

But what of the cardiomyopathy in my bloodline? Even though his sister Ricochet died of it at five years, Rowdy had lived eight very healthy years to that time and the odds were leaning toward him not being affected. His father, Qanah, was still alive at 10 years of age and did not succumb directly to the disease. Qanah was euthanized with diminished motor skills close to 12 years of age in 2006, but as mentioned earlier, later facts on the mode of inheritance make him an interesting case study, especially as he was the father of the three dead young bitches.

Rowdy's mother, Pandora, had unquestionably died of the disease. As Dr. Kathryn Meurs' final determination that autosomal dominant was the correct mode was still several years away, I erroneously calculated under the autosomal recessive mode of inheritance. In that mode, it would have given Rowdy a 50 percent chance of following his sister to the grave as an affected dog, or a 50 percent chance of being a carrier of the recessive and never experiencing manifestation of the disease.

Then there was the prospective mother, Halle, herself a grand daughter of a cardiomyopathy victim. As Jet Setter had died of the disease, it meant that in autosomal recessive mode, his son, who fathered Halle and did not die of the disease, had to be a carrier. What I knew of the history though, I gambled that the bitch he bred to produce Halle was a clear, which in genetic percentages, gave Halle a 50 percent chance of being clear and a 50 percent chance of being a carrier.

In retrospect, I probably should have had an echocardiogram performed to determine if either dog was affected. But tests for cardiomyopathy today do not necessarily portend what might develop in the future, and time was of the essence because the breeding had already taken place. I did not have a lot

of choices and was backed into a diminishing corner.

I rolled the dice. I was banking on Rowdy being a carrier and the prospective mother being clear: a good possibility that the litter would produce 50 percent clears and 50 percent carriers. If I wanted to save the bloodline and keep the type I had worked so hard to maintain since the 1960's, then I should gamble and let nature take its course. Oh my goodness, if I had only known the real mode of inheritance at the time....

The Bikila/Marks-Tey "V" litter of eight pups – two females and six males, was born in June 2005, and was all that I hoped it would be. My ol' mate Rowdy seemed to bask in the glory of producing offspring. It was heartwarming to watch him tolerate the exuberance of youth and thoroughly enjoy playtime with his children.

The pups were approaching six months old when, in late 2005, I went to awaken a peaceful Rowdy, by now nine years of age, who was curled up on his bed in his kennel. When I opened the kennel door and called him there was no response. His heart had stopped... and my heart sank to the pit of my stomach.

I have some good physical and mental foundations to work with in my "V" litter. But with an autosomal dominant mode and Rowdy having died of DCM, then I will have affected animals and only the genomes of the two parents will dictate how many. If Rowdy was homozygous dominant (CC), it will not matter what the mother is because the entire litter will be affected and all eight would be expected to die of DCM (see Punnett squares in preceding article). If Rowdy was heterozygous (Cc) and the mother is clear (cc), then only half the litter (on average) will die of DCM, but which half? If Rowdy was heterozygous and the mother later proves to be

heterozygous (Cc) — which means the mother would also eventually die of the disease — then on average 75 percent, or six of eight pups will have the disease. But which would be clear?

I have moved to incorporate potential buffers against DCM for the future: a champion bitch purchased from Mike Penny and Nikki McClendon (4-year-old Ch. Paradigm's Parasol) and a champion red boy bred by Nikki McClendon (3-year-old Ch. Merrimac's Gentleman Jim). Both are great grand children of Qanah and both have recently passed early cardiac testing and have been mated to help my breeding program. I also have semen from Qanah's brother, Bikila's Quarterback of Marks-Tey who had a great stud career in Australia and passed cardiac tests early in his life and lived to 12 years before being euthanized with diminishing motor skills. He appears to have a wide spread of clear offspring and after a full evaluation he may be a prime candidate to help the ailing breeding program.

I have detailed this saga of death to show why I am so passionate about the subject of dilated cardiomyopathy. It was a horrendous time in my life as a Doberman breeder and I sincerely want to help others so that they will avoid a similar fate.

I was at a point of surrender after my breeding program came crashing down with an avalanche of deaths. Now that I have decided to dig my way out, dust myself off and start all over again, I am determined to try to do something about it. It is not only for the breed as a whole but selfishly for me because I want to continue to breed and I need information on breeding prospects.

This article is not a witch-hunt and no individual dog, kennel or breeder will be, or should be, held responsible for the predicament I am in today. The same is true for

the many caring breeders at large who have been touched by cardiomyopathy.

As a breeder I made decisions to go out of my family and then performed my normal close breeding; and unwittingly set my kennel on a massive collision course with DCM.

Nobody held a gun to my head to use stud dogs offered by people with the utmost integrity and ones who care about the breed and its future as much as I do. The difficulty for all of us, apart from no DNA test, is the late onset of the disease — a time after which we may already be several generations down the road.

All of which brings me to the crusade for an independent open registry, a database — some kind of open exchange of information — to track cardiomyopathy in Doberman Pinschers.

## The State of the Union: The Doberman Breed Is Under Assault Externally and Internally

When I made a permanent move to the United States on November 30, 1977, to write a tennis book in Philadelphia, the Doberman Pinscher was in the midst of an unprecedented boom. By the time I had finished the book in late 1978, a record 81,964 registrations had been recorded that year by the American Kennel Club.

By the end of the 1980s the breed had dropped to 21,782 registrations. In 1998, exactly 20 years after the record registrations, the number had dropped to 15,637. At last count in 2006, it was down to 11,546... a full 70,418 less, or an 86 percent drop from three decades ago.

I am fully aware that the initial decline in registrations came after the Doberman was no longer the guard-dog-of-the-moment and the money hungry puppy producers who had overplayed their hands and damaged the breed in the 1970s, finally moved on to the Rottweiler and then the Pit Bull. The Rottweiler has also seen its annual registrations drop 83 percent to 14,709 in the past 10 years.

I am also aware the Doberman breed is under assault from the lobby which labels them as a threatening aggressive dog in ordinances across America. Some homeowners' insurance is denied. There are also the militant animal rights groups which, as part of a sweeping agenda to allow humans no animal pets whatsoever, want to ban all ear cropping and tail docking as a step toward that goal.

Internationally, cropping and docking bans are now in place in Continental Europe, Britain and Australia. Doberman breeders (and other breeds) have had to deal with the new laws the best they can. In England, where cropping is outlawed, docking was banned in 2007 and breeders who want to continue either break the law (up to a year in prison and hefty fines); send their pups to Belgium or join clubs in Northern Ireland to have them docked. Docked dogs are banned from shows in which the public pays for admission and the situation is truly grim.

In my native Australia, cropping has never been legal and docking has been outlawed for four years in all states and territories and breeders are scrambling. Those who want to dock have to form breeding partnerships in Western Australia or the Northern Territory where there are some loopholes in the law at this time. In my home state of New South Wales, the most populace state, only 22 Doberman litters have been registered in the past two years... and 19 of those litters are by "back-yard breeders." There is a motion to ban all docked dogs from the show ring in Australia and, coupled with the changes in Britain, Dobermans in both countries are on an extremely perilous path.

We are facing similar onslaughts in the United States and the walls are closing in. The animal rights lobby is pressuring and donating funds to our lawmakers and we, the dog enthusiasts, have no truly effective lobby.

External pressures are one thing, but the internal health pressures maybe even more destructive. Cardiomyopathy is rampant and is a worldwide problem. Dobermans in all countries are from the same original gene pool and research shows the disease is prevalent no matter the country.

I do not want to appear like the man on the street corner with the sandwich board that reads: "The End is Near," or even Chicken Little, but any clear thinking person must see that the breed is in dire straits. When I look around me both here and overseas, I truly wonder if the Doberman will be a rare breed or even a historic afterthought at its official bi-centennial in 2099.

Most of the dog show world in America plays on blithely, caring more about ribbons than the travails of their international brethren and the future of their own breeds. It reminds me of the band playing music on the deck of the Titanic while the ship was taking on water.

The market is depressed otherwise Dobermans would not be at 11,546 registrations and dropping. Depressed breeders overseas are wondering if they can ever win the battle against the animal rights people.

Meanwhile breeders worldwide keep running slap bang into health problems led by cardiomyopathy. Some Doberman people just do not want to fight what they consider a losing battle on health and cropping and docking and eventually move on.

There are no hard numbers for cardiomyopathy deaths because so many pets die of “heart attacks” and were never diagnosed by a veterinarian. Also, deaths in breeding programs are mostly muted because of the perceived stigma of a genetic problem. What I do know is that the No. 2 ranked dog in America in 1978 is now the No. 1 ranked breed in dilated cardiomyopathy deaths. I read several accounts where it is estimated that the Doberman has more deaths from the disease than all the other breeds put together. When Texas A & M University began studying pedigrees of DCM victims the exclamation was: “It’s everywhere!” You know it has to be everywhere when every affected dog produces litters containing between 50 percent and 100 percent of affected offspring.

I originally thought that Dr. Clay Calvert’s estimate in 1986 that 8,000-10,000 Dobermans were dying each year from DCM, was a gross exaggeration. I have to re-evaluate, as the American Veterinary Medical Association estimates that over 3 million dogs are afflicted with heart disease. Maybe Dr. Calvert’s numbers were way high, but even if 1,000 are dying each year – and I certainly would not challenge that number – it is chilling. Stop and think a moment about the facts of Dr. Meurs’ study in which 41 animals from one family were documented: 25 out of 35 dogs (71 per cent) fully diagnosed in the study were affected with DCM. There were 6 dogs that could not be determined.

### Where The Disease Came From... and Where We Should Take It

Genetic mutations such as dilated cardiomyopathy have been around for a very long time. Consider this: canines, felines, bovines, etc. and the human race are all touched by dilated cardiomyopathy. So where did it start? Could it have been during the evolution of the species? After all, all organisms share so many physical features that it lends credence to the argument that every living creature on this earth had a single origin of life 30 to 40 billion years ago.

Charles Darwin wrote as such in his 1859 epic on evolution, “Origin of Species”: “Analogy would lead me one step farther, namely, to the belief that all animals and plants are descended from some one prototype. But analogy may be a deceitful guide. Nevertheless all living things have much in common, in their chemical composition, their cellular structure, their laws of growth, and their liability to injurious influences.”

Cardiomyopathy is indeed an injurious influence which is spread over many different species. Was it a spontaneous mutation in DNA messaging that was first introduced aeons ago, or were humans, dogs, etc. afflicted by some powerful mutagen common to all lives at some point in time?

When I first started in the Doberman breed in the 1960s, sudden heart failure was viewed as a simple heart attack. One assumed it was clogged arteries or something possibly caused by the food we were feeding them at that time. In my early days many dog owners in Australia fed only raw kangaroo meat. They are carnivores are they not? So it must have come from the red meat which is not good for the heart, right? Who knew it was a genetic mutation?

Old-timers who remember the first Doberman magazine, the mimeographed Doberman Pinscher News and Views, will find in the December 1964 issue, the first alarm bell for our breed. There was a call for help from the Doberman fancy on the increasing incidence of “sudden deaths.” The article mentioned that artery disease had been dismissed and Dr. Thomas James of the cardiovascular research department of the Henry Ford Hospital in Detroit was calling for owners who had dogs die suddenly of a heart attack to submit the hearts for research.

A year later the first scientific report of cardiomyopathy in the breed was published by Dr. James and Dr. E. H. Drake, and was followed by a more extensive report by the same men in 1968. Dr. Calvert wrote that congestive heart failure in Dobermans resulting from cardiomyopathy was commonly diagnosed during the 1960s and 1970s at the Animal Medical Center in New York. Calvert, who wrote a scientific paper on the disease, first encountered congestive heart failure in Dobermans in 1971.

It really wasn’t until the 1980s – through mounting evidence of personal experiences and Dr. Calvert’s article in the Doberman Quarterly in 1986 that it began to hit home what we were really dealing with in our breed.

Peggy Adamson certainly had no idea in 1951 when she wrote her famous article about Ilena and the Seven Sires of the 1940’s. The Seven Sires were Ch. Domossi of Marienland, Ch. Emperor of Marienland, Ch. Dictator von Glenhugel, Ch. Alcor von Millsdod, Ch. Favoriet v Franzhof, Ch. Westphalia’s Rameses and Ch. Westphalia’s Uranus; and Ilena was Ch. Dow’s Ilena of Marienland. These animals, which came from the best of the imports from Germany, were the foundation of everything we have today. They were what are often referred to as “matadors” – studs which are bred to a wide variety of bitches. They were also mixed and matched to offspring of each other.