

Life Is Like a Box of Chocolates

BY PAUL D. PION, DVM, DACVIM (CARDIOLOGY)

2008 is a special year for me. It marks the 25th anniversary of my becoming a veterinarian and the 40th year that the Winn Feline Foundation has been helping advance the health of cats worldwide.

These events might appear unrelated, but they are closely intertwined. I owe much of whatever success I have achieved to the vision and wisdom of the Winn Feline Foundation and several individuals who have given me assistance, guidance and luck along the way.

A common question posed to veterinarians is, "Why did you become a veterinarian? Is it because you love animals?" I never was sure how to answer that until one rainy afternoon while watching *Forrest Gump*, one of my favorite movies. My response now goes something like this:

"One day, for no particular reason, I decided to apply to veterinary school. Maybe it was to make amends for accidentally killing my parakeet, Swifty, when I burned down my room in sixth grade. My mother thinks it is because it was the hardest school to get into at the time, and I can't resist a challenge."

Whether this is my penance to Swifty or rebellion against my mother's desire for me to be a "real doctor," it has been one hell of a fun and rewarding journey—one that a special non-profit called the Winn Feline Foundation helped pave.

In October 1986, I was a 29-year-old cardiology resident at the University of California-Davis. My pipe dream, established in veterinary school, was to cure feline hypertrophic cardiomyopathy (HCM), a disease of the heart muscle that is the most common heart disease seen in cats of all types. I began that quest in 1982 as a second-year veterinary student, when I applied for and received my first research grant, "Pharmacokinetic and Electrocardiographic Study of Verapamil in the Cat." The Cornell Alumni Association took a chance on me with a \$3,000 grant.

The rationale behind the study was that Verapamil is a drug that reduces the amount of calcium entering heart cells during each beat. At the time, HCM in humans and cats was believed to be due primarily to excess calcium entering the heart cells. Verapamil never

became a widely used drug in cats, but a related drug, diltiazem, is still commonly (and perhaps inappropriately) used in cats with HCM.

The third research grant I received, in 1986, was funded by what is now called the Miller Trust (currently administered by the Winn Feline Foundation). The project looked at the effects of tPA (a clot-dissolving drug) on the saddle thrombi that often leads to death in cats with cardiomyopathy.

The first cat I treated with tPA was named El Blanco. El Blanco didn't have HCM; instead he had dilated cardiomyopathy (DCM). The drug dissolved El Blanco's clot, but unfortunately, El Blanco died soon after that "successful" treatment. Yet, that clinical failure provided the clues to an unsolved mystery. Until that time, veterinarians considered DCM an end-stage, incurable condition. No one knew why it happened, but in these patients, the heart muscle was considered "spent"—once function was gone, we thought, it could never be restored.

The key to unraveling this mystery was that Cindi Glassaur, El Blanco's owner, knew that El Blanco had a taurine deficiency. Taurine is an essential amino acid for cats. It was well-known that taurine deficiency could cause blindness in the form of feline central retinal degeneration (FCRD). It was suspected that taurine deficiency could also cause reproductive and other problems. In fact, there was a small group of roughly 200 scientists worldwide who focused their research on the role of taurine in animals and people. But other than effects on the retina, no real clinical effects of taurine deficiency had yet been proven.

That was about to change.

Cindi was determined that some good come from El Blanco's death. We talked about the taurine deficiency and whether it could have caused El Blanco's clots or heart disease. When a client asks, "Can there be an association?" a veterinarian's usual response is "unlikely." But in this case, two factors converged to ensure that the question was pursued. First, Cindi was persistent. No way in the world was I going to be able to ignore her questions. And

second, I'd been prepared for that question. While working on my first research grant in veterinary school, I read scientific papers arguing that taurine was critical for normal heart function. But no one had yet demonstrated a link between taurine deficiency and heart disease, or any other malady apart from FCRD and similar retinal problems in infants fed formulas.

It is almost as if fate wanted me to pursue this. So much so that I sometimes wonder if the finding was science or serendipity.

In a typical month, the University of California-Davis Veterinary Teaching Hospital would see one or two cats with DCM. During the next week, I was presented with three additional cats with DCM. Until then, I never measured taurine in the blood of any patient. At UCD, a wonderful professor (now retired), Quinton Rogers, PhD, was working with James Morris, PhD, in feline nutrition. They had a large colony of cats involved in nutritional studies and a laboratory where they measured taurine concentrations daily in blood and tissue.

Interestingly, each of the three cats with DCM that we saw the next week were eating the same food as El Blanco and had FCRD. We took blood from each cat and waited to learn if they



to were taurine deficient. A week later we learned that these three cats, all with DCM and FCRD, like El Blanco had low taurine concentration in their blood.

Intrigued and excited, I went to see Rogers. I told him the story of El Blanco and the other three cases with similar histories. For reasons I won't go into here, Rogers was skeptical about there being a real association. But, being the inquisitive and giving scientist that he is, he told me that if I wanted to look at some more taurine-deficient cats, he had 11 that were involved in a study in his colony. The kicker was that if I wanted to study them, I'd have to do it very soon because after four years of study, any day now, the cats were scheduled to be changed from a diet low in taurine to a diet high in taurine.

In those days, cardiac ultrasound machines were not the size of laptop computers, as some are today. They were BIG. The next day, after begging my cardiology mentors, William Thomas, DVM, MS, DACVIM, (a pioneer in the field of veterinary cardiac ultrasound), and Mark Kittleson, DVM, PhD, DACVIM (who helped prove in a study largely funded by the Winn Feline Foundation that feline HCM was a heritable gene defect), I rented a truck with a tailgate lift so I could transport our BIG ultrasound machine to the cat colony.

To Rogers' amazement, two of the cats tested positive for DCM. It was not enough to prove the condition was caused by taurine deficiency, but it raised our suspicions.

A month later, in early December, we were ready to try treating cats with DCM with taurine. It was safe—and anyway, what did we have to lose? On average, cats with DCM at our clinic lived just 30 days.

On December 1, 1986, Elizabeth Stoltz's cat, Cecil, became the first cat with DCM to be treated with taurine. A week later, Cecil was still alive but his heart ultrasound remained unchanged. By mid-December, it had been five weeks that Rogers' cats were fed taurine and their heart ultrasound findings were similarly unchanged.

A month later, although his heart appeared unchanged, Cecil was alive and feeling so good that we began decreasing the medication used to treat his congestive heart failure. Amazingly, the hearts of the two DCM-positive cats in Rogers' colony began to look more normal.

By early February, Cecil's heart was also beginning to appear more normal. My colleagues and I were getting very excited. The

“impossible” cure for what was then the most common and fatal heart disease in cats—motivated by Cindi and her cat El Blanco—might be real. Taurine deficiency, largely caused by a misformulation in commercial cat foods, was easily reversed by feeding a cat more, inexpensive taurine.

We had a thousand ideas about what we needed to do to prove this was real. We felt ethically bound to prove our suspicions before giving false hope to owners of cats with DCM or publicly implicating pet foods as the cause. There was just one problem: We had no money for clinical or basic cardiology or nutrition research studies.

Rogers suggested the Winn Feline Foundation—one of the few grant agencies willing to fund feline medical and nutrition research. We had one BIG problem, though: It was now late January or early February 1987 and the deadline for submitting grant proposals to Winn had long passed. In fact, it was close to the time their committee met to select what research they would fund that year. It seemed our luck had run out and progress would have to wait until we could identify a funding source, write grants, go through the funding cycle, and all the rest. That could take a year, and cats were dying!

I made a call to the Winn Feline Foundation offices and explained our situation. A day later, I received a call from Winn's president, Joan Miller. The conversation didn't begin well. Miller obviously was conflicted. The foundation was, after all, bound to follow a grant process delineated by deadlines.

Still, I could hear this cat lover and enthusiast wavering. Finally, Miller explained that she consulted the foundation's board of directors and medical advisers, and some of them told her they had known me as a student and intern and that they had confidence in me. They said if I felt we were on to something important, she should set aside protocol and accept our grant application, if we could have it to them within a few days.

Luckily, Winn Foundation grant applications aren't as laborious as others. We worked furiously and mailed off 12 copies as directed. One or two weeks later we got the call: Our grant was funded.

The \$10,000 awarded—the limit for Winn grants in those days—made all the difference to our work. To this day, I believe that no other organization in the world would have made that choice.

From that first introduction to the Winn Feline Foundation, through several other experiences as a successful grant applicant and many years as a Winn Foundation medical advisor and member of the grant selection committee, I have watched the Winn Foundation make a huge impact on feline medicine. Winn manages to “bet” on the right studies submitted by the right people who demonstrate dedication to using the funds to make a difference for cats worldwide. The entire organization is committed to not becoming a bureaucratic tangle, as has happened with so many of the larger granting agencies.

Because the Winn Foundation was, and continues to be the type of organization that it proved to be in early 1987, I have remained one of Winn's greatest fans and encouraged other foundations I have served with to look to Winn as an example for how to conduct themselves and their programs.

In large part thanks to the Winn Foundation, our work made a difference. We were rapidly able to complete clinical and basic cardiology and nutrition studies that proved the association between taurine deficiency and DCM. By mid-1987, most cat food formulations in the world had been changed to include more taurine. By the early 1990s, the prevalence of DCM in cats had plummeted to the point where it is now considered a very rare condition.

Ours is but one of many success stories made possible by the small and very dedicated Winn Feline Foundation and the great people who continue to further its mission.

Life is like a box of chocolates. You never know what you're going to get. And that's what makes it fun—and why we need organizations like the Winn Feline Foundation to keep doing what they are doing. Happy birthday, Winn!

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