FOUR LEG NEWS

Juvenile Conditions & Issues

Welcome to the puppy issue. All articles & reviews in this edition pertain to juvenile conditions… We'll discuss carpal laxity syndrome, hereditary ataxia, inherited myopathy, and swimmers syndrome. These are things to think about with your puppy patients, between snuggling, cuddling and smooching them!! Enjoy!

Happiness is a warm puppy.

~Charles M. Schulz

Carpal Laxity Syndrome

Çetinkaya MA, Yardimci C, Sa lam M. Carpal laxity syndrome in forty-three puppies. Vet Comp Orthop Traumatol 2007; 20: 126–130.

- Describes either hyperextension or hyperflexion of the carpal joint; also known as Carpal Hyperextension, Carpal Hyperflexion, Carpal Flexural Deformity, Carpal Instability, as well as Carpal Flexion Syndrome.[1]
- The real cause is unknown, factors may be poor muscle tone, weakness between the extensor and flexor muscle groups, or unbalanced growth. It has been suggested that the presence of an asynchronous development between skeleton and the musculotendinous apparatus, with faster growth of bone tissue, causes a relative musculotendinous shortening which results in carpal hyperflexion.[1]
- Doberman Pinchers and Shar Pei puppies may be predisposed.[1]
- A study on puppies with carpal flexural deformity showed that blood Ca, P and Mg values did not change significantly.[2]
- There is no observed correlation between carpal laxity syndrome and sex.[1]
- Treatment recommendations include: balanced diets, exercise on surface providing good muscle traction, limiting exercise, Robert Jones bandage, splints, tenotomy and arthrodesis. The disease is often self-limiting, and has a favourable prognosis with a short duration. In rare cases, the deformity will persist into adulthood.[1]
- Hyperextension of patients that do not resolve conservatively may require carpal arthrodesis to relieve pain due to degenerative joint disease, but physeal closure must be achieved before this operation.[3]
- Soft bandages without splints may be used to prevent abnormal stand. Splinting the leg provides support until the ligaments strengthen and can be used to prevent the development of degenerative joint disease in the carpal joint and the abnormal development of the forelimb, but splint application prevents movement and strengthening of muscles, which may lead to muscle atrophy.[1]
- Flexural deformity of the carpus has been seen in newborn foals due to teratogenic agents, intrauterine mal-positioning, diseases of the mother during pregnancy, etc; and acquired in rapidly growing 10– to 18-month-old foals (due to trauma, infectious polyarthritis and malnutrition).[1]

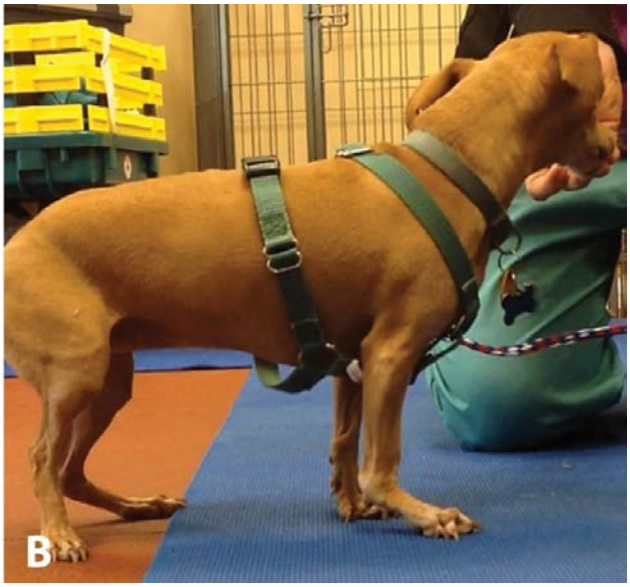
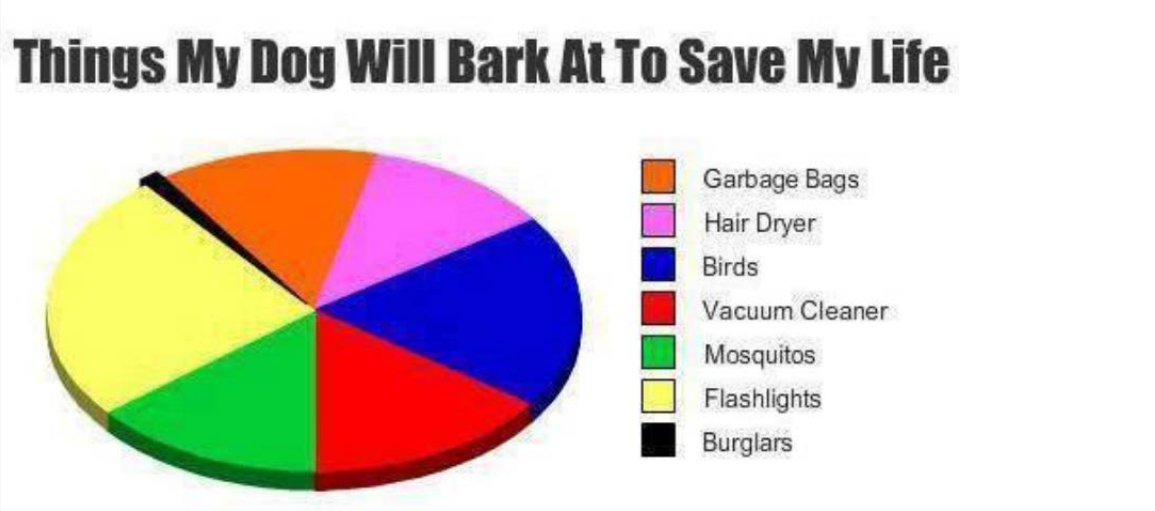


Photo credit:

https://todaysveterinarypractice.com/juvenile-orthopedic-disease-in-dogs-catspart-2-congenital-neonatal-orthopedic-diseases/

As poor or over nutrition of rapidly growing puppies has been suggested to cause weakness between extensor and flexor muscle groups, resulting in laxity of the carpal joint; clinicians determined that appropriate exercise and balanced diets is usually enough to achieve recovery. In their study, 43 puppies with carpal laxity syndrome, aged 5 to 27 weeks, recovered within one to four weeks. Treatment recommendations were exercise on a surface that provided good traction and swimming as well as feeding balanced dietary puppy foods. One puppy with flexor tendon contractures was treated with a splint.This patient also had hyperextension in the contralateral carpal joint that was treated without any splint application. Only two cases showed swelling and pain during palpation and manipulation, one had severe flexor tendon contractures and the other had Hypertrophic Osteodystrophy. Case histories taken from pet owners suggested a sudden onset of abnormal gait and deformity, involving one or both forelimbs, with no obvious trauma. In these puppies, 26 hyperflexion and 41 hyperextension deformities were found. More than half of the cases in this study were mixed breed and Kangal puppies; and aged six to eight weeks. The number of male puppies was greater than the number of female puppies, likely due to more rapid growth and weight gain in males. These puppies were fed either with unbalanced diets or with diets of poor nutritional value. The healing periods for hyperflexion and hyperextension deformities were similar, with healing occurring by week two in the majority of cases. Deformity in the contralateral limb was observed, during the healing period, in three cases. Two cases (from the same litter) had recurrence of deformity in the same limb. Early closure of bilateral distal ulnar growth plate occurred in one case, probably due to the negative effects of abnormal weight distribution on growth plates. Conservative therapy including exercise (on surfaces which provide good muscle traction) and balanced nutrition should be sufficient for the treatment of carpal laxity without muscle contracture.[1]

1. Çetinkaya MA, Yardimci C, Sa lam M. Carpal laxity syndrome in forty-three puppies. Vet Comp Orthop Traumatol 2007; 20: 126–130.
2. Altunatmaz K, Ozsoy S. Carpal Flexural Deformity in Puppies. Vet Med-Czech 2006; 51: 71–74.
3. Stanton MLE. Treatment of Hyperextension and Hyperflexion in Puppies. In: Bojrab MJ, Ellison GW, Slocum B, editors. Current Techniques in Small Animal Surgery. 4th ed. Baltimore: Williams & Wilkins Co.; 1998; 1120–1121.



Hereditary Ataxia

Urkasemsin G, Nielsen DM, Singleton A, et al. Genetics of Hereditary Ataxia in Scottish Terriers. J Vet Intern Med 2017;31:1132–1139.

- Hereditary ataxias are a heterogeneous group of neurodegenerative diseases with a wide range of clinical and pathological manifestations affecting purebred dogs.
- Mutations in 8 different genes have been associated with this disease in more than 10 dog breeds. These genes encode proteins that have a variety of different functions including protein degradation and autophagy, CNS cytoskeleton integrity, cation trafficking, and mitochondrial function and cholesterol trafficking. An autosomal recessive mode of inheritance has been identified in these breeds, and a X-linked mode of inheritance has been reported in the English pointer dogs.
- In Scottish Terriers, hereditary ataxia or hereditary cerebellar degeneration is characterized by severe loss of Purkinje cells, depletion of granule cells and atrophy of the molecular layer as well as polyglucosan body accumulation.
- Clinical signs reflect cerebellar dysfunction including a wide-based stance, dysmetria producing a hypermetric gait, difficulty negotiating stairs and intention tremors. Onset of signs usually occurs during the first year of life and in many dogs, progression is slow with signs ultimately stabilizing, resulting in a lifelong relatively mild phenotype.

Researchers aimed to identify chromosomal regions associated with hereditary ataxia in Scottish Terriers. In their prospective study, Scottish Terriers were recruited through the Scottish Terrier Club of America. A total of 153 dogs were genotyped in this study, with 46 cases (27 males, 19 females), 97 controls (36 males and 61 females), and 10 undetermined (6 males and 4 females). Dogs were classified as affected if they had slowly progressive cerebellar signs. When possible, magnetic resonance imaging and histopathological evaluation of the brain were completed as diagnostic aids. To identify genomic regions connected with the disease, genome-wide mapping was performed using both linkage- and association- based approaches. They found a region on CFA X strongly associated with the disease trait. Homozygosity mapping revealed a 4 Mb region of interest. Pedigree evaluation failed to identify the possible mode of inheritance due to the lack of complete litter information. The number of female cases was lower than males with an overall ratio of 0.7 females to 1 male, but still higher than expected for a recessive X-linked trait. The pedigree contains 3 litters with complete information where both parents were reported to be normal, and only male offspring were affected,

and the females were normal. In another family, the female offspring from an affected dam was normal, whereas the male offspring was affected. These families suggest the disease could potentially be transmitted in an X-linked recessive manner. However, there is 1 affected female that has normal parents, which is inconsistent with an X-linked recessive mode of inheritance, so an X-linked dominant trait with incomplete penetrance was considered. It is possible that the causal mutation of hereditary ataxia in Scottish Terriers is in an escaped gene on the X chromosome reflecting the apparent segregation of the disease in the population as an autosomal recessive trait. However, the identified region cannot be located in the PAR. The possibility that more than one form of hereditary ataxia is segregating within the Scottish Terrier breed, as was found in Parson/Jack Russell Terriers, has also been considered. Their findings suggests that further genetic investigation of the potential region of interest on CFA X should be considered in order to identify the causal mutation as well as develop a genetic test to eliminate the disease from this breed.Inherited Myopathy

Inherited Myopathy

Lujan Feliu-Pascual A., Shelton GD, Targett MP, et al. Inherited Myopathy of Great Danes. Journal of Small Animal Practice (2006) 47, 249–254.

- Onset of clinical signs is usually before one year of age, affecting both sexes. Clinical signs are characterized by exercise intolerance, muscle wasting, and an exercise-induced tremor. Most affected dogs have a severe form of the disease, although occasionally dogs may have a less severe form with an acceptable quality of life.
- In Great Danes, litters containing affected puppies are born to unaffected parents, and an autosomal recessive pattern of inheritance is likely.
- Elevated serum creatinine kinase concentrations and spontaneous electrical activity in skeletal muscles are frequently found.
- Studies have shown that it is a condition restricted to the skeletal muscles, with sparing of the cardiac and smooth muscle.
- Several of the inherited myopathies identified in dogs have a human counterpart, and direct comparisons can be made. For example, dystrophin-deficient muscular dystrophy is similar in clinical and histological phenotypes, and have similar mutations in the dystrophin gene. A centronuclear myopathy has recently been characterized in a French pedigree of Labrador retrievers. For some of the inherited myopathies, a human counterpart cannot be identified, such as the inherited myopathy occurring in great Danes.

For comparative purposes, the hereditary, non-inflammatory myopathy occurring in young great Danes with distinctive histological features should be reviewed and reclassified. This study reviews this inherited myopathy in great Danes and describes the currently available information necessary to facilitate recognition and diagnosis. Clinical data from 25 histologically confirmed affected dogs were reviewed. Onset of signs were from six to 19 months of age, with both sexes affected. The clinical presentation was progressive exercise intolerance, muscle wasting, abnormal posture, and body tremor. Noticeable respiratory effort were observed in two cases. All the cases were fawn or brindle in colour. Physical examination revealed mild to moderate, generalized muscle atrophy, particularly of the biceps femoris, quadriceps, temporalis, gluteal, supraspinatus, and infraspinatus muscles. There was no myalgia. Affected dogs were often stunted and had a characteristic posture with the pelvic limbs tucked under the abdomen and an extended tail carriage. Tremor was evident when standing and became more marked with movement and excitement. Affected dogs had a short-strided and stiff pelvic limb gait, with 'bunny hopping' apparent at faster gaits. The neurological examination revealed reduced patellar reflexes in two cases and decreased segmental spinal reflexes in two other cases, but was otherwise unremarkable. Bloodwork results were normal. Serum creatine kinase (CK) activity was typically increased, but there were also mild elevations or normal concentrations. Electromyography (EMG) was performed in 18 animals. Fibrillation potentials and positive sharp waves were found in all skeletal muscles examined in 16 dogs. Two cases developed muscle tremors under propofol infusion anaesthesia but were not hyperthermic.

Confirmation of the diagnosis of this inherited myopathy of great Danes (IMGD) depends on histological and histochemical evaluation of muscle biopsy specimens.

An important feature of the disease is its association with malignant hyperthermia, an autosomal, dominant, hypermetabolic disorder of skeletal muscle that is triggered by inhalational anaesthetics and depolarizing muscle relaxants. Malignant hyperthermia is also caused by mutations in the RYR1 gene in human beings. The presence and persistence of a well-defined clinical entity in a related group in a single breed is strongly suggestive of a genetic basis. In the future, the establishment of a genetic screening test to identify carriers and facilitate breeding programmes would be of value in controlling this disease.

Swimmers Syndrome

Sun-A Kim, Ki-Jeong Na, Jong-Ki Cho, Nam-Shik Shin. Home-care treatment of swimmer syndrome in a miniature schnauzer dog. Can Vet J 2013;54:869–872.

- Swimmers syndrome is also known as swimming-puppy syndrome, flat-puppy syndrome, swimmer puppy syndrome, flat-pup syndrome, twisted legs, or turtle pup, is an uncommon developmental deformity of newborn dogs (and cats).
- The limbs, often the hind limbs, are splayed laterally and they have difficulty standing or walking. Forward movement with this affliction is achieved by lateral pedaling motions.
- This syndrome is generally considered untreatable. However, there have been cases in which clinicians successfully treated a kitten and 3 puppies suffering from the syndrome with a combination of physiotherapy, bandaging, external splinting, and hospitalization.

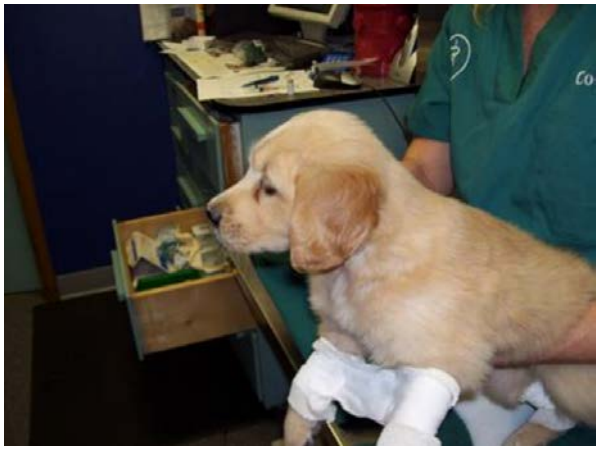
A 50-day-old, female miniature schnauzer dog was presented for astasia, dorsoventral flattening of the thorax, hypoplasia of hind-limb muscles, stiffness of hind-limb joints, paddling leg motion, and panting; diagnosed with swimmers syndrome. Neither of the hind legs could be placed in a normal standing position even with manual support. At rest, the dog remained in sternal recumbency; if positioned in dorsal recumbency the dog was unable to right herself. The owner explained that the dog was smaller than its littermates and that this, combined with its immobility, meant it had difficulty feeding. Moreover, the owner noted that the dog regurgitated its food after every feed, which contributed to the dog's low weight of 850 g when she arrived at the hospital. In addition, the dog's ventrum was covered with erosive lesions from urine and fecal scalding. The dog recovered completely following 40 days of home-care treatment that involved environmental and nutritional management along with intensive physiotherapy.

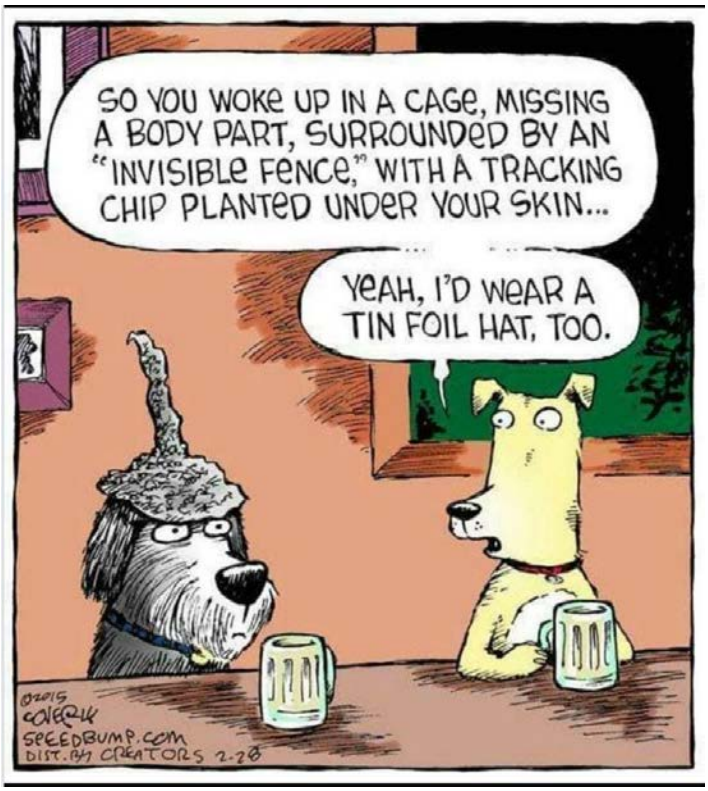
As an environmental modification, a non-slippery floor was constructed with a cotton carpet, and an absorbent pad, which was frequently changed, was placed on the bed. In addition, the ventrum was wiped regularly with a wet cloth, and baby powder was applied regularly to prevent further erosions. A mixture of 2 pet foods — Science Diet Prescription p/d canned and a/d canned, 10 g each — was fed to the dog every 2 h, 9 times a day. After feeding, the dog was lifted upward and massaged gently from head to tail for 5 min to prevent regurgitation. A standardized physiotherapy routine was performed every 2 h for 40 d following admission. The dog was placed in dorsal

recumbency, and the physiotherapy session began with a gentle stroking massage from the head to the hind limbs for 20 s. Then, for the flat thorax, both lateral sides of the thorax were gently pressed and released using the palms of the hands, every 5 s for 1 min. For the hind limbs, an effleurage massage was applied from the hip to the digits for 20 s, followed by a kneading massage that was applied from the digits to the hip for 20 s. After the massage session, each of the hind-limb joints was put through a series of passive range of motion exercises in the sequence outlined. First, to improve digit flexion and extension, the practitioner gently flexed and extended the dog's digits for 10 s, while supporting the carpus with 1 hand and the digits with the other. Second, to improve the stifle flexion and extension, the practitioner gently flexed and extended the dog's stifle for 10 s, while supporting the distal femur with 1 hand and the tibia with the other. Third, to improve hip flexion and extension, the practitioner gently flexed and extended the dog's hip for 10 s, while supporting the proximal femur in 1 hand and the pelvis in the other. Fourth, to improve movement in the hip joints, the practitioner applied a gentle stroking massage with the fingers to both the dog's hip joints for 20 s. To improve rotation in the joints, gentle pressure was applied medially, and excessive pressure was avoided during all massages and passive range of movements. Finally, the dog was held in a normal standing position with all 4 limbs touching the ground for 1 min. After 40 d of treatment, the puppy was able to ambulate normally but experienced bilateral grade I medial patella luxation up to 1 y of age, after which the condition spontaneously resolved. The dog, now 10 y old, has not suffered from any health issues, other than those seen in the first year of treatment.

Physiotherapy is beneficial in increasing muscle tone and strength, activating limb coordination, and stimulating circulation of the tissues. The stroking technique exerts a calming and soothing effect that helps animals become accustomed to being touched, reduces tension and anxiety, lowers muscle tone, and thus serves as a useful way to start and to finish massage sessions. The effleurage technique reduces swelling and edema, removes chemical by-products of inflammation, maintains mobility of soft tissues, and stretches the muscles. Kneading increases circulation and lymphatic flow, mobilizes soft tissues, removes chemical by-products of inflammation, increases sensory stimulation and invigoration, relaxes the animal, and lowers muscle tension. Passive movement is the movement of a joint by external forces and is generally used when a patient is incapable of moving the joint on its own or when active motion may be injurious to the patient. The ability to maintain or increase muscle length and flexibility is one benefit of passive movements. Other benefits include the prevention of adhesions in articular capsules and joints that helps maintain joint range; improvement of articular nutrition by increasing synovial fluid production and diffusion; and maintaining mobility between different tissues. Furthermore, producing or maintaining normal patterns of movement and stimulating mechanoreceptors in joints, muscles, skin, and other soft tissues help to improve

proprioceptive awareness and increase circulatory and lymphatic return. Thus, physiotherapy includes massages and passive range of motion exercises to develop and strengthen muscles and joint flexibility. As muscles develop and are strengthened through therapy, animals will begin to self-correct their own walking. As our case shows, swimmers syndrome is treatable with appropriate home-care treatment that involves environmental and nutritional management along with intensive, diligent physiotherapy. This case has been followed for over 10 y, and is, therefore, an invaluable resource for information about the prognosis. Owner education for treatment is important, with emphasis on the importance of maintaining regular physiotherapy.





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