

# *F* THE IRISH WOLFHOUND FOUNDATION *Focus*



Winter 2006 - 2007

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#### *Focus*

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## **PNEUMONIA STUDY UPDATE**

*by Margret L. Casal, DVM, PhD*

### **Chronic Pneumonia, Rhinitis, and PCD: Are They Related?**

**A** syndrome of rhinitis in Irish Wolfhounds was first described by Wilkinson in 1969. The primary symptom is a watery nasal discharge that progresses to a purulent or even blood-tinged discharge. The discharge usually is present at birth and may be chronic or recur at short intervals.

Many dogs develop a chronic moist cough and typically die at a young age due to pneumonia. Most often only a part of a litter is affected and the rest, even when housed with affecteds, are healthy. This disease has apparently been present in the Wolfhounds for decades and has not disappeared. For years it was thought to be caused by a virus or by an immune deficiency (an incomplete immune system that leads to frequent infections). No one has ever been able to isolate a virus. More recently, we have been able to perform studies that look at the immune system in great detail and have not found any abnormalities, suggesting that there is a different cause for rhinitis.

We have performed studies examining the function of the cilia, the fine hair that line the airways (nasal passages, trachea and larger bronchi). The cilia are part of the mucociliary apparatus that is responsible for transporting particles (viruses, bacteria, dust) out of the lungs, similar to an escalator. We have been able to observe the cilia of normal and affected Wolfhounds in action, we have performed electronmicroscopic evaluation of the cilia from Wolfhounds with rhinitis, and we have done mucociliary clearance studies. We have been able to show that the cilia from affected Wolfhounds do not beat in a coordinated fashion and that the cilia are not lined up in parallel

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# 2006 Irish Wolfhound CERF Clinic Report

Dr. Amy Hunkeler DVM, DACVO, provided veterinary ophthalmic services to the Irish Wolfhound Specialty Show on Thursday afternoon of the event. Nine owners and handlers took advantage of the opportunity to have eyes checked for possible genetic abnormalities.

Of the 26 eyes checked, 10 were normal and 16 had abnormalities. By far, the most common abnormality was distichiasis—distichia are normal lashes growing from an abnormal location. Twelve eyes were identified with distichia and none of the dogs showed any clinical symptoms of distress from the hairs. Iris cysts or uveal cysts were noted in 3 eyes. Cysts tend to be an incidental finding in the IW, but are monitored more closely in the Golden Retriever and Great Dane because of their association with inflammation and glaucoma. Cataracts, opacity of the lens, were identified in 2 eyes of different dogs and were of unknown significance. If 'significance unknown' is marked, then the dog will still be eligible for a CERF number. Finally, one dog was noted to have a spot on the retina, which did not appear to be a heritable lesion and was marked accordingly. All in all less than 1% of the IW's at this specialty show were examined. Historically we only have a small percentage of the IW's in the CERF database. The goal

of CERF is to examine the eyes of breeding animals so an informed decision can be made about future pairings and reduce or eliminate heritable eye diseases from the breed.

For those unfamiliar with the process of getting your dog 'CERF'ed, here is a crash course. First, please bring your registration papers to the CERF clinic; we need to collect the AKC number and the permanent identification number (tattoo or microchip) and the dog's date of birth. When you arrive, the veterinarian or a designated person, will apply dilating drops to both of the dog's eyes. It takes about 20 minutes for the medication to dilate the eyes, which is plenty of time for you to fill in the official form.



The eyes will remain dilated for 6-8 hours and most dogs will partially close their eyes if they find the light uncomfortable. Once the eyes are dilated, the dog is brought to a darkened room for eye examination with an indirect ophthalmoscope and a slit beam ophthalmoscope. It is necessary for the dog to stand or sit steady while the examiner looks at the structures of the eyes—it takes but a minute if the patient is patient! The examiner is hoping to find normal eyes, but is looking for lesions known or suspected to be heritable in the breed. When the examination is complete the ophthalmologist records his or her observations, signs the form and passes you a copy. On the back of the copy are instructions for submitting the form to the CERF (Canine Eye Registry Foundation) to receive a certification number. The registered animal will receive a number from CERF if the eyes were marked normal or if lesions were found to be unrelated to genetic heritage. Data in CERF registry can be accessed at the CERF website and searched by dog's name, registration number or CERF number. It is important to have the eyes examined every year to identify heritable problems, some conditions do not occur until later in life and we want each breeder to be as informed as possible about the animals under their care. Please consider participating in a 2007 CERF clinic.

## Pneumonia Update

*cont'd. from page one*

on electronmicroscopy. The mucociliary clearance studies demonstrated that the particles do not clear the lungs at the expected speed, in fact, they often did not move at all. All of these results are highly suggestive of primary ciliary dyskinesia (PCD).

There is a second fairly common syndrome in the Wolfhounds, which we call chronic pneumonia. These dogs have pneumonias as early as 6 months of age, but never showed any signs of rhinitis early in life (remember: rhinitis is just a symptom and not a cause or diagnosis). Many of the affected dogs will have chronic recurrent pneumonia, which as most owners/breeders know is not readily apparent to the "untrained eye." Affected Wolfhounds (as all Wolfhounds) are of a very stoic nature and the only sign of illness may be the reluctance to lie down or loss of appetite. In general, the affected dogs do not cough at first, but there are often already severe changes present on lung radiographs (X-rayed). The dogs need to be treated immediately and our recommendation is to give an initial dose of Baytril and ampicillin IV and then switch to azithromycin orally. Furthermore, couple-steam inhalation (nebulization) should be performed and possibly a mucolytic agent given. For complete details, see your veterinarian or contact us at the University of Pennsylvania.

What makes this chronic pneumonia interesting? It is quite possible that this is an incomplete form of PCD. We and others have established that

PCD is inherited as an autosomal recessive trait, meaning that it takes two copies of the "bad" or PCD gene to cause the disease in the offspring (one copy came from the mother and one from the father). Two copies make the cilia virtually

non-functional. On the other hand, if a dog were to inherit only one PCD gene and a normal one from the other parent, then it is possible that half of the cilia function normally and the other half doesn't. As the dog grows older, the mucociliary system "wears out" (gets overwhelmed) and the normal cilia cannot keep up. The result is pneumonia. At this time this is just a theory and has yet to be proven.

### Treating CHRONIC PNEUMONIA

There are some dogs that require maintenance antibiotics all the time. These dogs may be kept on first generation cephalosporins or Clavamox for maintenance. Third generation cephalosporins, or azithromycin (Zithromax), are reserved for acute infections with clinical signs. Occasionally, resistance to some of these antibiotics develops, and it may be necessary to change to a combination of enrofloxacin (Baytril) and ampicillin. Doses should be determined by your veterinarian.

Dr Casal uses the following protocol in a university clinic setting:

**Cephalexin:** 22-30 mg/kg twice daily for dogs who have had chronic pneumonia until the next pneumonia occurs, then

**Zithromax:** 5-10 mg/kg. To prevent nausea from the Zithromax, the first day's dosage is 5mg/kg, then increase the dose to 10mg/kg from the second day on. Continue the high dosage amount of Zithromax until 5 days after the last clinical signs have disappeared.

So what are we doing to get to the bottom of all of these diseases (or this complex of rhinitis/chronic pneumonia)? Cilia have the same basic structure as do sperm tails. This turns out to be handy, because it is easier and less traumatic to obtain sperm tails than it is to obtain tracheal biopsies (the seat of the cilia). The plan is to compare the proteins present in sperm tails from healthy wolfhounds compared to affected hounds. Jennifer Scheidt, now a veterinary student, had been working on examining the immune system in PCD Wolfhounds as a technician. She has now just completed developing a method to clearly separate sperm tails from the sperm heads. This was a time consuming task, as the preparation has to be completely free of heads, because this would contaminate the analysis. With this method in hand, we will submit the various samples for protein comparison and anticipate that a difference in proteins will become clear. The differences will be analyzed and we should be able to deduct which gene is causing the disease. As a last step (not always an easy one), we will sequence the gene to find the mutation. Once we have the mutation, we will be able to offer a genetic test.

Some breeders of Irish Wolfhounds, in the attempt to improve longevity and reduce the incidence of hereditary health problems, seek to breed apparently unrelated individuals in hopes of “breeding away from” problems. Some of these breeders rely heavily on computed coefficients of inbreeding. An inbreeding coefficient is a mathematical computation showing the number of common ancestors in the background of a given animal. The lower the number; the fewer the common ancestors and, thus in theory, less chance of producing defects. Although this approach would initially seem sensible, its ability to totally eliminate genetic problems is limited by two factors: the small size of the Irish Wolfhound gene pool, and the pervasive nature of certain genetic health issues.

No matter what you do, you can't make the Irish Wolfhound gene pool bigger. It is small. Period. Although the Irish Wolfhound is an ancient breed, its reconstruction is relatively recent, and it involved a small number of animals. As late as the early 1900s, outcross breedings have appeared in IW pedigrees. Felixstowe Bob (Great Dane) x Felixstowe Lufra (Scottish Deerhound) produced Felixstowe Sheelagh, who appears in many modern pedigrees. Also, there were two bottlenecks: the first during World War I, and another during World War II, when very few breedings occurred. As recently as the 1990's, the gene pool was further limited by the use of several popular sires, each of whom sired 30 to 40 litters. In addition, a few health scares have further reduced the gene pool by removing animals that were known or suspected carriers of those conditions. These factors in combination mean that we have had a small isolated population for about 25 generations. The number of possible genetic combinations is limited solely by mathematics. So, how does this actually look on paper, and what does it mean?

Let's look at two examples, both of which have the Felixstowe outcross mentioned above in the pedigree. The first example is a bitch whelped in September of 2005, that we will call “Yearling.” “Yearling” was produced by a loose inbreeding (linebreeding). The second breeding example is a “what if” breeding planned for 2007 that we will call “Wishful Thinking.” This potential breeding is what some would call an



Yearling has a pedigree with 30 generations, but has only 18 complete IW generations because Felixstowe Bob and Felixstowe Lufra appear in generation 19. In an 18 generation pedigree, there are 524,286 positions. For “Yearling,” of the 524,286 positions, there are only 854 unique individuals. The inbreeding coefficient at 6 generations is 8.72 and at 10 generations it is 18.16 for this IW. The average inbreeding coefficient at 10 generations for Irish Wolfhounds is about 15. The breeding was done to establish the type that the breeder wants. The breeding of “Yearling” will more than likely accomplish the goal set by her breeder.

For “Wishful Thinking,” the inbreeding coefficient at 6 generations is 0.00, and for 10 generations it is 6.15. Since both of these figures are lower than the corresponding ones for “Yearling,” this looks like an outcross—but it really isn't that far from the last bottleneck. For “Wishful Thinking's” pedigree, there are 2,046 positions, but only 709 unique individuals at 10

generations. At 16 generations there are only 1396 unique individuals in 131,070 pedigree positions. “Wishful Thinking's” breeding was proposed to increase longevity and to decrease fatal diseases (such as cardiomyopathy and osteosarcoma) by the sole use of an outcross (low inbreeding coefficient). However, “Wishful Thinking's” proposed breeding is just that – wishful thinking!

Even though “Wishful Thinking” has more unique ancestors than “Yearling,” there is still a strikingly limited number of unique animals in the pedigree. The bottom line is that the number of genes in the gene pool has already been established, and this fact limits just how distant two Irish Wolfhounds can be from each other. All wolfhounds are related or they would not be wolfhounds. Therefore, what may at first glance appear to be an outcross is in reality more of a very loose inbreeding.

Some of the genetic diseases that affect wolfhounds—epilepsy, liver shunt, PRA, and PCD—appear in highly increased numbers in some families. This suggests a gene (or genes) specific to only animals descended from a certain individual or individuals who are carriers. Heart disease and osteosarcoma, on the other hand, show up throughout the breed, rather than in small clusters, indicating that these problems are pervasive rather than isolated. At the present time, at least, outcrossing will be ineffective at totally “breeding out” these diseases without making the gene pool even smaller and thereby also threatening type. The hereditary aspects are in the gene pool to stay.

Having said that, there are some things that breeders can and should do. A breeder can research the age of onset of these diseases in the bloodlines of prospective mates. There are different types of both cardiomyopathy and osteosarcoma, and the breeder can opt to use pedigrees that show animals with the latest-onset or most treatable forms of these diseases. For instance, some types of cardiac disease occur relatively late in life, respond to treatment, and progress slowly, while others occur much earlier and progress to fatality very quickly. Similar variation has been observed for osteosarcoma, though it remains a terminal diagnosis in the Irish Wolfhound. Lastly, regular testing needs to be done not only for the health of your kennel and of our breed,

# data from the heart

by mariellen dentino, md

## the lifetime cardiac study

2007 marks the sixth year of the Irish Wolfhound Lifetime Cardiac Study. The health histories we are compiling on each wolfhound, together with the IW DNA Blood Bank containing samples of blood donated by many study hounds, will continue to provide significant information for our breed. In addition, researchers will be able to correlate specific health issues with genotypic information, providing additional benefit for future studies. We are already seeing “spin-off” studies proceeding as a direct result of some of the findings which needed more exploration.

Although we are a long way from completing the study, it's interesting to look at the yearly statistics compiled from EKG results and owner-completed questionnaires. The data provides us with a window onto the health of the Irish Wolfhound in the U.S.

## cardiac health

In overall cardiac health, we have seen a slight increase in the number of wolfhounds with atrial fibrillation. The percentage widely used to estimate overall wolfhounds with atrial fibrillation is 11%, but our data indicates 12% of IWs have this problem. The total number of IWs with abnormal EKG results is 34.7%, which also slightly exceeds previous estimates. Of dogs entering the study with normal EKGs, some 15.4% have developed some EKG abnormality. 5.3% of these abnormalities are atrial fibrillation.

As we know, the percentage of wolfhounds with atrial fibrillation increases with age. Approximately 16% of dogs between the ages of 6 and 8 years are in atrial fibrillation. This is far fewer dogs than the 30% of the 107 dogs aged 6 or over that were tested by Dr. Harpster, and may reflect more diversity in the population participating in the Lifetime Cardiac Study than in the population Dr. Harpster sampled. Of the wolfhounds with atrial fibrillation, 70 have died since the study began. Forty-two of these deaths were from heart disease.

Sixty-two percent, or 5.2% of all wolfhounds in the study, have first-degree AV (atrio-ventricular) block. This benign condition is secondary to delayed/prolonged electrical conduction through the atrioventricular node. First-degree AV block may be a congenital abnormality; however, it is very likely an acquired condition, secondary to benign fibrosis of the atrioventricular node. Unless first degree AV block progresses to second- or third-degree AV block, it does not warrant any concern.

Thirty-seven dogs have atrial premature contractions (APCs). This abnormality may be a precursor to atrial fibrillation, so follow-up on these wolfhounds will help

us determine whether or not this is the case. APCs alone do not require treatment.

Fifty-two IWs had some tachycardia (abnormally fast heart rate) considered significant by the veterinary cardiologist. Increases in the R-wave amplitude were seen in 154 dogs.

Ninety-five dogs have had premature ventricular contractions (VPCs) on one or more EKG. Fifty-eight of these dogs had no other EKG abnormality. Ventricular premature contractions (VPCs) were recorded in 89 wolfhounds. VPCs in wolfhounds appear to be more benign than they are in Boxers and Dobermans—in these two breeds, they can signal a condition which frequently leads to sudden death. Nonetheless, VPCs are often present in wolfhounds with impaired cardiac function as well as those with no heart disease. The IWF is funding a study to determine what the significance of VPCs is for our breed.

One interesting note is that the average age of dogs who have died or been euthanised for cardiac disease is 7.24 years, which is actually older than the average age of death (all causes) recorded thus far in our study, 6.55 years.

## mortality

Mortality statistics appear to be very similar to those previously reported for our breed. Average age at death is 6.52 years, which is slightly improved from previous statistics. 270 dogs have died thus far. Cancer (all kinds) is the most common cause of death in wolfhounds, accounting for 28% of deaths. Of cancer deaths, 70% were from osteosarcoma. Average age of death from cancer is 6.45 years.

Heart disease was reported as the second leading cause of death, with 21% of deaths, and an average age

of 7.25 years. for deaths from this cause. Third leading cause of death is bloat, at 5.2%, with average age of death 6.1 years. Approximately 4.4% of deaths are caused by euthanasia from rear-paralysis, with average age of death at 8.4 years. Respiratory disease, with the majority from pneumonia, was next at 4.1% and average age of 5.7 years. Renal failure was found in 2.2% of wolfhounds with average age of 5.9 years. Other causes of death were listed as unknown (3.7%, average age 5.98 years) and sudden death (3%, average age 5.9 years).

In human actuarial science, remaining life expectancy at a given age increases, so that the life expectancy for a fifty-year-old is greater as the individual ages. This is exactly what happens with our wolfhounds. Although, on average, the age of death is 6.55 years, for wolfhounds who reach the age of five, the age-adjusted life expectancy is an additional 2.6 years, so average age of death is 7.6 years.

*Wolfhounds who live to be 5 have an age-adjusted life expectancy of an additional 2.6 years, so average age of death for this group becomes 7.6 years. The average age of death for all hounds is 6.55 years.*



# Coat Color



by Jill Richards Bregy  
Wildisle, Reg.

I have long been interested in the effects of coat color on eye color and pigmentation. I met with a breeder of Greyhounds and Afghans to discuss the color dilution in one of the breeds and its effect on eye color. If puppies appear with different green, blue or yellow eye colors, what coat colors influence that and does it always relate to coat color?

In these two breeds, like ours, the standard calls for a dark eye. However, that is difficult, if not impossible, to maintain in dogs that are blue-factored. In one particular instance, a red brindle was bred to a blue brindle. What are the possible outcomes?

In Dachshunds, breeders have incorrectly bred double dapples to same and produced no eyes, blue eyes and small eyes. In reds, breeding for several generations of these colors together and then having an inbreeding resulted in green eyes and Dudley noses. In Shelties, it has generally been the practice to breed blue merles to black, not to sable. Recently blues have been bred to sables, producing something called a sable merle, which I am told can be distinguished at a young age but, when older, looks like a sable. This sable merle, in turn, bred to a sable can and has produced dogs with one blue and one brown eye. Not something being received well by the responsible breeders!

In our breed, we should be alert to eye color and/or pigmentation, as dogs with questionable pigmentation bred incorrectly can result in still worse pigmentation and/or green or blue eyes. This blue factor can appear in what seems to be a grey; in reds brindles; and in wheaten brindles but with an underlying white with a black brindle, and probably others as well. Eye color, rather than being a dark gold or brown, can be a washed out yellow color or amber, which is a color that occurs in the blue brindle or blue fawn greyhounds. These dilute eyes appear flat, rather than having depth to them, as you look into them.

What can happen when large amounts of white appear in the coat? In Boston Terriers, excessive white sometimes comes in conjunction with blue eyes, which can be related to other health issues.

It is interesting to note that in Deerhounds, under "Color," it says, "White is condemned by all authorities but a white chest and white toes, occurring as they do in many of the darkest-colored dogs, are not objected to, although the less the better for the Deerhound is a self-colored dog. A white blaze on the head or a white collar, should entirely disqualify." I bring this up as one might wonder if the early authors of this Standard might be telling us something about the effects of excessive white markings and the problems this might create if left unattended in a breeding program.

In Rhodesian Ridgebacks, excessive white is also addressed—and this in a breed that calls for light wheaten to red wheaten in coat color. In Otter Hounds, the Standard says that "eyes are dark, but may vary with the color of the hound," and, additionally, under color, "Any color or combination of colors is acceptable. The nose should be darkly pigmented, black or liver, depending on the color of the hound."

The late Brig. Gen. A. W. DeQuoy, who was a master historian in our breed and a man with a brilliant mind, discussed eye color by saying that while the "List of Points in the Order of Merit" called for a dark eye as No. 16, it did not say brown....arguing that a gold eye could still be dark. An interesting statement, as this is exactly what some Greyhound breeders have said....thirty years later.

A study of our Irish Wolfhound Standard, along with the study of other breed standards, can only enhance our ability to truly understand the dog in front of us. You must have a vision of the ultimate dog in your mind, and this can only be done if you actually understand all of the parts, and how they create form and function.

A desire to learn and understand....not just to breed....is what sets the breeders and protectors of the breed apart from those just breeding dogs and calling themselves breeders. The issue of color dilution is just one of many issues that need to be addressed in a breeding program.



# New IWF-Sponsored Study Evaluating VPC Arrhythmia in Irish Wolfhounds

The Irish Wolfhound Foundation is funding a new research study by veterinary cardiologist Dr. William D. Tyrrell, Jr., and resident cardiologist Dr. Gina Pasioka as chief investigators. The goal of their research is to characterize ventricular premature contractions (VPCs) in wolfhounds who have no underlying heart disease.

Ventricular premature contractions (VPCs) are heart rhythm disturbances that interrupt the normal heart rhythm and cause an irregular beat. While one or two isolated VPCs can be harmless, when they occur very often or repetitively, they can lead to a very serious condition called ventricular tachycardia. For this reason, all dogs with VPCs should be closely monitored.

In Boxers and Dobermans, VPCs can signal a condition which frequently leads to sudden death. In wolfhounds, VPCs appear to be more benign. However, VPCs have been found in wolfhounds with impaired cardiac function as well as those with no heart disease.

Twenty wolfhounds will be evaluated. Drs. Tyrrell and Pasioka will collect data from echocardiograms, electrocardiograms, bloodwork, thyroid tests, and tick panels. They will then have each dog wear a holter monitor to record a 24-hour continuous EKG.

This study will give us a much better idea of whether or not VPCs are truly benign in wolfhounds, or whether they can indicate more serious problems.

If your IW's EKG has had VPCs please consider entering your IW in this study. Contact Kathy Wilson at (540) 592-3705 or [glendorling@earthlink.net](mailto:glendorling@earthlink.net).

## Focus on Health

### Neurohormone Levels in Wolfhounds with Atrial Fib Are Focus of Purdue Research

Dr. Henry Green, III, veterinary cardiologist from Purdue University, led a team from the Purdue University School of Veterinary Medicine to characterize cardiovascular neurohormone levels in wolfhounds with primary atrial fibrillation (atrial fibrillation with no underlying heart disease). For this study, Dr. Green evaluated 32 Irish Wolfhounds to try to characterize what types of changes —if any—occurred in neurohormone levels in wolfhounds with AF.

Atrial fibrillation in the absence of underlying cardiac disease is referred to as primary atrial fibrillation. This disease results in decreased ventricular filling secondary to loss of atrial contraction and subsequent decrease in cardiac output.

Of the 32 IWs participating in this study, 15 (47%) had primary AF and 17 (53%) were in sinus (normal) rhythm. All cardiovascular medications were discontinued for at least two

weeks prior to entry into the study. The neurohormones that were measured included plasma renin activity (PRA), brain natriuretic peptide (BNP), atrial natriuretic peptide (ANP), epinephrine (EPI), norepinephrine (NE) and aldosterone (ALD). Echocardiograms were performed on all study dogs.

The study concluded that Irish wolfhounds with primary AF have significantly larger left atrial diameters and significantly higher plasma ANP levels than Irish Wolfhounds in sinus rhythm. ANP (atrial natriuretic peptide, or atriopeptin) is involved in the control of body water and sodium. It is released by cells in the atria of the heart, in response to signals of raised blood pressure, and it acts to reduce the water and sodium loads on the circulatory system, helping to return blood pressure to more normal levels. One of the reasons it is secreted is in response to stretching of the atria. One of the causes of stretching can be atrial fibrillation. The overall effects of ANP release is a reduction in blood volume, and therefore central venous pressure, cardiac output, and arterial blood pressure. It increases renal sodium secretion and excretion. It also increases lipolysis. ANP's overall effect is to counter the blood pressure-raising effects of the renin-angiotensin system.

These findings enabled Dr. Green and his co-investigators to conclude that primary AF in Irish wolfhounds is associated with markers of increased left atrial pressure, and possibly ventricular dysfunction. Further studies will be necessary to determine the chronic effect of primary AF on ventricular function.

# IWF Echocardiogram Clinic & EKG Testing at the 2007 IWCA Specialty

On Thursday and Friday, April 12 and 13, the Irish Wolfhound Foundation will sponsor an echocardiogram clinic open to all Irish Wolfhounds over the age of two. The fee for each echocardiogram is just \$75, which is much less than the usual \$250 - \$400 charged for this service.

Dr. William D. Tyrrell, Chesapeake Veterinary Cardiology, Leesburg, VA, will be performing echoes for us on the show grounds of the IWCA Specialty Show in Parker, Texas. Reservations are necessary, but sign-up sheets will be available at the Health Testing Area on Wednesday, April 11.

In addition to the echo clinic, we will offer electrocardiogram (EKG) and blood pressure screenings and

urinalysis. EKG and blood pressure testing are free to all dogs enrolled in the Lifetime Cardiac Study; there is a small fee for these tests for all other dogs.

Many heart problems, including atrial fibrillation, are first diagnosed in hounds who are totally asymptomatic. It is always a good idea to have your hound checked whenever possible. By the time symptoms are apparent, the dog is usually in heart failure and damage to the heart may be irreversible.

Urinalysis is free, and results are available within an hour of the testing. For more information on health testing at the specialty, please contact Kathy Wilson at [glendorling@earthlink.net](mailto:glendorling@earthlink.net).

## CHECK OUT the NEW IWF "HEALTH TALK"

The Foundation's online health bulletin board is redesigned and is up and running. IW Health Talk is the place to exchange views and information on health questions about wolfhounds. Log on at [www.iwfoundation.org](http://www.iwfoundation.org) and click on IW Health Talk.

## IWF Helps Fund Osteosarcoma Study at University of Illinois

A study evaluating the efficacy of pamidronate, adriamycin, and radiation therapy as palliative treatment for canine osteosarcoma is underway at the University of Illinois in Champaign-Urbana. Pamidronate is a bisphosphonate drug in the same class as Fosamax (alendronate), the drug used in the IW Osteosarcoma Study.

Dr. Timothy Fan and Dr. Louis-Philippe de Lorimier, veterinary oncologists, are chief investigators in this double blind, placebo controlled study. All dogs with osteosarcoma are eligible to participate, but they must be able to travel to the U of I for regular treatments and follow-up visits. Treatment costs and most other expenses are covered by the study; owners pay only a fraction of the actual cost involved.

If you would like more information or are interested in participating in this study, please contact Nancy George of the U of I Oncology Service at (217) 244-7789. All dogs receive radiation and adriamycin in this study. Some dogs will also receive IV pamidronate, while others will receive a placebo treatment.

## Are You In ...or Out?

Think back to the last time you had to tell your vet that you were ready to have your wolfhound put to sleep. Whether your hound was two or ten, it was too soon. It is always too soon.

No matter what the cause of death, we wonder if there was more that could have been done. We have all been there—devastated and disheartened almost to the point of giving up. But in the end, who will be the stewards of this breed if not us? Isn't it time we put our muscle and our money to work for the sake of our hounds—to 'walk the walk,' not just 'talk the talk.' That's why the IWF was originally created. And we—the IWF—are taking steps to ensure that this breed has a healthier future.

You've probably seen the IWF at work. We are the people who do the echo clinics and EKG testing at specialty shows, and who started the DNA bank for genetic research for Irish Wolfhounds. We're the people who are finding and funding health studies of lasting significance to the Irish Wolfhound. If you're reading this newsletter, you care about these projects, too.

The work we do isn't glamorous—in fact, sometimes it's just plain exhausting. But we can't forget that look in a wolfhound's eyes when he's in pain, and he seems to be asking us why he feels that way....

So, when we ask you to join us by becoming a member and making a donation, we want you to know that if there's something that will improve the longevity or quality of life for our hounds, you can count us in. And we're in it for the long haul.

But what about you? Can we count you in, too?



## GENE POOL

*cont'd. from page 3*

but for the sake of the individual hound. Problems like atrial fibrillation are nearly always a surprise diagnosis, and early treatment can positively impact the course of this disease and the comfort of your hound.

Selecting a proper mating is undeniably the ultimate challenge each breeder faces. But the Irish Wolfhound is worth it.

# Miles and Timothy Go to Camp

by Frances Abrams

Frankly, until two years ago, I never made the connection between dog camps and Irish Wolfhounds. Always thought of dog camps as places where city folks took their hyperactive herding and mixed breeds to wear off some energy. My hounds live in the country and never seemed in need of special entertainment beyond the occasional visit to town. A few of them have enjoyed lure coursing and they put up with my dog shows, but none of them ever clipped out an ad from Bark and shoved it under my nose.

It was Rebecca, my friend and dog sitter, who got us started. She works at “Dog Skills Adventures” Camp every year. When my puppies were about 5 months old, it was her idea to have a one-year reunion for the entire litter at the dog camp where she works. As it turned out, I was the only person able to make it to “Dog Skills Adventure,” but that first year I managed to get almost every dog I owned to camp for a day at least.

The puppies, Timothy and Tabitha, loved it. Tim and Tabby had good lure coursing runs in the morning. Tim showed a surprising aptitude for tracking, and Tabby was intrigued by the agility course, to the point of trying to pull me through the tire jump!

We watched the herding dogs as they worked sheep. I suspect the wolfhounds had other ideas about what to do with the sheep, but they kept their intentions to themselves. Miles was honored as a veteran and made the first round in the limbo contest (no small feat when competing against shelties and dachshunds). I learned a little about clicker training and a lot about my dogs. We had a wonderful, if exhausting, time.

This year was a little different. First the girls all decided to come in season for the week of dog camp so it was just me and the boys, Miles (8) and Tim (2). I resolved that we would spend more time relaxing,

especially Miles, and less time trying to get to a class every minute. The lovely outdoor setting of the camp, with lake and trees and lawns was a perfect place to relax and enjoy the company of my guys. I also did classes with one guy at a time, usually trading off dogs halfway through class. That way each had some time of undivided attention with me.

To escape from Alcatraz, the team had to do various things that wouldn't be part of any standard obedience training. Teams were scored not only on being able to accomplish the tricks, but on creativity and the “WOW factor”. Wolfhounds start out with an advantage in that category because no one expects them to do much of anything. I wasn't too opti-



**THE DARING ESCAPE FROM ALCATRAZ!**

Most of all we enjoyed the “Clicker Challenge—Escape from Alcatraz,” the brainchild of Leslie Nelson, one of the instructors. For anyone who hasn't been exposed to clicker training, it involves lots of food. Since food is my guys' favorite subject, we teamed up with a group of dogs and their people for the challenge.

mistic, frankly. The girls are the escape artists in my family. Miles and Tim are usually glued to my side.

Fortunately there were some places for wolfhounds to shine. One exercise called for the dog to back up down a hall with a right angle in it. I have just such a hall at home, often







**LIFTOFF!**

The jump was probably what won us the day, though. The agile jumpers on our team, especially beautiful little Alex, showed they could clear Miles with "miles to spare." It isn't any dog that will stand like a statue, slowly wagging his tail, as another dog comes fly-

We did some more relaxing this year, watching the agility and herding dogs. Tim did a bit of wading, but declined to chase any silly decoys or leave me on the bank of the lake. I am sure had I gone in with him he would have followed me anywhere.

Tim and Miles weren't the only dogs that didn't fit my stereotype of "camp dogs." At dog camp we have met dogs from miniature poodles and pugs to St. Bernards and Mastiffs with almost every breed and combination of breeds in between. Probably the funniest thing I have ever seen is the sheep-herding pug. Breeds I never thought I would like turned out to have some lovely representatives.

In the end, dog camp isn't about which dog can do the most or the best, it is about learning what your dog can do and what he enjoys. Camp is also about making friends with people and dogs you

clogged with wolfhounds. As a consequence, everyone in my "family" knows how to back up when I start down the hall and say "excuse me." Miles demonstrated that skill in competition. I doubt anyone expected that sort of agility from a veteran wolfhound but Miles was just thinking that I was headed for the kitchen, whence all good things come.

Then there was the wall and the tunnel. In one exercise we needed dogs to jump over dogs (the wall) and in another we needed to have dogs go under at least two other dogs in tunneling out of the "prison." I figured Miles and Tim could walk over most dogs and any dog could go under them. One thing they are good at is standing still, especially if I am in front of them with a pocket full of food.

The tunnel worked great except that it was hard for me to continuously reward two wolfhounds simultaneously while they were standing side by side and people were coaxing their dogs to go under. We ended up with Tim and one of the more moderate sized dogs acting as the tunnel.

ing at and over him, but Miles did it time and again! He may have thought it pretty silly, but he loved the applause and I am sure he never suspected they were clapping for Alex.

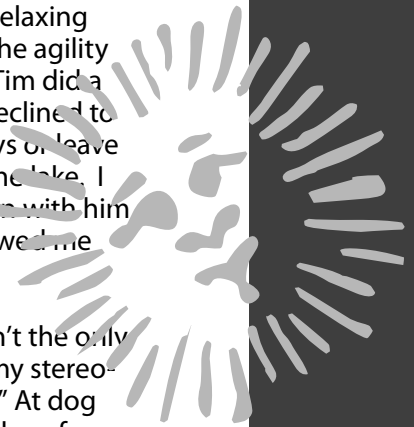
Every evening we hung out in our dormitory with other dogs and people, practicing, talking, training and just relaxing. Miles and Tim slept next to me. The meal hall didn't allow dogs but they always had an extra roll or something to smuggle out to my waiting guys. The weather was perfect autumn, with clear, cool days and nights.

Classes were relaxing, including such topics as the "Really Reliable Recall" and "Backpacking with Dogs" and "Rally." Training classes emphasized methods rather than typical obedience exercises. Dogs and handlers were expected to show good manners. This was more of a challenge for Tim than Miles, but it is amazing how dogs can behave when the environment is relaxed. I offered both dogs an opportunity to lure course, but they found it more fun to observe. Amazing how many non-sighthounds would run themselves into the ground if given the chance!



**AIRBORNE!**

might never otherwise meet. Wolfhounds like to take it easy, but they also like to learn new things and get your undivided attention for a few minutes or hours.



Antibiotics and the bacteria they are designed to eliminate face off in a battle important to our wolfhounds—and to us. Is there anything we can do to stop the spread of antibiotic resistance?

# RESISTANCE is FUTILE

by Kathy Wilson

**V**ery simply put, antibiotics either kill or stop the growth of susceptible bacteria. Antibiotic resistance is the ability of bacteria to escape or neutralize the effects of an antibiotic which is designed to cure (or prevent) infection. When bacteria are able to outwit an antibiotic which previously could control its harmful effects, it is considered resistant.

**B**acteria have the ability to adapt rapidly to changes in their environment. They are single-cell organisms with few genes, so even one small, random mutation may prove extremely important. Bacteria can acquire pieces of DNA that code for resistance from other bacteria, meaning that bacteria can become resistant to many antibiotics because of the transfer of just one piece of DNA. Every time an antibiotic is used, sensitive bacteria are killed, but a few resistant cells may be left to grow and multiply. And because bacteria can reproduce rapidly, they can evolve rapidly. Any mutation that helps them outwit an antibiotic can quickly become the dominant strain in the bacterial population. Not only the target bacteria is exposed to antibiotics when a sick animal is treated. Most of the normal flora which are either helpful or harmless to the host animal will be exposed to any antibiotic drugs used. So these “innocent bystanders” also have the opportunity to develop resistance. Thus, exposure to antibiotics provides selective pressure, making any surviving bacteria more likely to be resistant.

**A**t the same time that more kinds of bacteria are becoming resistant, multiple-drug resistance is also increasing. Approximately 30% of infections caused by streptococcus pneumoniae (including pneumonia, meningitis, etc.) are resistant to penicillin. In the 1970's, virtually all were susceptible to penicillin. Diseases we once believed we could eradicate, such as tuberculosis and gonorrhea, are alive, well, and increasingly resistant to antibiotics. Many other infections which are not as well-documented (because they are not considered public health risks), like inner-ear infections and urinary tract infections, are now routinely caused by resistant bacteria.

**B**ecause all antibiotic use—whether for humans or animals—clearly contributes to increasingly widespread resistance, the WHO, CDC, FDA, and USDA have programs to monitor the spread of antibiotic resistance and to decrease the overuse of antibiotics. About half of all antibiotics produced in the United States are used to treat animals, and the majority of these are used in poultry and livestock production. The AMA, American Society for Microbiology, and American Public Health Association have all called for a ban on non-therapeutic use of antibiotics in food animals. Lobbies for the food and pharmaceutical producers are so adept at challenging any attempt at restriction, though, that when the FDA proposed to eliminate all fluoroquinolone use in poultry production, it took five years before they were able to put the ban in place.

**S**crutiny of the relationship between antibiotic use in companion animals and the spread of resistance is fairly recent. It has become apparent that companion animals can serve as a potential reservoir for antibiotic-resistant zoonoses (diseases that can be transmitted from man to animals). There are well-documented cases in which companion animals have become infected with resistant strains of bacteria acquired from humans, and vice versa. These cases include a resistant strain of e. coli spread from a dog to its owner, and cases of MRSA, a resistant staph infection, spread from owners to dogs, horses, and cats.

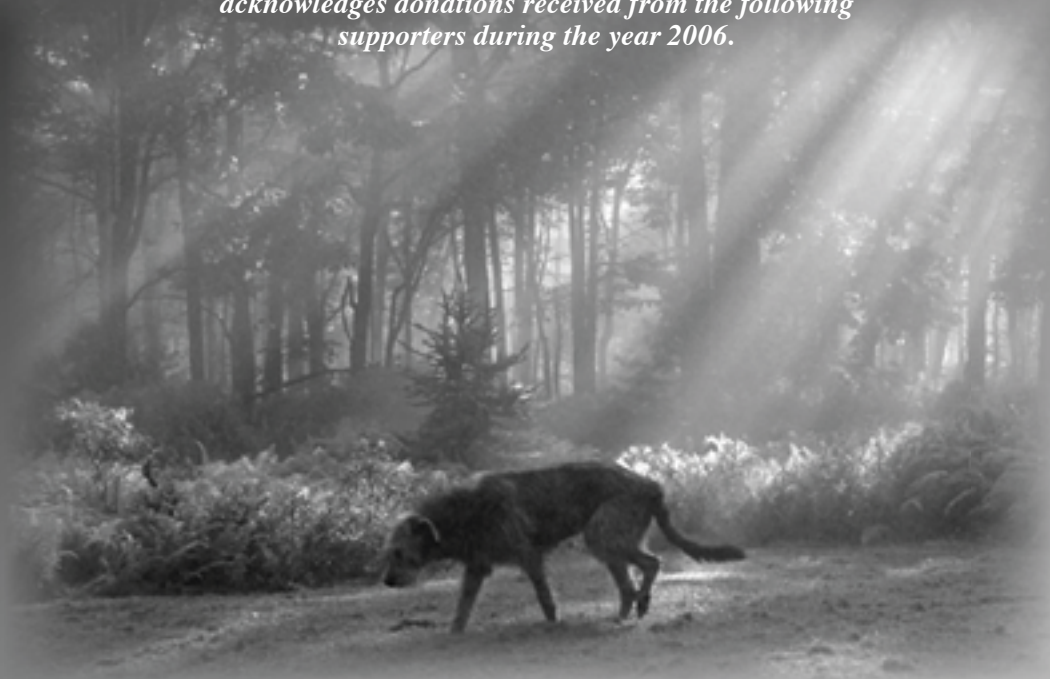
**I**n the last few years, the AVMA, ACVIM, AAHA, CVMA (Canada), BSAVA (Britain), and FVE (Europe) have all developed formal statements concerning the ‘prudent use’ of antibiotics. These documents provide advice on minimizing the development and spread of resistant bacteria in veterinary medicine. Suggestions include parameters for duration of use, optimal dosing, selecting the narrowest-spectrum antibiotic, culture/susceptibility testing recommendation, and ethical use of “extra-label” antibiotics.

**M**any of the largest veterinary hospitals have initiated their own guidelines for using antibiotics. Drugs are classified as first choice (amoxicillin, first generation cephalosporins, etc.), which can be used empirically; second choice (fluoroquinolones, second and third generation cephalosporins, etc.), drugs which must be justified by culture and susceptibility testing; and last choice antibiotics (vancomycin, etc.) which require culture/susceptibility testing and consultation with an infectious disease specialist.

**W**hy is this so important for us to understand, and is there anything we can do to help stop the rise of bacterial resistance? First of all, make sure you always complete the course of antibiotics prescribed for your wolfhound and for yourself, too. Ask for culture/susceptibility testing whenever practical. Be aware that although still very rare, resistant strains of staph, like MRSA, can be transferred between humans and companion animals. Both humans and domestic animals can be carriers without showing overt signs of disease. There is doubtless more transfer of bacteria (both resistant and non-resistant) between us and the animals we share our homes with—it just hasn't been documented. Lastly, don't be surprised if your veterinarian is reluctant to prescribe anything but first-choice antibiotics for your IW without insisting on tests.

**I**n our quick-fix society, it's so easy to reach for a pill without thinking about the consequences. Antibiotics were arguably the biggest medical breakthrough of the twentieth century. What if the marvel of the twentieth century no longer works for us in the twenty-first? What a tragedy that would be.

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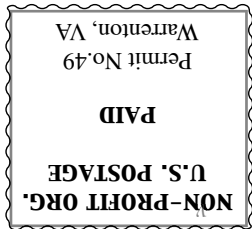
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