I receive many inquiries about Norwich puppies. Many prospective owners ask about inherited health problems, but the problem most often asked about is epilepsy.

Epilepsy is the most common neurological symptom veterinarians encounter. Between 0.5 and 5.7 percent of all dogs, including mixed breeds, experience seizures in their lifetime, with the reported incidence slightly higher among males than females.

Epilepsy is a result of cerebral dysfunction. Seizure, fit and convulsion are synonyms for irregular brain spasms that begin suddenly and end spontaneously. Most episodes involve loss of consciousness and involuntary spasms of the limbs and face, but the range of symptoms is very wide. Episodes may be much less severe and may not involve any loss of consciousness.

Primary (idiopathic, genetic, true or inherited) epilepsy is probably caused by a chemical defect within the brain cells, although no known cause can be demonstrated through diagnostic tests currently available. Some researchers believe idiopathic canine epilepsy has strong genetic mechanisms.

With its strong genetic implications, primary epilepsy is of great concern to the breeder. Test breedings of epileptic Beagles, Siberian Huskies and German Shepherd Dogs produced 38, 66 and 100 percent, respectively, of offspring with seizures at an early age. Since only 0.5 to 5.7 percent of the general canine population has seizures in their lifetime, these results strongly suggest the epilepsy in the offspring of affected parents was transmitted genetically.

Secondary (acquired or symptomatic) epilepsy is the result of damage to the brain cells from one or more of a myriad of causes. Metabolic disorders involving the liver or thyroid gland; intracranial diseases such as encephalitis; brain tumors; traumas to the head and toxic reactions to flea preventives and vegetation sprays have all been implicated, as have the normal hormonal changes that occur during estrus and pregnancy. Dogs suffering from secondary epilepsy are more likely to show abnormal diagnostic results than dogs with primary epilepsy, which usually test normal between episodes.

Diagnosing the difference between primary and secondary epilepsy depends on data that should include as its baseline a serum chemistry profile, complete blood count and urinalysis.

Most researchers believe epilepsy-related abnormalities occur in the cells found in the cerebral cortex. Seizures are a result of uncontrolled, rapid discharges of neurons, but it is unknown what initially excites the nerve cells. While some studies implicate abnormal levels of neurotransmitters and the enzymes that control them, probably there are a number of mechanisms, since almost any change in the neuron’s environment can, in some dogs, result in spontaneous discharge.

The series of events that excites neurons to spontaneous episode is called a seizure threshold. Each dog has a threshold which, if exceeded, will result in a seizure. The problem lies in the extremely low
level at which some dogs seize with no detectable stimulus; their low threshold is probably genetically
determined.

Until DNA testing can pinpoint the genetic marker, there is no easy solution for conscientious
breeders trying to maintain a healthy line. Some scientists say a recessive gene is probably the
controlling mechanism involved in primary epilepsy and we can breed around it. Others suggest a
two-locus mechanism on the gene — one autosomal recessive, the other a sex-linked suppressor
which might explain why the trait can skip generations.

Armed with pedigrees of affected dogs, I went over old lines, as far back as 30 years, and observed
that seizures and primary epilepsy have been found in Norwich since at least the early 1960s. If a
simple recessive gene were responsible, there should be much more evidence of epilepsy than the
low incidence reported. The numbers are so low, epilepsy is not even listed as a problem in Norwich,
although many cases are not reported to teaching hospitals for entry in data banks.

Until we more completely understand the genetic mechanisms involved, it would be prudent for
breeders to consider the following. Primary epilepsy probably has a major genetic component. It is
probably impossible without multiple test breedings to determine the degree of involvement of either
sire or dam in producing epileptic offspring. It is unwise to breed an epileptic dog or to repeat a
breeding that produced it. It is probably unwise to breed the littermates of an epileptic dog.

As a relatively new breed we still have an opportunity to block and dilute the genetic components of
many of our serious health problems, but it will not happen until we accept the responsibility of
informed, careful breeding. It is not acceptable to shrug and say, as others have in the past, It’s always
been in the breed. Maybe so, but if we acknowledge that in the long run, public interest depends on
private virtue, it behooves us to practice truth in breeding and share without condemnation our
failures as well as our successes. Let’s prioritize our goals and work together toward a bright future for
our little dogs.

My main references were articles by Linda G. Shell, DVM, DACVIM, Virginia Polytechnic College of
Veterinary Medicine.

— Linda Haring, Guest AKC Gazette Breed Columnist

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