

HEALTH MATTERS

At seven months of age, Mia showed no symptoms of PN. The disease claimed her life within five months of this picture.

Photographs of Mia courtesy of Todd McGuire.



Perspectives on Polyneuropathy

A malamute owner, a breeder, a veterinary neurologist and two researchers discuss their experiences with this inherited disease.

A Pet Owner's Perspective

By Todd McGuire

Mia was my first Alaskan malamute, and she was the best pup anyone could ask for. Sadly, her young life was cut short by a form of Polyneuropathy (PN), a horrible disease that I hope no one else ever has to experience.

To give you a better understanding of polyneuropathy in Alaskan malamutes, I encourage you to read an article written by Vicky Maclean a few years ago for the AMCA: <http://www.alaskanmalamute.org/health/polyneur.asp>

In Mia's case I first noticed something was wrong when she was eight months old, and she started to bunny hop while running around at the dog park. Thinking she may have put her back out, we went to our vet for a chiropractic adjustment, but a few days later she was the

same again. Thus began three months of vet exams, physiotherapy visits, muscle building exercises, swimming, underwater treadmill, etc. She had good range of motion in her back legs, but was definitely lacking some muscle there and did not like to bend her legs much. Mia would sit funny with her legs stretched straight out to one side, and when walking she flicked them out to the side rather than lift and bend at the knee.

We did x-rays to check her hips. They were fine, so we proceeded with the muscle building exercises and swimming/treadmill in an attempt to build up the muscle again. After two months of this, we saw no improvement, and she was getting stiffer and slower at getting up. One day at the physiotherapy session, we checked Mia's nerve reflex responses and were a bit surprised at the results. Her back legs had almost no response, especially the femoral

nerve; yet her front legs had above average responses. Typically, dogs have good responses in their back legs and poor responses in their front legs, so something wasn't right.

In April, we saw a specialist vet who deals with surgical neurological problems, and it was the first time that we actually got a yelp out of her while a vet was poking and prodding her. He suggested that she could have a bulging disc in her back, which could explain the poor nerve reflex and muscle loss. So Mia went back for more testing; this time another round of x-rays, an MRI, and a spinal fluid sample to check for any environmental causes. All the tests came back negative, so we knew her problem was not environmental or musculo-skeletal.

Five days after getting all her results back, the veterinarian rang to say he had discussed Mia's case with a number of colleagues, and they all agreed that the likely problem was a form of neuropathy. He went on to tell me that the day would come when Mia would no longer be able to get up without help, or stand, or even go to the toilet, and when that day came, I would likely have to put her to sleep.

To say this gutted me was an understatement. Here was my beautiful pup who had helped me through one of the hardest times in my life, and I was being told she may not have much longer to go. At this point, I was unsure of what to do, so I started searching the Internet for anything and everything I could find on neuropathy in malamutes, and this was when I stumbled across Vicky's article. Reading it was like marking things off a checklist, and I knew then that this was what Mia had. To make things even worse, I emailed my breeder and the other pet people who owned Mia's littermates, and within five minutes of sending my email, the lady who owns Mia's sister, Misty, in Tasmania was ringing me to find out the symptoms – it turns out that Misty is likely affected, too.

Initially, we thought that Misty was worse than Mia, as she was collapsing all the time and struggling to get back up. Mia, on the other hand, was on steroids and was having acupuncture done and I was adding a Chinese



Mia at 11 months old, one week before she died. Note the severe loss of muscle in her hind leg, due to polyneuropathy.

herb powder to her food, in an attempt to stimulate her nerves. After the first week, she was showing a slight improvement, but that was short lived. Less than three weeks after learning she had PN, Mia's condition went downhill very quickly, and that day I dreaded was upon me. Depending upon the severity of the PN, some dogs can go into remission later in life, and some can even bounce back from not being able to support their own weight if someone is there to nurse them through it. In Mia's case, her back leg muscles had deteriorated to the point that they were virtually non-existent, initially from the disease and then from lack of use. Even if Mia had pulled through, she had no muscle left to support herself, and her previously strong front end was losing strength fast.

On May 4, 2010, I had to make the hardest decision of my life, choosing between Mia's well being and my selfishness to keep her with me. On that day, we said our final goodbyes and let her go to a place where she could run and play again like all pups should. She was 11 days shy of her first birthday.

Unfortunately, very little is known about PN, despite recorded cases in malamutes being around since the 1970's. I had an autopsy done on Mia to try and gain a better understanding of this disease, and in the hope that no one else ever has to go through

this again. In Mia's case, the autopsy revealed damage to the myelin sheath that protects the nerves, almost no muscle in her rear legs, and muscle deterioration in her front end. Further testing ruled out any environmental or musculo-skeletal causes, leaving inherited/genetic causes as the most likely. While we are able to test for damage of the myelin sheath on dogs while they are alive, this is an invasive procedure and should only be done if a dog is suspected of being affected with PN. At this stage there is no test for breeding dogs to see if they are a carrier of the disease.

On a more positive note, there are currently studies being conducted to try to develop a test for PN. Keep reading for more information.

If you have any questions about PN, please do not hesitate to contact me as I am happy to discuss it. To facilitate discussions, I have created a Facebook group called 'Alaskan Malamutes with Polyneuropathy.' I encourage all of you to become members and help raise awareness of this serious health issue.

Lastly, I would just like to thank everyone who has helped and supported me through this. It has meant a lot.

Editor's Note: Todd McGuire's story is published in memory of Mia McGuire. This article appeared in the Alaskan Malamute Club of Victoria's "Malamute Mail" column, and is reprinted with permission.



Left - Ben showed no signs of polyneuropathy until he was one year old. He was a wonderful show dog and finished his championship at ten months of age.

Right - Ben at just two years of age, though he looks older. At this time he had shown signs of polyneuropathy for a year.



A Breeder's Perspective

By Edie Thomas

I was invited and encouraged to write about my experience with Polyneuropathy (PN) from a breeder's point of view. Let me first say that I don't think anyone plans a breeding expecting to get a serious health problem. I didn't even know what PN was until I experienced this devastating condition with my very first Alaskan malamute litter.

I purchased my first show quality Alaskan malamute in early 1998. Her name was Ch Wild Wind's Royal Diamond, aka Hope. I finished Hope myself at ten months of age from the puppy class. She went on to become the 1998 AMCA National Specialty Grand Sweeps winner and Reserve Winners Bitch, a multiple group placer, Top 20 contender, and Best of Opposite Sex winner at the 2001 Eukanuba Classic. Needless to say, she was everything I wanted in a show dog and I adored her. Hope was bred only once in her life, to a well known sire, and a litter of seven puppies was born on June 24, 2000. At that time, I had no idea what would happen as those puppies matured.

From that litter, I kept a male named Ben, Ch WindStar's Royal Guns Salute. Ben finished his championship at the age of ten months, but by 12 months of age, I noticed that he was limping and would "bunny hop" when he ran. My veterinarian thought Ben might have panosteitis, an inflammation of the

bones that is seen in fast-growing large-breed dogs. As the months went by, Ben kept getting worse, and my vet recommended I take him to the University of Georgia Veterinary Teaching Hospital in Athens for further diagnostics. On September 11, 2001, we went to UGA. The neurologists did a hands-on examination, performing various reflex tests on Ben's feet. Due to his slow responses, they explained that they suspected PN but would need to run more extensive tests for a positive diagnosis. After doing electromyography, nerve conduction velocities, and muscle and nerve biopsies, the doctors confirmed that Ben did indeed have idiopathic PN.

As the weeks continued, Ben lost the use of his legs and could not stand at all for periods of time. I

spoke to another malamute breeder, who advised me that Ben would stand again and to just "hang on" for awhile. Ben did finally stand again, but his legs would tremble the entire time, similar to someone with Parkinson's disease. Ben also developed mega-esophagus, which is common in PN dogs. Mega-esophagus affects the eating and drinking abilities of dogs and makes them sound like they have been debarked. Ben could not run or jump and was very unsteady on his feet. In many cases of PN, dogs will get better for periods of time, but unfortunately Ben had a very severe case and was never able to move and be a normal dog. By the age of three, the mega-esophagus was much worse, and I could not watch my dog waste away. I decided to



"I don't think anyone plans a breeding expecting to get a serious health problem."

Breeder Edie Thomas

Lightning was the second dog in the litter to develop polyneuropathy; he started showing symptoms at 16 months of age. He is pictured at one year of age, before he showed any evidence of the disease.

ethanize Ben on November 26, 2003.

Shortly after I first took Ben to UGA, his brother Lightning, aka WindStar's Fury, started showing signs of PN. While at my home for show training, I noticed the same "bunny hopping" gait that I had seen in Ben. I told Lightning's owner about Ben's PN and offered to take him to UGA for testing. When I contacted veterinary neurologist Dr. Marc Kent at UGA and told him I suspected another dog in the litter had PN, he asked to do a study on my litter at no charge to me. This was a major financial offer, as Ben's PN tests had cost over \$2,000. Lightning was tested and diagnosed with a severe case of idiopathic PN. His owners did not respond to my offers to replace him, and I doubt very much that he lived long, considering how severe his case was.

It was February of 2002, just four months after Lightning had started showing symptoms of PN, when another brother, Petey (WindStar's Pistol Pete), began stumbling occasionally. Because I had told his owners about the PN and what symptoms to look for, they contacted me immediately with their concerns. I called Dr. Kent and arranged for Petey to be tested at UGA. He was diagnosed with a mild form of idiopathic PN. Petey was the only one of the boys to survive to an older age, as he lived a normal life without any issues until he was ten years old.

Petey is an example of the scary variability of PN. His form was so mild that, unless a person knew what to look for, you would not think anything was wrong with him. He was tested and diagnosed with PN only due to his



Petey had a very mild case of polyneuropathy and lived a normal life until ten years of age.

littermates having PN. Even in the mild cases, PN symptoms do reoccur, normally with the addition of mega-esophagus. At that point, dogs have to be euthanized or they suffer greatly. Petey developed mega-esophagus at nine-and-a-half years old and was put to rest on November 23, 2010. I am very grateful that his owners contacted me before they lost him, as we were able to save DNA from Petey to be sent to Canine Health Information Center. His body was donated to Auburn University.

After Petey was diagnosed, we thought we were clear of any others in the litter developing PN, but unfortunately, that would not prove to be the case. Magnum, WindStar's High Calibur Gunz, started showing symptoms in May of 2002, at 23 months of age. Magnum was tested at UGA and diagnosed with a moderate form of idiopathic PN. Magnum needed hip surgery but never truly recovered from it due to his PN. Some say there is no pain with PN, but I disagree. I believe Magnum was in a lot of pain. He was

ethanized on May 13, 2004 - just shy of his fourth birthday.

Since four of the seven littermates were positively diagnosed with PN, Dr. Kent asked to test the rest of the litter to complete the study. I agreed to have my girl Annie, Ch WindStar's Annie Getcha Gun, ROM, tested even though she showed no signs of having the disease. The tests proved that she was not affected with PN, nor did she ever produce it. The owners of the other two littermates declined testing on their dogs.

After some time, I decided that Annie's offspring should be taken out of the gene pool, and I never bred her again. After doing extensive research, I felt this was what I wanted and needed to do. It was not worth the chance of passing PN on to future generations if Annie could be a carrier.

This was the most heartbreaking experience in my short time of showing and breeding dogs. I hope that writing this article about the health problem I experienced helps others not be afraid to speak out. I share this information with my puppy buyers and try my best to educate them about PN and what I did to remove it from my breeding program. I believe many breeders think that if they speak out about a health problem, it will hurt puppy sales. I can speak from experience about that. It has not hurt me. In fact, most buyers appreciate my honesty. As breeders, we must look beyond individual dogs and concentrate on our love for the breed as a whole. Sometimes we have to make those tough decisions for the sake of the breed's future. If we don't, the Alaskan malamute will suffer in the long run.



Magnum was the fourth and final dog in his litter of seven to be diagnosed with PN. Symptoms began when he was 23 months old, and the disease claimed his life before four years of age.

Photographs of the WindStar dogs courtesy of Edie Thomas.



A Clinician's Perspective

By Marc Kent, DVM, DACVIM (Neurology and Internal Medicine)

Polyneuropathy (PN) in the Alaskan malamute was first described at the 1982 Nordic Veterinary Congress. In 1997, a report was published documenting the disease in eleven affected Alaskan malamutes in the United States. In that report, the average age of affected malamutes was only 14 months old. Both male and female dogs were affected. In nine dogs, clinical signs progressed rapidly over approximately two to four months, to the point that all nine dogs were euthanized because of the severity of their condition. In two dogs, signs remained static for two to three years despite experiencing an initial deterioration. In 2000, a report of 13 affected dogs was presented at the European Society of Veterinary Neurology, in which littermates, the sire and dam, as well as related dogs were studied. Since that report, few studies investigating PN in malamutes have been published in the veterinary medical literature. As a result, our understanding of this devastating condition remains limited. This article attempts to summarize what we know about PN in malamutes and what we can learn from similar conditions in other breeds. Only through a better understanding of PN will we be able to improve our ability to diagnose, determine a prognosis, and ultimately design treatment strategies to help affected dogs.

Despite its use in defining a disease, the term polyneuropathy is more of a pathological description rather than a specific disease process itself. Taken literally, the term polyneuropathy means poly "multiple" and neuropathy "pathology or disease of the nerve." Although the term polyneuropathy is applied to many diseases, PN in Alaskan malamutes shares many commonalities with PN in other breeds of dogs.

In order to fully appreciate the clinical signs, an understanding of the anatomy and function of nerves is necessary. In general, nerves are involved in either creating movements (motor nerves) or in perceiving sensations (sensory nerves) such as touch, temperature, or pain. Regardless of their function, all nerves share a similar structure. Simplistically, nerve fibers can be thought of as insulated wires carrying electricity. An electrical charge or nerve impulse, called a depolarization, begins at one end of the nerve fiber and is conducted to the other end. For motor nerves, the depolarization begins in the spinal cord and is conducted down the nerve to the muscle it innervates and leads to a muscle contraction. Like an insulated wire, each nerve fiber (called an axon) is wrapped in insulation (myelin). On a microscopic level, PN is a degeneration of the axon and myelin. Without the axon, depolarizations are not conducted down the nerve and consequently, muscle contractions become diminished (demonstrated clinically as weakness) or absent (paralysis). Likewise, without myelin, the depolarization conducts more slowly, also resulting in reduced muscle function (weakness).

The clinical signs of PN are dominated by weakness. The degree of weakness reflects the severity of the condition. Mildly affected dogs may show few signs at all.

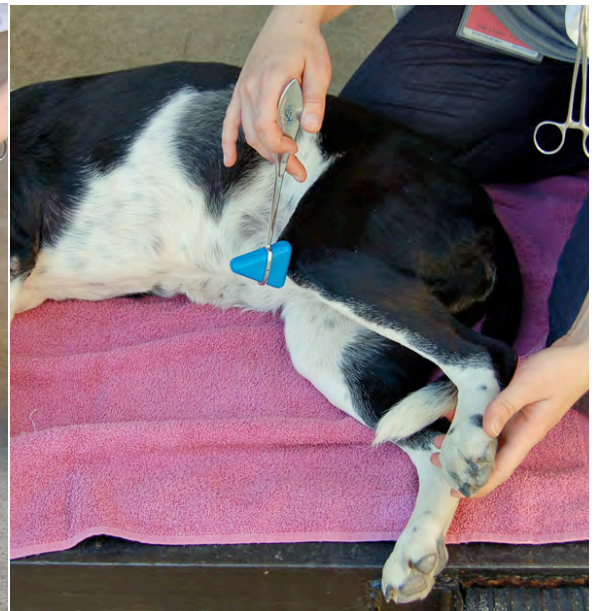
Subtle signs of weakness may be exercise intolerance or a loss of endurance. In more severely affected dogs, the gait may become short-strided and have a stiff appearing, choppy quality. Affected dogs may have difficulty standing up from a prone position. Standing up may appear to be a strenuous activity. When standing, the posture may be crouched. The stance in the back legs becomes plantigrade, which means standing with the hocks flexed so that the point of the hock is closer to the ground. The front legs may be pulled back along the trunk with the elbows rotated inwardly under the chest to help support weight. Severely affected dogs may be only capable of taking a few steps before having to sit or lay down. Often the hind legs are weaker than the front legs. Rarely, the nerves that innervate the larynx are affected. If they are, the character of the bark may be different, and the dogs may cough while drinking or eating. A loud and harsh sound may occur with panting or breathing heavily. Importantly, the clinical signs of PN are not specific. Many conditions can result in similar signs. For example, orthopedic conditions like severe arthritis can cause similar gait changes. One of the most important steps at arriving at a diagnosis of PN is to eliminate from consideration other conditions that may have a similar appearance, by thorough physical, orthopedic, and neurological examinations.

Diagnosing PN begins with a complete neurological examination, including careful assessment of the dog's gait from a side view as well as with the patient walking toward and away from the examiner. Position sense (proprioception) is then evaluated by tests called postural reactions. One common test is "knuckling," where the dog's foot is turned over so that the dog bears weight on the top of the foot; proprioception is gauged by how quickly the patient turns the foot over to the proper position. In some dogs with PN, the foot may be replaced in a normal position slowly or not at all. The most important finding on the neurological examination is reduced or absent reflexes. In the hind legs, the two main reflexes that are assessed are the patellar (knee jerk) and the flexor-withdrawal. As in humans, the patellar reflex is elicited by tapping on the patellar tendon with a reflex hammer and seeing that the knee extends (leg kicks out). The flexor-withdrawal test is performed by pinching the toes and seeing that the patient withdraws the limb by bending the limb at the hock, knee, and hip. As the dog withdraws the limb, the examiner holds the limb to assess the strength and completeness of the withdrawal. Often, dogs with PN do not flex their hock with appropriate strength and have a diminished patellar reflex. In the front legs, only the flexor-withdrawal reflex can be reliably assessed. Additionally, the patient may have muscle atrophy (muscle wasting). Given the thick coat of a malamute, it is imperative to palpate up and down the limb to feel for muscle loss, as atrophy can be easily missed by visual inspection alone.

As alluded to above, a diagnosis is established by initially ruling out other conditions. Basic blood work, including a complete blood count, chemistry profile, and urinalysis should be performed in all dogs displaying

Left: “Knuckling” is a way to evaluate position sense. A normal dog quickly returns the foot to its proper position, but a dog with PN may not.

Right: Tapping on the patellar tendon with a reflex hammer causes the leg to kick out in a normal dog, but reduced or absent reflexes are often seen in PN.



clinical signs of weakness. Based on results, more specialized tests may be needed to exclude other disease processes. In dogs with PN, the blood work should be normal. In older dogs or those that display trouble breathing or swallowing, X-rays of the chest cavity may be warranted.

Unfortunately, there is no gold standard or definitive test to diagnose PN. However, one helpful diagnostic tool is electrophysiological testing called electromyography (EMG) and direct evoked motor nerve potentials. As their names suggest, they are tests that directly assess the “electrical” function of the muscles and nerves. The tests are performed with the dog under general anesthesia using small needles, like those used for acupuncture, which are inserted into the muscles and nerves to record electrical activity. EMG assesses the muscle. The classical EMG findings in PN are the observation of muscles that spontaneously depolarize (show electrical activity) called fibrillation potentials or positive sharp waves. With direct evoked motor nerve potentials, the function of the nerves can be directly assessed. In dogs with PN, the direct evoked motor nerve potential shows a reduction in the number of functional nerve fibers, as well as a decrease in the speed at which nerve fibers conduct electrical activity.

One eye-opening experience for me working with some affected malamutes has been uncovering electrophysiological abnormalities in malamutes that did not display any clinical signs of PN! This has also been observed by others. Another important diagnostic step is a microscopic evaluation of a nerve biopsy specimen. Nerve biopsies do carry some inherent risk of worsening the degree of weakness. However, if performed by a skilled veterinarian using proper technique,

nerve biopsies can be done with little risk to the patient. Microscopic evaluation reveals a degeneration of the axon (nerve fiber) and loss of myelin (insulation) resulting in an overall loss in the number of nerve fibers. Often, the ends of the nerves toward the extremities undergo more severe degeneration.

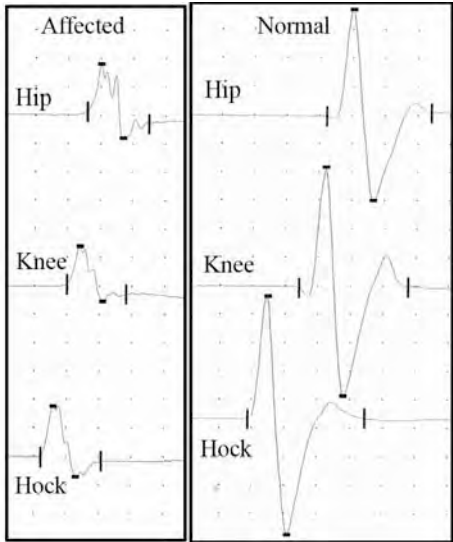
Unfortunately, a proven form of treatment is not available. Treatments with corticosteroids or other immunosuppressive drugs have failed to help affected dogs. In other breeds with PN, several nutraceuticals (nutritional supplements thought to have medicinal effects) such as L-carnitine, CoEnzyme Q10, and B vitamins have been advocated. To date, controlled studies have not been performed, and success stories are limited to anecdotes related to an individual dog's success. In severely affected dogs, the progression of signs may continue to the point where the dog is unable to walk and sadly often leads to euthanasia.

Without a doubt, more questions remain than are answered. Likely, PN in malamutes demonstrates as a spectrum of disease spanning from those severely affected to those with milder signs to even individuals that have abnormalities on electrophysiological testing yet appear clinically normal. What will it take to find answers to questions like how is the disease passed from one generation to the next? Are there genetic markers through which blood tests can be designed so patients at risk can be identified? Are there novel therapeutic interventions that can help affected dogs?

In humans, a polyneuropathy syndrome called Charot-Marie-Tooth syndrome (CMT; named after the physicians who first described the condition) encompasses a wide range of clinical disorders that may possess close similarities to PN in dog breeds. It is now



Top: Nerve conduction testing is performed under anesthesia. Tiny needles (compare to the penny pictured above the dog) are used to stimulate the nerve at the hock, knee, and hip. After stimulation, the resultant muscle contraction is measured.



Bottom: Notice how the tracings from the affected malamute are smaller and irregularly shaped compared to the normal dog's tracings. These findings are consistent with the loss of nerve fibers observed in polyneuropathy in malamutes.

possible to use genetic testing to help diagnose the various forms of CMT in people.

Like PN in malamutes, a more recent description of PN in the Leonberger dog has been reported. The disease process in the Leonberger dog appears to be genetic and as a result is passed from the sire and dam to their offspring. The basis of the inheritance is still under debate but may represent an X chromosome linked condition or may be a recessive condition. The importance of these reports lies in the lessons that can be drawn from the study of Leonbergers and applied to answering questions with PN in malamutes. What will it take? As in the Leonberger, the first step is identifying the prevalence of the condition (the number of affected dogs in the population). Since some dogs may appear normal yet be affected based on electrophysiology, it is necessary to evaluate both

healthy appearing and clinically affected dogs. From such preliminary studies, it may be possible to identify potential genetic linkages. Now with the canine genome (DNA) having been sequenced, modern molecular genetic tests such as single nucleotide polymorphism (SNP; pronounced "snip") may help uncover a genetic defect that either causes or predisposes to the development of PN in malamutes. Once thought impossible, these state of the art diagnostic tools are being used today to study other genetic conditions in some breeds.

Likewise, until affected individuals can be identified, critical evaluation of novel therapies cannot be pursued.

In the end, until there is clarity as to the genetics involved and improvements in the diagnostic tests used to identify dogs with PN, recommendations on how to truly eliminate PN will not come to fruition.

A Research Perspective

Representing the Scandinavian Polyneuropathy research group, this article is written by Professor Lars Moe and Associate Professor Karin Hultin Jäderlund of the Norwegian School of Veterinary Science, Department of Companion Animal Clinical Sciences in Oslo, Norway.

In 1979, the first cases of polyneuropathy in Alaskan malamute dogs were diagnosed in Norway, and in the early 1980's, a number of Alaskan malamute dogs in Norway were affected. They were all closely related. Pedigree analysis indicated that the disease could be inherited, and an autosomal, recessive mode of inheritance fitted the phenotypic findings in the litters examined.

A test mating was planned and performed in 1983 to test the hypothesis of a recessive genetic transmission. The mother of the litter was an affected but well-functioning bitch. Of the six dogs in the litter, four (three males and a female) became affected with PN.

The onset of clinical signs varied from seven to 18 months of age. Dogs of either sex were affected. The early signs were slowly progressive limb weakness, which might develop into tetraparesis (weakness in all four



An Alaskan malamute litter resulting from a test breeding for inherited polyneuropathy.

legs), and recumbency (inability to stand). Coughing and regurgitation were also seen. The degree of muscle atrophy and weakness varied during the clinical course of the disease and also from dog to dog. If the dog was not euthanized while in the recumbent stage, improvement usually occurred without treatment after some weeks. Some dogs lived several years after regaining the ability to walk. Deterioration was commonly seen later in life in affected dogs.

The Alaskan malamute clubs in Norway and Sweden were advised to follow a breeding strategy, based on the hypothesis that had been strengthened by the test mating results; namely that polyneuropathy looked to be inherited in a recessive manner. The breeding strategy seemed to work, and cases of PN were not diagnosed in Norway and Sweden for about 20 years.

However, during the last few years, we have again seen this disease in the Alaskan malamute breed. There have recently been cases in Norway, Denmark, and Sweden, and anecdotally also in Finland. We are again now doing research spanning all the Scandinavian countries, where our aim is to understand the pathogenesis of this condition, its cause(s), the prognosis of PN, and possibly to identify any preventative or therapeutic measures. The research is conducted in collaboration with the national Alaskan malamute clubs.



An Alaskan malamute affected by inherited polyneuropathy, showing the abnormal gait that is seen in some cases.

The Scandinavian Polyneuropathy research group consists of researchers from Denmark (Professor M. Berendt and Professor M. Fredholm), Norway (Professor I. Bjerkås, Professor A. Espenes, Professor L. Moe, Associate Professor Ø. Stigen and Associate Professor K Hultin Jäderlund), and Sweden (C. Rohdin, Dipl ECVN).

About the Authors

Todd McGuire loves Alaskan malamutes and became a first-time owner of mal Mia in July 2009. After losing Mia at just 11.5 months of age due to PN, Todd created a Facebook group "Alaskan Malamutes with Polyneuropathy," and is now a contact in Australia for anyone wanting to find out about polyneuropathy in malamutes. He also joined the Alaskan Malamute & Siberian Husky Social Club of Queensland and the Alaskan Malamute Club of Victoria to stay involved in the breed. Todd serves as Treasurer for the Northern Exposure Gig Racing Club, Queensland's dog sledding club.

Edie Thomas of WindStar Alaskan Malamutes has been showing and breeding Alaskan malamutes for over 12 years. A member of the Alaskan Malamute Club of America since 1999, she served as the Southern Area Newsletter Reporter for many years. A native and current resident of Lawrenceville, Georgia, Edie has been a member of the Lawrenceville Kennel Club since 1998. She invites you to contact her with any questions about polyneuropathy at Windstarmals@charter.net.

Marc Kent, DVM, DACVIM serves as one of three full-time veterinary neurologists on staff at the University of Georgia College of Veterinary Medicine, considered to be the leading veterinary college in the US for neurology. Marc received his Bachelor's degree from Columbia University in New York, followed by his Master's and Doctor of Veterinary Medicine degrees from Tufts University School of Veterinary Medicine in Massachusetts. He completed his internship in Small Animal Medicine and Surgery at the Animal Medical Center in New York, followed by his residency in Small Animal Internal Medicine back at Tufts University School of Veterinary Medicine. He then completed a second residency in Neurology at the University of Georgia in Athens, and now

is an Associate Professor at the UGA College of Veterinary Medicine. He is a Diplomate of the American College of Veterinary Internal Medicine, in the fields of Internal Medicine and Neurology.

Currently a Professor of Companion Animal Internal Medicine at the Norwegian School of Veterinary Science in Oslo, veterinarian **Lars Moe** graduated from the Norwegian School of Veterinary Science in 1977. He earned his PhD in 1986, and has worked as a researcher and clinical teacher for many years, in both large and small animals. Serving on a wide range of scientific and administrative committees both nationally and internationally, he also was elected President of the European (International) Society of Veterinary Nephrology and Urology from 1997 - 1999, and Past-President since 2000. In 2001, Lars was elected as the Rector and Head of the Norwegian School of Veterinary Science, a position he held for 9.5 years (2002-2010) - the maximum period allowed! He has also served as Head of the Department of Small Animal Clinical Sciences at Copenhagen University. Lars' main research interests have been small animal oncology, neurology, and nephrology and urology.

Veterinarian **Karin Hultin Jäderlund** holds a position as Associate Professor at the Small Animal Section of the Department of Companion Animal Clinical Sciences, at the Norwegian School of Veterinary Science in Oslo, Norway. She is a Veterinary Neurologist (Diplomate of the European College of Veterinary Neurology) and has a PhD degree from the Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, in Uppsala, Sweden. Her doctoral thesis is about an inherited neurological syndrome in Golden Retrievers, a disease that has some similarities to polyneuropathy in Alaskan malamutes.