NEUROPATHY IN THE ENGLISH COCKER SPANIEL

By Elizabeth H. Neff, Patricia Janzen and Joyce B. Winkels

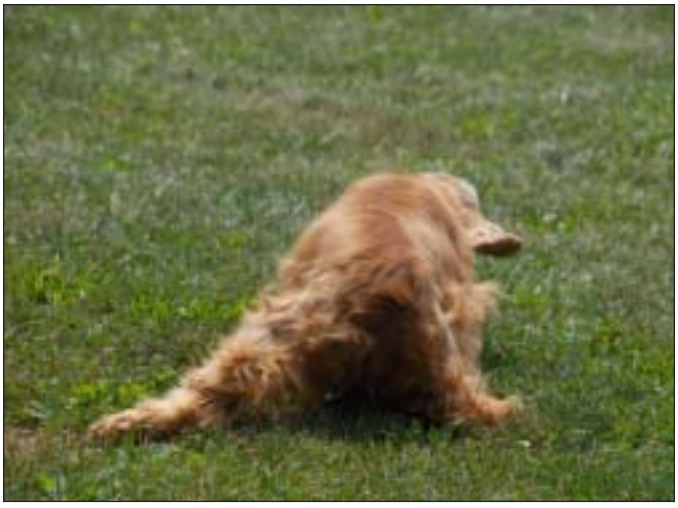
This article is an initial attempt to present our understanding of a disease diagnosed as general Neuropathy in a small number of our solid colored English Cockers. In our pursuit of a diagnosis for this condition, we have collected data involving several solids and one parti-colored English Cockers with similar symptoms, although the description of the symptoms of the parti dog differs somewhat to what we have experienced. We do not assign blame to any dog for causing this disorder, although we do believe there is a familial basis with the identified solid dogs and all of these dogs have some ancestral similarities. This article will only mention names owned or bred by the authors. Others can add their affected dogs to the list, if they desire, in an attempt to compile as much breeding information as possible in order to help eliminate this condition from our breed. Pedigrees from possible affected dogs and especially video will be very much appreciated and can be sent to Liz Neff or Addi Pittman along with written permission from the owner for this information to be shared with the researchers working with us on this problem. The professors would very much appreciate video of other affected dogs at a variety of stages of progression and on different surfaces.

My first knowledge of a problem was when Scarlette, Ch. Canterbury's Lady in Red, began to appear weaker in the rear and to tap her rear foot; she was seemingly unaware that there was any problem so I thought it was aging. This symptom first appeared at eight years of age. Scarlette gradually seemed less able to direct where she wanted her rear legs to take her and just appeared "wobbly" in the rear.

Unaware of other dogs with Scarlette's problem, I began having her vets do as many tests as they could come up with to see what we were dealing with. All of the x-rays and blood work came back normal for Scarlette and so we just watched her become increasingly weak and did our best to keep her comfortable as long as we could. She never seemed to be in any pain but she was the most stoic, big hearted dog I had ever known so I doubt she would have ever shown any sign if it did become painful. Scarlette was a bitch whose tail wagged even when she was under anesthesia, as to which her doctors will attest.

She declined gradually over a four year period until I was no longer able to watch her become so incapacitated and unable to function in any way. She never lost her enthusiasm for life but it came to a point where there was no excuse to keep her going for my own selfish purposes and she died at the age of eleven. Just before she died she kissed my face and made me feel we were doing the right thing and that this was in her best interest.

When it came to the point where I was able to talk about Scarlette's loss, I called her breeder and told her what had happened. She said that Scarlette's brother had also had the same symptoms and had died within a month of



The photographs show the lack of control in keeping her legs under her as well as the muscle atrophy from not being able to exercise and that she is very down in the front as the has disease progressed forward.

Scarlette. I then talked to Patty Janzen, who had bred their sire, Ch. Ebonwood Ashgrove Trademark, who along with at least one of his sisters, was also affected. We began to discuss this more often and came to find that there were several dogs within the same family with the same problem.

This information was retained until several years later when I noticed that first and ominous sign of foot-tapping in Scarlette's daughter Marigold, Ch. Canterbury's Velvet Touch ECM.

Marigold did not show signs until after she had passed her ninth birthday. I was reminded of a conversation from someone who had seen the disease and who had felt that subsequent generations showed signs later and later in life. I am not sure how true this has been for others, but it gave me a little hope that Marigold's symptoms would progress more slowly than Scarlette, which Marigold has to a small extent. Also of interest is that of Scarlette's thirteen children, Marigold is the only one that we know of, who has become affected to date and all of these children are nine years or older.

We have proven that when we work together we can accomplish great things and leaving any segment of our breed out of the loop is a disserve to them as well the dogs.

Now that I had two generations of my own and knew that Marigold was the third generation to become affected we, Patty and I, felt it was our duty to the dogs and to our breed to do as much as possible to figure this out. Specialists were sought to do any testing which might be helpful, short of causing Marigold any pain. We would also like it to be understood that the dogs we own and those we have been made aware of, never appear to be in any pain, they continue doing their daily routine with their usual enthusiasm, just much slower. The doctors who have examined these dogs seem to agree that there appears to be no pain associated with this so far.

The vets began with x-rays and blood tests as had been done with Scarlette and again, all were negative. Reflex testing was performed and this revealed that Marigold had slightly reduced reflexes in her rear knees but did have full reflexes in her rear feet. It was at this point we were referred to Dr. Gerald Northington, who is a well regarded Doctor of Neurology at the Metropolitan Veterinary Hospital in Pennsylvania. He also did extensive reflex testing and discovered that this was NOT a disease limited to the rear legs but that her entire spinal cord was affected. Marigold's disease had progressed to the point of affecting her head carriage, her front legs, and her proprioception, that is, her "relationship to space." For example, she would reach out to get to the exam table when she was not close enough to actually do so. Marigold would also start making the motions to walk well before she was set on the floor. She demonstrated great difficulty in rising from the floor, having the most trouble with getting her rear legs under her. There was a noticeable weakness and a widened stance in her front legs as well as some bobbing of her head while trying to stand.

The Vets were asked if it appeared to be similar to any diseases diagnosed in other breeds, for example Degenerative Meylopathy (DM). Dr. Northington felt certain that it was not DM, nor was it the disease found in Rottweillers called Cerebellar Abiotrophy (CA) although it appeared to be more similar the CA than to DM. He stated that he felt we were seeing something that was new to neurology and might be of interest for a study. We continued to pursue a diagnosis which entailed additional blood work as well as sending a urine sample to the University of Pennsylvania. The U of Penn is conducting a DNA study using urine samples. The extrapolated DNA was compared to known genetic defects in a variety of neurological diseases in an attempt to find genetic similarity with Marigold's condition. None were found. The additional labs were again within normal limits except for her thyroid function which showed a value of 7. Normal is 8 to 12. It was determined that a value of 7 is actually quite acceptable for a dog of her age. She was eleven years old at the time this lab work was performed.

What's next? Now, we were at the point where the next step would be the big and expensive tests. We proceeded to schedule an MRI of Marigold's head and cervical spine to the C4 spinal segment. This imaging study is done under general anesthesia for dogs. The MRI actually went further down her spine than C4 and the only abnormality that was found was a slight bulging of the disc between C3 and C4. Although it is pressing on her spine, the doctors felt that it is minor and does not account for the symptoms Marigold exhibited.

Next we tried a spinal tap, again under anesthesia, again NOTHING. These tests did rule out several diseases as did more blood tests. The cause was clearly not showing up as a tumor or death of tissue in the brain and showed no inflammatory nature, and the tests seemed to rule out any known genetic disorders.

We were still nowhere and so Dr. Northington suggested we see Dr. Vite, who is a Professor of Neurology and Researcher at the U of Penn's Vet School, and Dr.

Giger, Professor of Genetic Research at U of Penn. Both of these doctors have Scarlette's and Marigold's full veterinary files and are currently reviewing them.

Marigold has now been observed by Dr.Vite and the Neurology Department at Penn and they have performed an Electromyography (EMG) which is a technique for evaluating and recording the activation signal of muscles. EMG is performed using an instrument called an electromyograph. An electromyograph detects the electrical potential generated by muscle fibers when these fibers contract, and also when the fibers are at rest. It is the same test done in humans to help diagnose Carpel Tunnel and other nerve disorders.

The EMG results were perfectly normal and so it has been determined that Marigold's muscles and the nerves to the muscles are in normal condition. This result eliminates the need for biopsy as the muscle and nerves are functioning normally. The only conclusion that has been drawn from this is that the failure must be either in the brain or the spinal cord and is a disease of transmission to the nerves and not of the nerves themselves.

Dr. Vite seems to feel that this is a disease that is most likely new to neurology, although he knows it is not new to us. Dr. Vite is now discussing Marigold's symptoms with neurologists at the other veterinary schools to see if they have had any dogs brought to them with similar symptoms. He has also asked that we put out a request to other breeders for video of any other affected dogs. If you have a dog that you feel might be suffering from this disease, PLEASE send us video of your dog on various surfaces, walking, standing, getting up and lying down. I hope we will also have a way to store DNA from possibly affected dogs so that once Dr. Giger and his staff of geneticists get involved with us that we will have a pool of dogs to work from. Dr. Vite understands how difficult this next request will be but if there are any dogs who are reaching the most advanced stages of the disease, and when the time comes, it will be the most helpful if they could do a post mortem. Marigold will be taken to Dr. Vite when she expires but she is still enjoying life which we hope will continue for quite a while. I will ask him what is involved and if the dog must be sent to the U of Penn or if slides can be sent. Dr. Vite's number and address is below so any owner willing to help with this issue can have their Vet contact Dr. Vite prior to P.M. for guidance.

On a breeders note, Marigold and Scarlette were always very sturdy bitches and other than this problem, Marigold has had no other health issues. Marigold is OFA Good and PRA and FN normal with an outstanding temperament. When viewed as a whole, this problem is just part of her breeding profile. Marigold's first litter has reached an age where we might see a sign of the disease and as yet there have been none. This brings up the dilemma that this disease presents to us as breeders which is that, by the time symptoms appear, a dog has already passed the age of breeding. I feel breeding to old males is always a good idea since, were they to produce a problem, it would already have become known. My own feeling is that dogs from similar pedigrees also have a great deal to offer our gene pool and that sensible breeding choices will allow us to keep these genes while reducing the chances of perpetuating this disease. When we have a gene test for this disease, it will make the goal of eliminating this disease even more attainable. Our hope is that by sharing this information with everyone concerned for our breed's future, we will begin to solve this problem as we have for PRA and FN. We feel that breeders of both solid and parti-colors must be informed of this condition, as it is not clear that this disease affects only solid colored dogs. It is destructive to our breed as a whole to make such delineations. However, if this does turn out to be a disease of solids it will still take all of our efforts to find a solution. We have proven that when we work together we can accomplish great things and leaving any segment of our breeders out of the loop is a disservice to them as well as the dogs.

A great deal of credit and thanks is owed to many people who have been extremely supportive of these efforts and we would like to thank all of the breeders who have been very supportive and caring of Marigold throughout this process and have shared their own experiences with us. Thanks to all of you who have come to meet Marigold at American Spaniel Club and at the 2008 National to see firsthand what we are dealing with. She enjoys the attention very much. Credit must especially be given for the support from ECSCA President Kathy Moore, the entire ECSCA Board of Directors, to Addi Pittman, the ECSCA Health Committee and also to Dr. Bruce Barrett and of course Dr. Northington, Dr.Vite, Dr.Giger and their staffs. All of your concern and support has made this difficult time a bit easier and we are indebted to you all.

Pedigrees, video, and questions can be sent to:
Elizabeth H. Neff
190 Gelsinger Rd, Sinking Spring, PA 19608
dragonstarecs@comcast.net

Addi Pittman
1915 Olmos, Alice, TX 78332, addi@sbcglobal.net

Veterinary questions regarding post mortem procedure can be directed to
Charles H. Vite, DVM, PhD Dipl. ACVIM (Neurology)
MJ Ryan Veterinary Hospital
Assistant Professor, Section of Neurology
Department of Clinical Studies - Phila
School of Veterinary Medicine, University of Pennsylvania
3850 Spruce Street, Philadelphia, PA 19104
ph: 215-898-9473, fx: 215-898-9937