FCE- Fibrocartilaginous Emboli
by Ellen Kroll

I hope to help you understand what the disease is and separate the few facts that we know from the theories, guesses and outright rumors. Like all afflictions in our breed, we would all like it to be Black and White, a simple dominant gene with an easy solution. This is not that type of story. There are few facts and LOTS of theories.

FCE—Fibro cartilaginous Emboli was first diagnosed in man in 1961. Dogs in 1973.... certainly it occurred before that time, but our diagnostic tools just were not good enough to help us know what it was. Humans, pigs, cattle, one cat and one horse have been diagnosed with this ailment, but dogs are the most common species affected.

In most dog breeds, it occurs in adults most commonly from 3-7 years of age. In Irish Wolfhounds specifically, it occurs more frequently as puppies.... generally, between 6 and 16 weeks of age. (Some other large breeds have had it reported as young as 16 weeks) Since it was associated in timeframe with the vaccination age, people often made the leap that vaccines were causing this paralysis. In my own opinion and that of any author of journal article I have read, the timeframe of vaccination is completely coincidental. I know of at least 5 youngsters with presumed FCE who had not been vaccinated at all.

WHAT IS IT?

Fibrocartilaginous Emboli or Spinal Cord Infarction; also called Puppy Paralysis and Drag leg Syndrome
This is an emboli made up of fibrous/cartilage type of tissue. Let’s get the terms defined before we start throwing them all around.

Emboli is just plural for embolus. An embolism is:

OBSTRUCTION OF A VESSEL BY A SOLID OR GAS MATTER WHICH HAS BEEN TRANSPORTED THRU THE BLOODSTREAM.

And the embolus can be a blood clot or air or, in this case, a specific type of fibro-cartilaginous tissue that gets lodged in the bloodstream and blocks further blood flow. When that happens to a blood vessel, whatever cells it was serving no longer get the benefit of nutrient/oxygen exchange resulting in cell death. This is called:

ISCHEMIA or the lack of blood flow to a part or organ.

The damage that occurs is often referred to as:
INFARCTION or the tissue death that occurs when blood supply is lacking. You have probably heard the term myocardial infarction when this process happens to the heart muscle. Therefore, some people call FCE a spinal cord infarction. Doing a journal search, this
term will give you more connects than FCE. In FCE, the source of the embolus is from the disc in between the vertebrae in the spinal column. Let’s take a look at where this occurs.

VERTEBRAL FORAMEN A diagram of the space that the nerve roots and blood vessels pass through the spaces between the vertebrae.

VERTEBRAE WITH THE NUCLEOSUS PULPOSUS -- Shows the actual disc between the vertebrae with the central location of the nucleolus pulposus.
If you are looking at a diagram of the intervertebral disc, find the center of the disc. This central area is the location of the Nucleus Pulposus—a gel-like material and this is what is found to be the source of the emboli in FCE.

Now the $64,000 question is HOW? How does a bit of that disc material get into the blood vessel to block the blood flow? There are several theories, but nothing proven.

1. Trauma to the vessel bed causes communication of the disc material
2. Persistence of embryonic arteries of the disc and herniation of the disc material into those arteries. (This might be a likely theory in the case of young puppies)
3. New arteries forming in the disc due to chronic inflammation. (This theory might be more likely in the older animal.)
4. Herniation of the disc material into venous supply, then lodging in an artery.

None of these theories have been proven out consistently in autopsies on people or dogs.

FCE is usually a diagnosis of EXCLUSION. What that means is that the only way to positively identify this disease is by frozen microscopic sections of the nerve to find the emboli after euthanasia. So what if we would like to keep the dog alive? We diagnose FCE by elimination of other probable disease processes. In recent years, advanced MRI imaging and expert diagnosticians are successfully diagnosing FCE in live dogs. However, the advanced MRI and the experienced specialist are not available to many pet owners in the US.

This makes it a very difficult disease for data collection and research. The diagnosis is actually presumed FCE in any live dog.

**WHAT DOES FCE LOOK LIKE?**

It mimics many other conditions—there is no outward symptom that will be unique to FCE. A typical presentation of FCE follows:

Puppy, active and normal...perhaps a history of trauma. Slipping, falling, and dropped. Sitting position, unable to rise.
When placed in a stand, cannot walk forward...will often collapse into a sit
Deep pain response intact (somewhat controversial statement, but that is my personal experience with Wolfhound puppies)

PAIN is not usually consistent with an FCE diagnosis. I have received several calls in the past few years about pups that have a paralysis with an onset of severe pain...screaming in intense pain for 24 hrs or more with slowly improving condition and pain relief. My best guess is trauma...not FCE.

Many of the human patients who succumbed to FCE reported a transient sudden pain on onset. Many owners of dogs who were positively identified with FCE reported yelps of pain and then no pain thereafter, just paralysis.
DIAGNOSIS

In my opinion, you need to get a neurological exam immediately. ALL SPINAL INJURIES, WHETHER FCE OR TRAUMA NEED IMMEDIATE CARE!!!!!! THE FASTER YOU GET VETERINARY TREATMENT, THE BETTER THE PROGNOSIS. REMEMBER THE GOLDEN HOUR. It is critical to determine if the symptoms are due to trauma/injury or FCE.

If you remember nothing else about this discussion: All dogs that present with a paralysis should be immediately transported to a veterinarian who is willing to do a neurological exam and treat the dog as appropriate for the symptoms discovered. (Do not wait until morning!) Steroids have been the treatment of choice for FCE for many years. HOWEVER, recent research published in 2009 (Journal Vet Med Science 2/2009; 71(2) 171-6) suggests that steroid treatment in a presumed FCE case does NOT make any difference in outcome. This study was not focused on Irish Wolfhounds or even giant breeds, but definitely questions the wisdom of steroid treatment.

Diagnostic procedures will help us with a diagnosis, treatment and prognosis. If you find evidence of trauma (swelling, redness, etc.) the treatment needs to start immediately. There are conditions of the spine and paralysis that can involve infection where steroids alone could make matters worse. If infection (discospondylitis or neurospora/toxoplasmosis in very young pups) is suspected... the neurologists that I have spoken with would concurrently treat with antibiotics. The other common rule out with an older animal is cancer of the spine. Sudden onset is the picture you get with FCE, not a gradual onset over days.

Lower Motor Neuron signs versus Upper Motor Neuron signs. This separation has to do with the pathways that these nerves serve. In LMN disruption, you will see flaccid muscle tone where with UMN, there can be a heightened tone to the muscle. LMN pathways do not recover as well as UMN. Paralysis from FCE is usually complete in 24 hours. If you see worsening signs after that timeframe, especially ascending paralysis...it is usually indicative of a softening of the spinal cord and is a very grave sign. If there is little or no improvement over 14 days after onset, the prognosis for recovery becomes very guarded. There is a better prognosis for lateral signs (one sided).

Myelograms versus MRI in Wolfhounds: The myelogram is a radiography study where dye is injected into the spinal column to look for abnormalities, swellings, disc protrusions, etc. This procedure can be successful with dogs, but I have lost a young bitch in a myelogram procedure and know several other breeders who have had dogs die during this procedure. Certainly that is not the case with all Wolfhounds.... but I would recommend looking at an MRI as a less invasive procedure that may give better information for us to rule out other disease processes in suspect FCE cases. Especially MRI machines that are a TESLA 4 can give amazing detail for diagnosis. The MRI would be considered the gold standard in this type of differential diagnosis.

Tough Decisions: I often receive calls from owners of young Wolfhounds with suspect FCE. Their veterinarians are making recommendations on the diagnostic procedures involved and they are EXPENSIVE. My own opinion (for what it is worth) is that you need to have a frank
discussion with your veterinarian about the cost of procedures versus the benefit of the results. In other words, what will the information gained in that procedure do to affect the treatment that we choose? Bottom line, you make the decision based on what is best for your dog.

TREATMENT
Antibiotics as indicated. Treat any trauma. NOTE: One Wolfhound owner treated two pups several years ago with Intravenous DMSO in the first two days after onset. These pups had a strong recovery. I have not discovered any further literature or research to support this approach.

Supportive care: An article published in 2003 reviewed FCE and suspect FCE cases. Journal of Small Animal Practice, (2003) 44, 76-80. One of their conclusions is that the EARLIER that physiotherapy/hydrotherapy is started, the better the outcome. Because this ailment frequently strikes puppies in the middle of their growth, limitation of free exercise with plenty of rest is critical to preventing further injury to non-affected limbs. Although the location and severity of the paralysis can radically change the level of care needed, the following components must be addressed. Confinement/enforced rest with frequent passive range of motion exercises, excellent bedding, opportunities to eliminate with support and good nutrition. High level of care may be needed for first 10 days. Then, recovery to ambulatory state should be there if recovery chance is good/. Look at options of carts/slings if necessary (recovering animals need exercise...not limited motions)

Complementary recovery techniques

Physiotherapy: Range of motion, gait training, hydrotherapy
Swimming: Provides for use of muscles, limits atrophy, doesn’t depend on gravity. Warm water therapy pools may have advantages.
Acupuncture: -Opening|| of channels of energy along meridians. Eliminates stagnation and increase flow of -qi|| in Chinese medicine terms
Chiropractic: Assists in correction of compensatory limitations. Most animals with FCE or other paralysis develop stiffness, soreness or muscle adaptations to compensate for the affected limb. (opposite front leg restriction example)
Massage: Tellington/Jones, standard, range of motion, fascia release techniques. All can help to avoid compensatory damage
Nutrition: High quality proteins/vegies pulsed for repair and growth. Vitamin E/C/B complex. Trace minerals may be needed. Probiotics
Herbs: Arnica, rhus tox, homeopathics for inflammation and injury are indicated early in the onset of FCE. Asafoetida is a Chinese herb that may assist in longer-term recovery.

SO, IS IT GENETIC????- Questions for breeders

Ninety nine percent of all the vet neurologists out there will tell you NO WAY. However, An article from University of Utrech (2000) reviews 8 IW pups with presumed FCE. Certainly our breed has the tendency to FCE at a young age. But is it a tendency like bone cancer afflicting giant breeds or is it a genetic fault that could be bred away from?

My personal data has revealed more than 5-7 lines involved worldwide with few common ancestors in each of those lines.
In one example, one popular stud dog had been linebred several times and produced NO FCE pups. In 3 subsequent outcrosses, he produced at least one FCE pup in each litter. His sire had been bred both in outcross and linebred breedings with no FCE pups, but did produce one FCE pup in his last litter which was an outcross.

So we are left with the question, what do we do as breeders? As with most things, it will be a very personal decision. We have no proof that breeding an affected bitch increases the chance of FCE. Yes, affected bitches have been bred without producing FCE. But, without data collection on significant numbers of dogs, we will never be able to recognize a possible pattern.

Anne Janis, who leads the data collection for seizure disorders, rhinitis (primary ciliary dyskinesia) and liver shunt has offered to collect information on dogs with presumed FCE. Difficulties in the presumed diagnosis makes data less reliable than other diseases. Differing levels of expertise and diagnostics may result in false positives. My hope in collecting anecdotal information in large enough numbers is that we would see a trend toward random occurrence or distinct patterns of inheritance. I would encourage all breeders who have a presumed or confirmed FCE case to contact Anne Janis (jwstudy@earthlink.net) with pedigree information. Anne will gladly explain her data collection process.

The WITCH-HUNT PROBLEM—When improved diagnosis and testing allows us better identification of the dogs and perhaps bloodlines involved, will we use the information responsibly? Or will we treat those who have shared the information with us as somehow irresponsible and unworthy? It is my opinion that it is human nature to gossip and bad news seems to travel faster than good news. I ask each of you to consider positively supporting ALL breeders who benefit our breed by sharing information on the health of our dogs.

Reproduced by The Irish Wolfhound Health Group with the kind permission of Ellen Kroll

The IWHG is assisting the University of Nottingham with research into fibrocartilagenous embolism (FCE, puppy paralysis). Irish Wolfhounds are the only breed where this occurs in puppies, as most breeds are affected at around five or six. We hope that this research will discover the cause/s of FCE and determine the most effective treatment for a positive outcome.

If you have had experience of FCE or any form of puppy paralysis, please complete our survey and make your experience count.

The survey can be downloaded at: http://www.iwhealthgroup.co.uk/puppy-paralysis.html
References

Fibrocartilaginous embolism of the spinal cord diagnosed by characteristic clinical findings and magnetic resonance imaging in 26 dogs
Yuya Nakamoto, Tsuyoshi Ozawa, Kengo Katakabe, Koichi Nishiya, Nobuhiko Yasuda, Tadahisa Mashita, Yutaka Morita, Munekazu Nakaichi
Journal Veterinary Medical Science. February 2009; 71(2) 171-6.

Fibrocartilagenous embolism in 75 dogs: clinical findings and factors influencing the recovery rate
G Gandini, S. Cizinaurskas, J. Lang, R Fatzer and A Jaggy

Fibrocartilaginous Embolism of the Spinal Cord (FCE) in Juvenile Irish Wolfhounds
K Junker, SGAM van den Ingh, MM Bossard and JJ van Nes
(all at Utrecht University, the Netherlands)
Veterinary Quarterly 2000, 22 154-156

Fibrocartilaginous Embolism in Dogs  Laurent Cauzinelle, DMV
Veterinary Clinics of North America – Small Animal Practice
Volume 30, Number 1 January 2000

Fibrocartilaginous Embolism of the Spinal Cord in Dogs: Review of 36 Histologically Confirmed Cases and Retrospective Study of 26 Suspected Cases
Laurent Cauzinille and Joe N Kornegay
Journal of Veterinary Internal Medicine, Volume 10, No 4 (July-August) 1996: pp 241-245

Fibrocartilaginous Emboli  T. Mark Neer, DVM
Veterinary Clinics of North America—Small Animal Practice  Volume 22, Number 4, July 1992

Fibrocartilaginous Embolism  James R Cook, Jr. DVM
Veterinary Clinics of North America— Small Animal Practice  Volume 18, No 3, May 1988

Necrotizing Myelopathy Secondary to Presumed or Confirmed Fibrocartilaginous Embolism in 24 dogs
Dougal R Gilmore, BVSc and Alexander deLahunta, DVM
Journal of the American Animal Hospital Association  July/August 1987, Vol. 23

Fibrocartilaginous embolism and ischemic myelopathy in a 4 month old German Shepherd Dog
C. E. Doige and J.M. Parent