact that it is the TSH we need to monitor. We need to treat these cats that now have become iatrogenically hypothyroid. And we're all on the same page today, thank God.

KB: Yeah, wonderful. That's great. Okay, back to dogs for a minute. Let's talk about autoimmune thyroiditis. How does one screen… Well, actually, as a side note to link the cat issue to the dog issue, kitties and autoimmune thyroiditis, does it ever happen?

JD: Do cats ever get autoimmune thyroiditis?

KB: Yup.

JD: Yes, they can. It's not very well studied and I think it's a little bit of a hush-hush. About 20 years ago, there was a study in England where they looked at cats with adenomatous hyperplasia, you know, the tumors?

KB: Yup.

JD: When they looked at these tumors by fluorescence microscopy, they found some viral particles. These viral particles were retrovirus particles.

KB: Wow.

JD: And they were guessing that some of the trigger of adenomatous hyperplasia of the thyroid gland was caused by retro viruses just like the retroviruses in thymomas that are associated with myasthenia gravis.

KB: Wow.

JD: There's about 30 percent of this cohort of cats, and the whole issue disappeared. I'm not sure what that was about.

KB: That's very interesting.

JD: One person tried to look at it since them, but they may not have had the right cat cohort. It needs to be looked at.

KB: Yeah, I should say. That's a whole different topic, one that is not talked about probably because we don't know enough about it honestly.

JD: Correct. And we have retroviruses in cat obviously, so it seems natural seeing. As Bob Jacoby told us in the mid-'70s, we never saw hyperthyroidism in cats before that.

KB: Right.

JD: It suddenly appeared. What happened?

KB: Yup.

JD: Why did that happen? It's not that we didn't notice it; it would have been obvious.

[END OF PART 1]