CASE REPORT

Is it a seizure or dyskinesia? Episodic involuntary movements in a litter of German Shorthair Pointer puppies

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Introduction

Primary episodic movement disorders in dogs, termed paroxysmal dyskinesias, present a diagnostic challenge as affected animals are usually normal between episodes with no significant findings on physical and neurological examination. Veterinarians have traditionally had to rely on owners' descriptions of the abnormal movement episodes when making a presumptive diagnosis, but the ubiquity of smartphone technology means owners can capture episodes on video, leading to an increased recognition of these disorders (Cerda-Gonzalez *et al.* 2021).

In theory, the distinction between a movement disorder and an epileptic seizure appears to be straightforward. Both are neurological conditions characterised by involuntary muscle contraction and/or relaxation, but epileptic seizures are usually accompanied by an alteration in mentation and autonomic signs (such as urination, defecation and hypersalivation), along with altered post-ictal behaviour, which are not features of paroxysmal dyskinesia (Lowrie and Garosi 2017; Cerda-Gonzalez *et al.* 2021). However, changes in mentation can be subtle with focal seizures and animals experiencing a dyskinesia episode may not behave normally due to anxiety or stress. Furthermore, it is not practical to perform an electroencephalogram (EEG) in veterinary species during an abnormal episode to identify the altered waveforms that are a feature of seizures but not dyskinesias. In addition, recent advances in understanding the genetic basis of movement disorders in humans have revealed that mutations in patients with paroxysmal dyskinesia may also be associated with seizures or a high frequency of seizure disorders in their families (Urkasemsin and Olby 2014).

This article describes the occurrence of episodes of abnormal movements in multiple German Shorthair Pointer puppies from the same litter and uses these cases to discuss the presentation and diagnosis of paroxysmal dyskinesia in dogs. As visualisation of the abnormal episodes is useful in the recognition of these disorders, two QR codes have been provided (see right) which you can scan to access two short recordings of abnormal movement episodes in the affected puppies.

Case history

At three weeks of age, four German Shorthair Pointer puppies from a litter of 12 started to have short episodes where they would develop an abnormal gait, be unable to support themselves and fall over. The two female and two male affected puppies had grown and developed normally up until this point, and the sire, dam and other eight puppies were never observed to have similar episodes. The episodes were most common while the puppies were eating or within 20 minutes after eating, regardless of whether the puppies fed from the dam, were given puppy milk made from powder, or fed commercial puppy food. During the episodes, the puppies remained conscious and occasionally continued

Video 1. Episode during veterinary clinic visit. https://www.youtube.com/watch?v=_xLlsHlpmus

Video 2. Episode following eating. https://www.youtube.com/watch?v=_xLlsHlpmus

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to eat despite being unable to stand normally; in some instances, the puppy walked on either the fore or hindlimbs, while the other limbs remained flexed or extended. The episodes of abnormal movement were self-limiting and lasted less than five minutes, and once they had passed the puppies exhibited normal gait and behaviour. Episodes varied in their frequency, with the worst affected puppy having two episodes per day (coinciding with morning and afternoon feedings), while the least affected puppy typically had one episode every two days and was normal when eating at other times. The sire had been used for breeding previously with no reported abnormalities in the resulting puppies, while this was the dam's first litter.

Clinical examination

The two female affected puppies were examined by a veterinarian at 5 weeks of age. Handling and restraint for blood sampling induced an episode of abnormal movement in both puppies. When lifted into a standing position during the episode, the puppies would drop into sternal or lateral recumbency and sudden flexion or extension of the limbs would result in them rolling over. Both puppies remained conscious and responsive to stimuli and were able to move their limbs during the episode but were unable to right themselves or stand. No muscle tremors were observed, and the episodes did not appear to be painful, although the puppies did vocalise during the episodes. Cardiac auscultation before and during the episodes revealed both puppies had a normal sinus rhythm, no murmurs, and mucous membranes remained pink with a rapid capillary refill time. Full neurological examinations performed prior to an episode were normal except for a lack of menace response in both puppies. This was considered appropriate for their age as this is a learned response that may not be present until 12 weeks of age (Ofri 2012), although some sources state it may be seen in puppies at 3–4 weeks old (Lavely 2006). It was not possible to perform full neurological examinations at the time of the episodes, but assessment of cranial nerve function during the episodes was normal.

Laboratory findings

A blood sample was collected from the worst affected puppy and submitted to a commercial veterinary diagnostic laboratory for a complete blood count, serum biochemistry panel and *Neospora* titres. Numerous results were reported as being abnormal, but the reference ranges provided by the laboratory were based on adult dogs and the results for this puppy were all consistent with reported normal values in young puppies between 2–3 months of age (von Dehn 2014). Results that differed from the adult reference range included a low red blood cell count, haemoglobin, HCT, MCHC, sodium, chloride, creatinine, and globulins, along with mild increases in lymphocytes, potassium, phosphate, calcium, alkaline phosphatase, aspartate aminotransferase and creatine kinase. The immunofluorescent antibody test for *Neospora* was negative, and fasted and 2-hour post-prandial bile acid concentrations were within the reference range provided.

Case progression and diagnosis

The four affected puppies continued to have episodes of a similar duration and frequency over the following week and remained normal between episodes. The nature of the episodes and the lack of other neurological signs was most consistent with paroxysmal dyskinesia, but further diagnostic testing such as EEG and MRI were not performed to definitively rule out other differential diagnoses and no treatment was attempted. The episodes continued to occur mostly in relation to feeding so it was not possible to alleviate the trigger to reduce episode frequency. Given this, and the uncertainty surrounding the diagnosis and prognosis for these puppies, the owner decided they were not suitable for being placed in new homes and they were euthanised at 6 weeks of age. The other eight puppies in the litter remained unaffected and were rehomed as planned. The dam did not have any further litters and died 2 years later for unrelated reasons, while no further information was available regarding the sire.

Pathology

Postmortem examinations were performed on all four puppies within 24 hours of euthanasia. The puppies each weighed between 3.3 and 3.9 kg and there were no abnormalities on gross postmortem or histological examination of a wide range of tissues, including brain, spinal cord, peripheral nerves, and muscle. The brain, liver, and heart of each puppy were individually weighed and all were within normal limits for young dogs, based on published values as a percentage of bodyweight (McDonough and Southard 2016).

Genetic testing

Buccal swabs from the four affected puppies were collected immediately following euthanasia for DNA extraction. Buccal swabs were also collected from two normal siblings in the same litter, and later EDTA blood samples were collected from both the dam and sire. The bloods and swabs were stored at –80°C for approximately 6 months prior to further analysis.

DNA was extracted from buccal swabs using a Qiagen MagAttract HMW DNA Kit (Qiagen, Auckland, NZ) and eluted in 200 µl EB Buffer. Sequencing libraries were produced from 350 ng of DNA using an Illumina DNA Prep Kit (Illumina Australia and NZ, Melbourne, Australia), and sequenced on an Illumina NovaSeq 6000 using an S2 300 flowcell. Sequence reads were mapped to the German Shepherd-based CanFam4 genome assembly UU_Cfam_GSD_1.0 (Wang *et al.* 2021), using bwa mem2 software (v2.2.1) (Vasimuddin *et al.* 2019). Variants were called using Haplotypecaller (v4.2.4.1) (Poplin *et al.* 2018). After applying generic quality filtering criteria based on GATK guidelines (Caetano-Anolles 2023), and filtering to remove common multi-breed population variants (Plassais *et al.* 2019), variants were further filtered based on two disease hypotheses. The first included a mode of transmission based on autosomal recessive inheritance – specifically that unaffected parents should be heterozygous, unaffected siblings should be heterozygous or homozygous reference, and affected animals should be homozygous alternate. The alternative hypothesis assumed a de-novo germline mutation due to a dominant heterozygous mutation in affected animals, with unaffected (or very low-level mosaic) parents, with siblings assumed to be homozygous reference. Candidate variants were then annotated using SNPEff (Cingolani *et al.* 2012) to identify protein-coding candidates. These criteria yielded two frameshift variants and 45 variants with moderate effects (mostly missense variants) under the recessive hypothesis, and 27 nonsense variants under the de-novo dominant hypothesis. Examining the functions of the genes containing these variants highlighted four possible candidates (Table 1). Specific primers were then designed to amplify the regions of interest in these genes so that they could

be independently sequenced in the four affected puppies, two normal siblings, and both parents. However, the sequencing results contrasted with the whole genome results for these candidates, presenting genotypes that were discordant from the criteria used for genome-wide filtering in one or more individuals in every case (likely due to low-depth, short-read sequence at these positions).

While the gene sequencing indicated that none of the four identified candidate genes were likely to be the site of the causative mutation, we consider the disease still likely to be inherited given the presentation of multiple puppies in one litter and the similarity to other cases of inherited paroxysmal dyskinesia. However, the current analysis failed to identify the causative mutation and to our knowledge, no further cases have been reported to date.

Discussion

Canine paroxysmal dyskinesia is characterised by recurrent self-limiting episodes of abnormal, involuntary movement with impairment of voluntary movements (Cerda-Gonzalez *et al.* 2021). Paroxysmal dyskinesia (PD) is considered rare in dogs and is poorly characterised, but the diagnosis is generally limited to observation of an episode, which may lead to underdiagnosis or misdiagnosis as focal seizures (Cerda-Gonzalez *et al.* 2021). In humans, paroxysmal dyskinesias are classified according to the triggers of the episodes or their causative genetic mutations, and terms such as athetosis, ballism and chorea are used to describe the specific nature of the abnormal movements (Manso-Calderón 2019). These terms are not directly applicable to dogs, but some authors do classify canine PD based on when the episodes occur, either following sudden movements (paroxysmal kinesigenic dyskinesia), at rest/not associated with exercise (paroxysmal non-kinesigenic dyskinesia) or associated with fatigue (paroxysmal exertion-induced dyskinesia) (Mandigers *et al.* 2024).

In the puppies described in this report, the episodes of collapse and abnormal movements were described by the owner to be triggered by eating, and initial differential diagnoses included a seizure disorder or a portosystemic shunt causing post-prandial neurological signs. Observation of episodes in two of the puppies while in the veterinary clinic enabled better characterisation of the abnormalities. As the puppies remained conscious and responsive throughout and returned to normal behaviour immediately afterwards, the episodes were considered to be more consistent with PD than a seizure disorder (Lowrie and Garosi 2017). The episodes in the clinic appeared to be triggered by the stress of handling and blood collection, suggesting that the association of the episodes with eating may have been related to the excitement and movements of eating (paroxysmal kinesigenic dyskinesia), rather than being specifically related to food intake.

Serum bile acid testing was performed in one affected puppy to investigate the possibility of a portosystemic shunt or other hepatic abnormality leading to hepatic encephalopathy. While the results of bile acid testing were within the reference range provided, falsely decreased concentrations are possible in dogs under 16 weeks of age (Center 2025). Liver disease, including a portosystemic shunt, was subsequently excluded as a differential diagnosis based on the normal liver size and gross and histologic appearance postmortem in the four affected puppies, but these cases highlight some of the diagnostic challenges when working with paediatric patients. The young age of the affected puppies also contributed to the abnormal results reported in the complete blood count and serum biochemical analytes, as puppies and kittens <6 months old have reduced functional capacity in many organs and variation in enzyme activities relative to standard adult reference ranges (von Dehn 2014). Haematological and serum biochemical testing is considered an important part of the diagnostic workup of cases of suspected cases of PD as some metabolic and endocrine conditions can result in clinical signs similar to PD (Mandigers *et al.* 2024).

Advanced imaging (ideally MRI) is also recommended to rule out a structural brain disease as a cause of dyskinetic episodes, but this is not feasible for many owners or veterinarians in New Zealand and was not

Table 1. Candidate genes investigated as possible causes of episodic collapse in German Shorthair Pointer dogs based on whole genome sequencing results. The variants listed were sequenced in the four affected puppies, two clinically unaffected siblings and the dam and sire. DNA extracted from buccal swabs and blood samples and then amplified with specific primers for each variant. Wild type = the same as the canine reference genome (normal)

Gene variant *Inheritance hypothesis*	Chromosome (loci)	Effect of gene	Result
Missense variants (x2) in bassoon presynaptic cytomatrix protein (BSN); *de novo dominant*	Chr 20 (1786 G>A; 9310 T>G)	Scaffolding protein involved in presynaptic cytoskeleton; mutations in rodents cause spontaneous seizures[1]	Dam and unaffected siblings wild type, affected puppies and sire heterozygous
Missense variant in calcium voltage-gated channel subunit alpha1 D (CACNA1D); *de-novo dominant*	Chr 20 (6065 C>T)	Encodes a subunit of a voltage-gated calcium channel. Mutations associated with sinoatrial node dysfunction, aldosteronism and various neurologic abnormalities,[2,3] not reported in dogs with epilepsy[4]	Dam and unaffected siblings' wild type, affected puppies and sire heterozygous
Splicing variant in cyclin and CBS domain divalent metal cation transport mediator 1 (CNNM1); *de novo dominant*	Chr 28 (2025 A>C)	Copper storage protein in neuronal cells and magnesium transporter.[5] Mutations cause changes in spermatogenesis in mice, while mutations in the related gene CNNM2 cause hypomagnesaemia and seizures in infants	Sire and unaffected siblings' wild type, affected puppies and dam heterozygous
Frameshift variant in synaptojanin-1-like (LOC119877742); *recessive*	Chr 30 (397 GACGAGCC>G)	Synaptojanin-1 is involved in synaptic vesicle recycling. Mutations in humans are variably associated with neonatal seizures,[6] neurodegeneration, and Parkinson's disease. Zebrafish with mutations in synaptojanin-1 have vestibular defects and are unable to right themselves after repeated stimulation[7]	Deletion in two affected puppies, remainder of dogs are wild type

[1]Altrock *et al.* (2003); [2]Tang *et al.* (2024); [3]Alzahrani *et al.* (2023); [4]Ekenstedt *et al.* (2011); [5]Chen and Gehring (2023); [6]Al Zaabi *et al.* (2018); [7]Gao and Nicolson (2021)

performed in the current cases. However, a thorough postmortem examination of all four affected puppies, with histology of the brain, enabled structural CNS abnormalities to be excluded as a cause, supporting a diagnosis of primary paroxysmal dyskinesia. Abnormalities in direct and indirect pathways involving the basal nuclei, located subcortically in the telencephalon, are thought to be central to the pathophysiology of PD, although the cerebellum may also be involved in some cases (Cerda-Gonzalez *et al.* 2021), and some types of PD may represent ion channel disorders (Lowrie and Garosi 2017). Histology of these regions of the brain was normal in the affected puppies, but this is unsurprising given PD is a functional rather than structural disorder.

The majority of cases of paroxysmal dyskinesia in dogs are believed to be hereditary (Cerda-Gonzalez *et al.* 2021) and the disorder has been reported in a wide variety of breeds (Mandigers *et al.* 2024), including a single case in a 17-month-old German Shorthair Pointer (Harcourt-Brown 2008). Dyskinesia has also been reported in association with drug therapy (Kube *et al.* 2006) and in dogs with intracranial lesions (Lowrie and Garosi 2017), but these dogs also had neurological signs between episodes, in contrast to cases of primary paroxysmal dyskinesia. Causative mutations for canine PD have been described in five breeds to date: Cavalier King Charles Spaniels, Soft Coated Wheaten Terriers, Shetland Sheepdogs, Het Markiesje, and Weimaraner dogs (Mandigers *et al.* 2024). Suspected inherited paroxysmal dyskinesia, in the absence of a confirmed causative mutation, has been described in numerous other breeds, including the Chinook (Packer *et al.* 2010), Jack Russell Terrier, Labrador Retriever (Lowrie and Garosi 2016), Norwich Terrier (De Risio *et al.* 2016), and Welsh Terrier (Whittaker *et al.* 2022). In some breeds, paroxysmal dyskinesia is a manifestation of gluten sensitivity and improves with a gluten-free diet (Rogers *et al.* 2023). This is best characterised in Border Terriers where it was previously termed canine epileptoid cramping syndrome and is often accompanied by gastrointestinal signs such as borborygmi (Lowrie *et al.* 2015). However, improvement or resolution of episodes with a gluten-free diet has also been reported in Maltese dogs (Polidoro *et al.* 2020), various crossbreeds (Santifort and Lowrie 2017; Kim *et al.* 2024), and a German Spitz dog (Baptista da Silva *et al.* 2023).

An inherited basis is suspected in the puppies described in this report, given that four of twelve puppies in the litter were affected at the same young age, there was no history of drug administration or other changes in management or the environment, and the puppies were normal between episodes with no gastrointestinal signs. As both males and females were affected, and the parents were apparently normal, an autosomal recessive or *de novo* dominant (germline mutation) mode of inheritance was considered most likely, but the results of further sequence investigation did not support any of the candidate variants identified from whole genome sequencing (WGS). It is not unusual that *de novo* mutations found on WGS are not validated on further investigation, as genotypes based on WGS reads represent random samples of alleles at any given position – meaning that animals may appear homozygous in WGS data yet be heterozygous if resampled. An additional potential confounder was that buccal swabs were used for DNA extraction. In this case buccal swabs of the puppies were used since restraint for blood collection induced episodes of collapse in the affected dogs, but if the puppies had fed from the dam prior to this there could be a small amount of maternal DNA from the milk in the sample. When dealing with a case of suspected inherited disease where DNA may be required for further investigation (beyond commercially available genetic tests), the preferred sample for DNA extraction is EDTA blood, which is kept chilled prior to submission or is rapidly frozen for long-term storage.

Another explanation for the inability to identify the causative mutation could be that if the disease has incomplete penetrance, which is common in PD in humans (Liao *et al.* 2021). With incomplete penetrance, not all individuals with the mutation develop clinical signs, meaning the parents or siblings could also carry the mutation. It is also possible that the normal siblings could be more mildly affected or have a later onset of signs (after being rehomed), as marked phenotypic heterogenicity is described in PD (Lowrie and Garosi 2017). With the increasing availability and accessibility of genetic testing for diseases in humans, owners or breeders may have an expectation that similar tests will be available for their animals if an inherited disease is suspected, but finding mutations is generally challenging, particularly with small numbers of samples. The current understanding of the genetic basis of idiopathic epilepsy in dogs is similar poorly understood, with mutations only in two genes confirmed to be causative (Beckers *et al.* 2023). Whole genome sequencing is currently only useful from a research perspective and not as a diagnostic tool, as the amount of data generated requires advanced computing and bioinformatics capabilities, and samples from larger numbers of animals are needed to allow differentiation of pathologic variants from normal individual variation.

The prognosis for animals with PD is variable, but cases are often non-progressive (Mandigers *et al.* 2024) and in some breeds improvement over time or spontaneous remission is documented (Lowrie and Garosi 2016). In the previously reported case in a German Short Hair Pointer, the dog responded well to anticonvulsant therapy (Harcourt-Brown 2008), and it may be possible to minimise exposure to triggers, but the young age of onset, daily occurrence of episodes, and association with feeding in the present cases meant treatment was not attempted. Trialling a gluten-free diet may be beneficial in cases of PD given the reduction or resolution of episodes reported in some breeds (Rogers *et al.* 2023), as serological testing for gluten antibodies in dogs is not currently commercially available in New Zealand.

Conclusion

Paroxysmal dyskinesia is a differential diagnosis for epileptic seizures in dogs and as the diagnosis relies on observation of an episode, owners should be encouraged to video episodes and the immediate recovery period when they occur. In most cases, the neurological examination is normal in affected animals between episodes and there are no structural abnormalities in the brain on advanced imaging or postmortem examination. These cases in German Shorthair Pointer puppies highlight the challenges in diagnosing movement disorders and in the investigation of novel inherited diseases, and raise important points to consider when faced with these presentations in clinical practice.

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References

Al Zaabi N, Al Menhali N, Al-Jasmi F. SYNJ1 gene associated with neonatal onset of neurodegenerative disorder and intractable seizure. *Molecular Genetics and Genomic Medicine* 6, 109–13, 2018

Altrock WD, Tom Dieck S, Sokolov M, Meyer AC, Sigler A, Brakebusch C, Fässler R, Richter K, Boeckers TM, Potschka H, *et al.* Functional Inactivation of a Fraction of Excitatory Synapses in Mice Deficient for the Active Zone Protein Bassoon. *Neuron* 37, 787-800, 200

Alzahrani A, Alshalan M, Alfurayh M, Bin Akrish A, Alsubeeh NA, Al Mutairi F. Case Report: Clinical delineation of CACNA1D mutation: New cases and literature review. *Frontiers in Neurology* 14, 1131490, 2023

Baptista da Silva C, Hermans M, Ruiz-Suárez N, Verdoodt F, Bhatti SFM, Hesta M. Long-term nutritional management of an obese German Spitz with paroxysmal dyskinesia, calcium oxalate urolithiasis, and suspected pancreatitis-A case report. *Frontiers in Veterinary Science* 10, 1054251, 2023

Beckers E, Bhatti SFM, Van Poucke M, Polis I, Farnir F, Van Nieuwerburgh F, Mandigers P, Van Ham L, Peelman L, Broeckx BJG. Identification of a novel idiopathic epilepsy risk locus and a variant in the CCDC85A Gene in the Dutch Partridge Dog. *Animals (Basel)* 13, 2023

Caetano-Anolles D. *Hard-filtering germline short variants – GATK, GATK technical documentation – algorithms*. https://gatk.broadinstitute.org/hc/en-us/articles/360035890471-Hard-filtering-germline-short-variants (accessed 27 March 2023). 2023

Center SA. *Bile Acids*. https://eclinpath.com/chemistry/liver/liver-function-tests/bile-acids/ (accessed 13 January). Cornell University College of Veterinary Medicine, Ithaca, NY, USA, 2025

Cerda-Gonzalez S, Packer RA, Garosi L, Lowrie M, Mandigers PJJ, O'Brien DP, Volk HA. International veterinary canine dyskinesia task force ECVN consensus statement: Terminology and classification. *Journal of Veterinary Internal Medicine* 35, 1218-30, 2021

Chen YS, Gehring K. New insights into the structure and function of CNNM proteins. *The Febs Journal*, 290, 5475-95, 2023

Cingolani P, Platts A, Wang LL, Coon M, Nguyen T, Wang L, Land SJ, Lu X, Ruden DM. A program for annotating and predicting the effects of single nucleotide polymorphisms, SnpEff. *Fly 6*, 80-92, 2012

De Risio L, Forman OP, Mellersh CS, Freeman J. Paroxysmal dyskinesia in Norwich Terrier Dogs. *Movement Disorders Clinical Practice* 3, 573-9, 2016

Ekenstedt KJ, Patterson EE, Minor KM, Mickelson JR. Candidate genes for idiopathic epilepsy in four dog breeds. *BMC Genetics* 12, 38, 2011

Gao Y, Nicolson T. Temporal vestibular deficits in synaptojanin 1 (synj1) mutants. *Frontiers in Molecular Neuroscience* 13, 604189, 2021

Harcourt-Brown T. Anticonvulsant responsive, episodic movement disorder in a German Shorthaired Pointer. *Journal of Small Animal Practice* 49, 405-7, 2008

Kim M, Cho H, Kim U, Choen S, Yun Y, Song W-J. Suspected paroxysmal dyskinesia in four small-breed dogs: Clinical presentation, diagnosis, management and prognosis. *Veterinary Medicine and Science* 10, e70015, 2024

Kube SA, Vernau KM, LeCouteur RA. Dyskinesia associated with oral phenobarbital administration in a dog. *Journal of Veterinary Internal Medicine* 20, 1238-40, 2006

Lavely JA. Pediatric neurology of the dog and cat. *Veterinary Clinics: Small Animal Practice* 36, 475-501, 2006

Liao JY, Salles PA, Shuaib UA, Fernandez HH. Genetic updates on paroxysmal dyskinesias. *Journal of Neural Transmission* 128, 447-71, 2021

Lowrie M, Garosi L. Natural history of canine paroxysmal movement disorders in Labrador retrievers and Jack Russell terriers. *The Veterinary Journal* 213, 33-7, 2016

Lowrie M, Garosi L. Classification of involuntary movements in dogs: Paroxysmal dyskinesias. *The Veterinary Journal* 220, 65-71, 2017

Lowrie M, Garden OA, Hadjivassiliou M, Harvey RJ, Sanders DS, Powell R, Garosi L. The clinical and serological effect of a gluten-free diet in Border Terriers with epileptoid cramping syndrome. *Journal of Veterinary Internal Medicine* 29, 1564-8, 2015

Mandigers PJJ, Santifort KM, Lowrie M, Garosi L. Canine paroxysmal dyskinesia: a review. *Frontiers in Veterinary Science* 11, 1441332, 2024

Manso-Calderón R. The spectrum of paroxysmal dyskinesias. *Future Neurology* 14, FNL26, 2019

McDonough SP, Southard T. *Necropsy Guide for Dogs, Cats, and Small Mammals*. John Wiley & Sons, 2016

Ofri R. Neuroophthalmology. In: Maggs DJ, Miller PE, Ofri R (eds). *Slatter's Fundamentals of Veterinary Ophthalmology*. 5th Edtn. Elsevier - Health Sciences Division, Chantilly, VA, USA, 2012

Packer RA, Patterson EE, Taylor JF, Coates JR, Schnabel RD, O'Brien DP. Characterization and mode of inheritance of a paroxysmal dyskinesia in Chinook dogs. *Journal of Veterinary Internal Medicine* 24, 1305-13, 2010

Plassais J, Kim J, Davis BW, Karyadi DM, Hogan AN, Harris AC, Decker B, Parker HG, Ostrander EA. Whole genome sequencing of canids reveals genomic regions under selection and variants influencing morphology. *Nature Communications* 10, 1489, 2019

Polidoro D, Van Ham L, Santens P, Cornelis I, Charalambous M, Broeckx BJG, Bhatti SFM. Phenotypic characterization of paroxysmal dyskinesia in Maltese dogs. *Journal of Veterinary Internal Medicine* 34, 1541-6, 2020

Poplin R, Ruano-Rubio V, DePristo MA, Fennell TJ, Carneiro MO, Van der Auwera GA, Kling DE, Gauthier LD, Levy-Moonshine A, Roazen D, *et al.* Scaling accurate genetic variant discovery to tens of thousands of samples. *bioRxiv*, 201178, 2018

Rogers CB, Meyerhoff N, Volk HA. Gluten serological testing in various dog breeds with paroxysmal dyskinesia. *Frontiers in Veterinary Science* 10, 1119441, 2023

Santifort KM, Lowrie M. Paroxysmal non-kinesigenic dyskinesia characterised by dystonia in a crossbred dog. *Veterinary Record Case Reports* 5, e000429, 2017

Tang X, Ortner NJ, Nikonishyna YV, Fernández-Quintero ML, Kokot J, Striessnig J, Liedl KR. Pathogenicity of de novo CACNA1D Ca2+ channel variants predicted from sequence co-variation. *European Journal of Human Genetics* 32, 1065-73, 2024

Urkasemsin G, Olby NJ. Canine paroxysmal movement disorders. *Veterinary Clinics: Small Animal Practice* 44, 1091-102, 2014

Vasimuddin M, Misra S, Li H, Aluru S. Efficient architecture-aware acceleration of BWA-MEM for multicore systems. *2019 IEEE International Parallel and Distributed Processing Symposium (IPDPS).* Pp 314-24, 2019

von Dehn B. Pediatric Clinical Pathology. *Veterinary Clinics of North America: Small Animal Practice* 44, 205-19, 2014

Wang C, Wallerman O, Arendt M-L, Sundström E, Karlsson Å, Nordin J, Mäkeläinen S, Pielberg GR, Hanson J, Ohlsson Å, *et al.* A novel canine reference genome resolves genomic architecture and uncovers transcript complexity. *Communications Biology* 4, 185, 2021

Whittaker DE, Volk HA, De Decker S, Fenn J. Clinical characterisation of a novel paroxysmal dyskinesia in Welsh terrier dogs. *The Veterinary Journal* 281, 105801, 2022 ●