Universidade de Évora - Escola de Ciências e Tecnologia

Mestrado Integrado em Medicina Veterinária

Dissertação

Sacrococcygeal epidural injection for chronic pain management in dogs with lumbosacral stenosis: a retrospective study

Miguel Alexandre Ferreira de Magalhaes Freixo Lopes

Orientador(es) | Maria Teresa Oliveira

José Diogo Gonçalves dos Santos

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A dissertação foi objeto de apreciação e discussão pública pelo seguinte júri nomeado pelo Diretor da Escola de Ciências e Tecnologia:

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ABSTRACT

Sacrococcygeal epidural injection for chronic pain management in dogs with lumbosacral stenosis: a retrospective study

Degenerative lumbosacral stenosis in dogs is thought to be a multifactorial disease, with both mechanical and inflammatory underlying causes. Because of the decreased quality of life of the dogs affected by this condition, an accurate diagnosis and a directed and effective treatment should always be put in action. It is also important to keep investigating the pathogenesis of degenerative lumbosacral stenosis, since with better understanding of the disease, better and more accurate treatment options can be put in practice in the veterinary clinical routine.

This dissertation is composed of a literature review about canine degenerative lumbosacral stenosis, as well as a retrospective study to assess the outcome of a sacrococcygeal epidural injection of triamcinolone, lidocaine, and morphine for chronic pain management in six dogs affected with this condition, which was evaluated at one and three months after the treatment.

Although it was not possible to prove an improvement of most clinical signs after the treatment, the results of this study are encouraging since the efficacy of the sacrococcygeal injection was demonstrated and the overall quality of life seemed to improve in all six dogs.

Keywords: Dog; Lumbosacral stenosis; Sacrococcygeal; Epidural; Chronic pain.

RESUMO

Injeção epidural sacrococcígea para controlo de dor crónica em cães com estenose lombossagrada: um estudo retrospetivo

A estenose lombossagrada em cães é considerada uma doença degenerativa de origem multifatorial, em que existem fatores mecânicos e inflamatórios na sua génese. Devido ao impacto na qualidade de vida dos cães afetados por esta condição, é importante estabelecer um diagnóstico correto, assim como um tratamento eficaz e bem direcionado. É também importante que se aprofunde a investigação acerca da patogénese da estenose lombossagrada, uma vez que dispondo de um melhor conhecimento da doença, melhores tratamentos com maior eficácia poderão ser aplicados à prática clínica veterinária.

Esta dissertação é composta por uma revisão de literatura acerca da estenose lombossagrada canina degenerativa, assim como por um estudo retrospetivo para averiguar o efeito após um e três meses da administração de uma injeção epidural sacrococcígea de triamcinolona, lidocaína e morfina para controlo de dor crónica em seis cães afetados por esta condição.

Embora não tenha sido possível provar a melhoria de todos os sinais clínicos após o tratamento, os resultados deste estudo mostraram-se encorajadores, uma vez que a injeção sacrococcígea se demonstrou eficaz e, numa perspetiva geral, a qualidade de vida de todos os cães melhorou após o tratamento.

Palavras-chave: Cão; Estenose lombossagrada; Sacrococcígea; Epidural, Dor crónica.

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List of Abbreviations and Symbols

® - Registered brand

% - Percentage

> - Greater than sign

< - Less than sign

AF – Annulus Fibrosus

ASA – American Society of Anesthesiologists

CBC – Complete blood count

CBPI – Canine Brief Pain Inventory

CNS – Central nervous system

COI – Canine Orthopedic Index

CSF – Cerebrospinal fluid

CT – Computed Tomography

DLSS – Degenerative lumbosacral stenosis

ESI – Epidural steroid injection

FPGA – Force plate gait analysis

GAG – Glycosaminoglycans

GSD – German-Shepherd dog

HRQL – Health-related quality of life

HCPI – Helsinki Chronic Pain Index

HVCLC – *Hospital Veterinário Central da Linha de Cascais*

IASP – International Association for the Study of Pain

IVD – Intervertebral Disc

Kg - Kilogram

KGA – Kinematic gait analysis

LOAD – Liverpool Osteoarthritis in Dogs Questionnaire

LOR – Loss of resistance

LS – Lumbosacral

LSS – Lumbosacral stenosis

MRI – Magnetic resonance image

NP – Nucleus Pulposus

OA – Osteoarthritis

OCL – Occypital-coccygeal length

PG - Proteoglycans

PO – *Per os*

SCo – Sacrococcygeal

WSAVA – World Small Animal Veterinary Association

Preface

This dissertation was written for the conclusion of the veterinary medicine integrated master's degree at the University of Evora, Evora, Portugal.

The author went through two externships, the first at *Hospital Veterinário Muralha de Évora* and the second at *Hospital Veterinário Central da Linha de Cascais – Vetoeiras*. At *Hospital Veterinário Central da Linha de Cascais - Vetoeiras*, it was possible to follow the administration of sacrococcygeal epidural injections for pain management in dogs with degenerative lumbosacral stenosis. In both externships, the author had the opportunity to follow medical and surgical cases in small and exotic animals, perform several procedures and therefore, gain valuable practical knowledge for the future practice of veterinary medicine.

I) Literature review

1) Lumbosacral stenosis

1.1. Lumbosacral anatomy of the dog

1.1.1. The vertebral column

The lumbar and sacral portion of the vertebral column are composed of the seven lumbar vertebrae and the three sacral vertebrae. [1–3] In the adult, the three sacral vertebrae are bonded together forming one bone, the sacrum. [1,2] Both lumbar and sacral vertebrae protect the spinal cord segment as well as the roots of the spinal nerves that compose the lumbosacral region. [2] The vertebral bodies, adjoining intervertebral discs, the longitudinal ligaments, *ligamentum flavum* and the articular processes and their respective joint capsules offer the vertebral column a stable configuration. [4] Active stabilization of the lumbosacral motion segment is secured by the epaxial and hypaxial paravertebral muscles whose activity is coordinated through proprioceptive input. [2] Abdominal musculature also plays a role in active stabilization of this vertebral segment. [2]

Between two adjoining vertebrae, the intervertebral disc (IVD) firmly attaches the adjacent vertebrae together and it is essential for maintaining stability. [2,5,6] This effective attachment between vertebrae also provides sustenance for the whole axial skeleton and, at the same time, admits motion in different plans. [5,7] The IVD's center, known as the nucleus pulposus (NP), consists of a gelatinous material which is surrounded by multiple laminae of well-organized fibrous tissue, the annulus fibrosus (AF), which gives great stability and protection to the internal gelatinous nucleus (Fig. 1). [6,8] The NP acts as a shock absorber when compressive forces act upon the spine. [6] Its gelatinous texture can

contain up to 88% water, being the remaining percentage composed of type II collagen and negative charged proteoglycans (PG). [5–7] Hyaluronic acid gathers these highly charged PG, producing very large and highly negative charged agglomerates. [5–7] This will induce a high osmotic pressure, granting the attraction and retaining of water and some nutrients into the NP by both osmosis and diffusion. [5–7] The most peripheral layers composing the IVD are thin structures made of hyaline cartilage, the cartilaginous endplates, which act as semipermeable membranes allowing nutrients to enter the disc's most internal layers by diffusion or osmosis. [6,7]

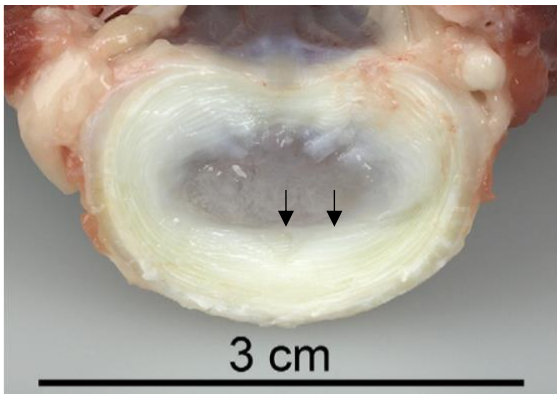


Fig. 1 Transverse perspective of a healthy canine IVD presenting a well-defined distinction (black arrows) between the AF and the NP (adapted from Meij & Bergknut, 2010).

The vertebral arch and body of each vertebra form the vertebral foramina, which all together create the vertebral canal. [2] Within the vertebral canal, the epidural space surrounds the spinal cord, the meninges, and the cerebrospinal fluid (CSF). [1,9] Also, between each pair of adjacent vertebrae it is formed the right and the left intervertebral foramina through which travel the respective spinal nerves, and blood vessels. [2] These lateral L7-S1 intervertebral foramina aren't just apertures but rather two tunnels, where the L7 nerve roots travel through from the inside of the vertebral canal to its exterior. [4]

In the sacrum, the sacral canal takes the place of the vertebral canal, where the final portion of the medulla runs through in some smaller breeds. [6] Two pairs of dorsally located sacral foramina and two pairs of pelvic (ventral) sacral foramina are located laterally to the fused vertebral bodies of the sacrum. [2] Besides the blood vessels, the dorsal and pelvic foramina also conduct the dorsal and ventral branches, respectively, of the first two sacral nerves. [2]

The L7-S1 segment allows for lateral bending and torsion motion, but most importantly for flexion-extension of the joint. [2,8] In the dog, a healthy L7-S1 configuration allows a superior flexion-extension range of motion when compared to other lumbar segments. [6] However, in dogs where degenerative lumbosacral stenosis (DLSS) is present, the flexion-extension mobility is reduced. [6,10]

The first coccygeal vertebra is articulated to the sacrum's caudal extremity, known as the apex, (Fig. 2) although it can sporadically be fused to the sacrum. [2]

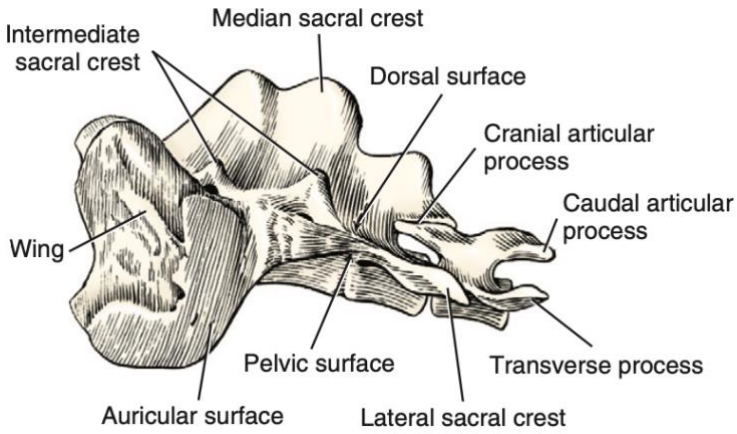


Fig. 2 Sacrum and first coccygeal vertebra, lateral view (Miller's Anatomy of the Dog, 2012)

1.1.2. The spinal cord

The brain and the spinal cord constitute the central nervous system (CNS). The vertebral canal contains the spinal cord, as well as the dorsal and ventral spinal roots that are part of the peripheral nervous system. [1,11] Both spinal cord and spinal roots are shrouded by three protecting coatings, the *meninges*. [1,11] The *dura mater*, the most superficial layer, is a thick and fibrous membrane that extends caudally past the termination of the spinal cord creating a structure known as the dural sac (Fig. 3). [1,11] The *arachnoid membrane* lies on the internal segment of the *dura mater*. [1,11] A subarachnoid space lies deep to the arachnoid membrane where the CSF is contained. [1,9,11] Arachnoid trabeculations cross the subarachnoid space attaching to the pia mater. [1,11] The *pia mater* is the innermost meninx, having the greatest blood vessels irrigation and being attached to glial cells located on the spinal cord's surface. [1,11]

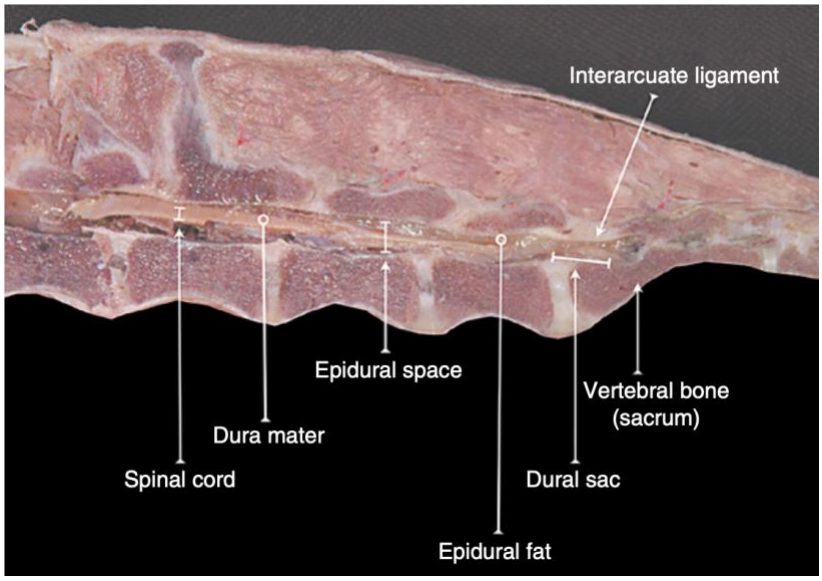


Fig. 3 Lateral perspective of the lumbar spine of a dog (Campoy's Small Animal Regional Anesthesia and Analgesia, 2013).

In newborn dogs, the spinal cord's length may reach the sacrum, while after growth, some authors state that it terminates in the caudal lumbar region. [11]

While some authors suggest the final portion of the spinal cord, known as *conus medullaris*, usually terminates close to the L6-L7 IVD, with exception of smaller breeds where it might extend further caudally, [1,3,6,11] there is disagreement in regard of the location of the dural sac in the veterinary field. [12] While Khan *et al* detected that in most of the adult dogs studied, the dural sac reached the lumbosacral (LS) space or extended further caudally to it, [13] other authors concluded that the dural sac reached the LS space in only 66% of Cavalier King Charles Spaniels. [14] A recent study by Zapata and colleagues also investigated the correlation between body weight and the position of the dural sac, observing that the dural sac terminated caudal to the LS space in 72.7% of the dogs in the <10 kg category and, although in lower percentage, it also reached the sacrum in the 10-20 kg and >20 kg categories (25% and 15.4%, respectively). [12] In the same study, brachycephalic dogs did not correlate to a further extension of the dural sac when compared to other non-brachycephalic breeds, although the authors do not rule out this hypothesis. [12] The *conus medullaris* consists of spinal cord segments S2, S3 and Ca1 to 5, which are enclosed by spinal roots pointed caudally (Fig. 4). [6,11]

In the vertebral canal, the structure known as *cauda equina* is composed by the sacral and caudal spinal roots that extend caudally, past the *conus medullaris*, then exiting at their corresponding intervertebral foramina. [11] *Cauda equina* is an important structure of the lumbosacral region, since it is composed of many spinal nerves extending from L6 to Cd5. [4,6,11,15] In the canine example, most of the *cauda equina* is located posterior to the lumbar cistern and its roots are independently wrapped by the meningeal layers. [11]

The nerve roots from the last lumbar vertebra travel from the lateral recess to the intervertebral foramen cranial to the IVD through a dorsolateral notch located at a caudal perspective of the L7's dorsal body. [4,15]

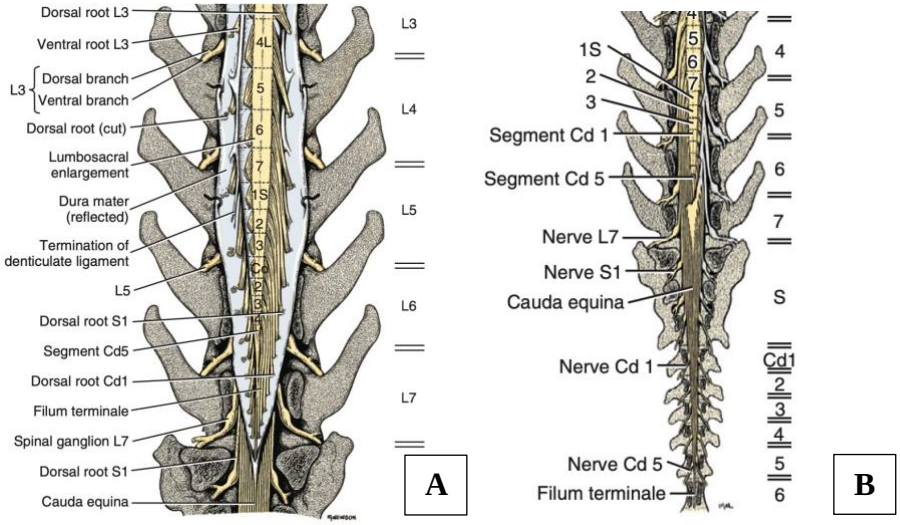


Fig. 4 Expanded view of the caudal section of the spinal cord, presenting a reflected dura mater (**A**), and nerve extension ending in the *Filum terminale* (**B**) (Miller's Anatomy of the dog, 2012).

1.1.3. Lumbosacral spinal nerves

From all 36 pairs of spinal nerves that usually exist in the dog, there are, on each side, seven lumbar nerves and three sacral nerves. [16] A spinal nerve comprises four sections: the roots, the main trunk, four primary branches, and several peripheral subdivisions. [16] Since the vertebral spine and the spinal cord keep growing at distinct speeds, the spinal cord's length is shorter when compared to the total length of the vertebral canal. Therefore, the latest lumbar, sacral and caudal nerves travel progressively longer lengths before they travel through their respective intervertebral foramina to leave the vertebral canal. [1,3,16] The spinal nerves typically abandon the vertebral canal through the intervertebral foramina. [16]

The last five lumbar nerve ventral branches and all the sacral nerve ventral branches merge to create the lumbosacral plexus, from which the nerves of the hindlimb originate (Fig. 5). [16]

The sacral nerves (Fig. 5) part from the three sacral sections of the spinal cord via extensive dorsal and ventral roots, because these spinal cord segments compose the portion of the *conus medullaris* that is located in the vertebral foramen of the fifth lumbar vertebra. [16]

While the lumbosacral plexus comprises the communicating ventral branches of the latest five lumbar nerves and the three sacral nerves, it may be separated into lumbar and sacral plexuses, although both always communicate. [16]

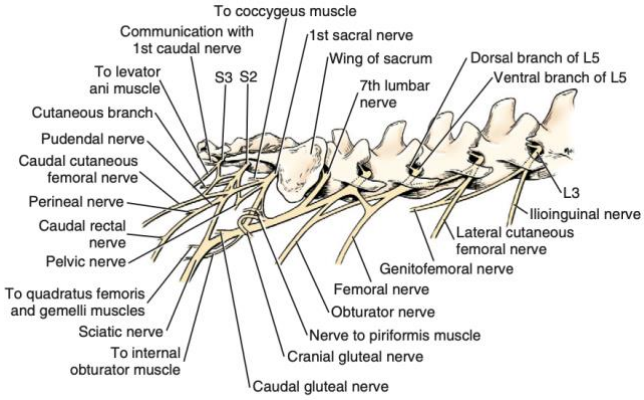


Fig. 5 Diagram of the lumbosacral plexus, right lateral view (Miller's Anatomy of the Dog, 2012)

1.1.4. The epidural space

Within the lumbosacral region vertebral canal, a virtual space referred to as “epidural space” lies between the spinal cord’s *dura mater* and the wall of the vertebral canal. [1,11] The space comprises adipose and connective tissue and, particularly by the floor of the canal, the internal ventral vertebral venous plexus. [1,9]

The vertebral canal’s dimension indicates the size and form of the inner spinal cord, since the dog’s vertebral canal contains little amount of epidural fat. [2] The LS intervertebral

space and the epidural space reach their largest at this location, so epidural infiltrations are often performed in at this location in small animals, since it provides the practitioner with a greater chance of achieving a successful instillation. [1,9] However, recent studies in veterinary medicine have demonstrated that, in some cases, using the sacrococcygeal (SCo) space for this purpose may be of better benefit. [17,18] The biggest strength of this approach relies on the lower risk of accidental thecal sac puncture and intrathecal injection when compared to the LS approach, [17,18] which has also been related in human medicine. [19]

The canal's shape remains almost unchanged in both L6 and L7 vertebrae and at this location it is larger than in any other vertebra caudal to T1. [2] The lumbar enlargement of the vertebral canal houses the LS enlargement, intumescence, of the spinal cord. [2] This enlargement is possibly caused by the small lumbar subarachnoid cistern, epidural fat, and the *cauda equina*. [2]

1.2. Canine degenerative lumbosacral stenosis

In 1989, Chamber referred to "degenerative lumbosacral stenosis" as a "syndrome of acquired narrowing of the vertebral canal, intervertebral *foramina*, or both, resulting in a compressive radiculopathy of the *cauda equina*". [4] A stenosis of the lumbosacral vertebral canal involves degenerative changes of the lumbosacral spine that lead to decreased available space for neural and vascular elements. [20] LS stenosis is a degenerative multifactorial disorder [21] where IVD degeneration plays a significant role since it is known as the most frequent forms of IVD herniation. [5,6,22] There are several conditions described in the literature that may contribute for DLSS, involving both bone and soft tissues. Either way, a degenerative stenosis often involves compression of the *cauda equina* or spinal nerves and their blood supply and when this happens, lameness, pain, and other neurological signs may be present. [6,23] When undergoing an extension movement, there is a reduction on the size of the intervertebral foramina at the LS junction but when degeneration is also present, it will lead to an even more evident reduction that can entrap the L7 nerve roots. [4] Usually, the pain originates from nerve distress of both

mechanical compression and consequent inflammation. [24,25] The inflammatory process related to the progression of the disease appears to be associated to the presence of clinical signs. [26,27] Evidence of an inflammatory process, such as cytokines and other proinflammatory markers, was already detected in biopsies of perineural structures, and CSF of symptomatic human patients. [25–27]

1.2.1. Pathogenesis

Degenerative changes are considered aging processes which are strongly induced by canine genetics and are enhanced by biomechanical stress and trauma, amid further causes. [5] It is suggested that the degeneration of the IVD (L7-S1) is triggered by an abnormal repetitive motion on the lumbosacral joint usually due to stress, genetic or congenital abnormalities. [6] An initial degradation of the proteoglycans that compose the NP will lead to fewer nutrients and less water being channeled into the disc, which will eventually dehydrate and degenerate, resulting in loss of disc width. [6]

The forces applied on the unstable spinal joint draw the load bearing effort from the center of the IVD to the outer structures of the spine (facet joints and ventral side of the vertebral bodies). [6] For tension relieve at the joint, the peripheral AF thickens, inducing bone proliferation, such as osteophytes and ventral spondylosis. [6] This will lead to additional damage of the nutritional supply to the disc, prompting the degeneration and structural failure of the IVD. [6,7]

Dynamic compression of the *cauda equina* induced by the angulated facet joints leading to compression is also possible when ventral subluxation of the sacrum occurs. [3,6]

Besides bone proliferation and compression, other contributing factors for spinal stenosis include general soft tissue proliferation that may compress the *cauda equina* or its blood supply, compromising the vascular irrigation of the spinal nerves. [6,10,28] Some known examples of surrounding soft tissue proliferation contributing to lumbosacral stenosis are hypertrophy of the interarcuate ligament, swelled joints capsules, and epidural fibrosis. [6]

This happens in compensation for the lack of stability at the lumbosacral joint associated to ventral subluxation of S1 and misalignment of the facet joints. [6]

Alongside the loss of IVD thickness and AF resistance to compressive forces, degeneration of the IVD may lead to Hansen type I (nucleus pulposus degeneration and extrusion) or type II disc herniation which represent common causes to DLSS, being the type II (AF degeneration and protrusion) the most common. [6,22] Congenital symmetry irregularities concerning the vertebral column structure or even the presence of extra vertebrae also contribute to DLSS. [6] Sacral osteochondrosis is a rare finding in clinical practice but it is also described as a potential pathology contributing to DLSS. [5,29] The activation of cell-mediated inflammatory reactions promotes neovascularization and nerve ingrowth into the injured disc, increasing lumbosacral pain. [6]

IVD disease is a broad, non-specific term that suggests the degeneration of IVD with or without the presence of IVD herniation. [6] IVD herniation itself is considered a general term that comprises any kind of IVDD causing damage of the mechanical integrity with some portion of the IVD bulging usually into the vertebral canal. [6] IVD prolapse, or displacement are two other terms used as a reference to IVD herniation and they all suggest either IVD extrusion or protrusion. [6] IVD extrusion refers to the herniation of internal constituents of the disc, mainly NP, through the AF. [6] Therefore, when speaking of IVD protrusion it usually refers to the herniation of the AF afar from its anatomical borders, being usually related to fibroid metaplasia – Hansen Type II IVD herniation. [6] As degeneration of the IVD occurs, the intervertebral space is reduced, prompting the protrusion of the AF and a decrease in the volume of the lateral intervertebral foramina, which may compress the spinal nerves.

A 2016 study assessing inflammatory profiles in canine IVD degeneration has shown that prostaglandin E2 (PGE2) levels, and chemokine ligand 2 (CCL2) quantities in degenerated and herniated tissues were expressively superior when matched with those that were neither degenerated nor herniated. [30]

1.3. Prevalence

DLSS is mostly reported in medium to large breed dogs and some studies have shown a predisposition for German Shepherd dogs (GSD) [10,31] and other working dogs such as the Belgian Malinois and retriever breeds. [4,6,23,32,33] Dogs of middle to older age are more commonly affected and they are typically over 25 kg of bodyweight. [3,4] The male population seems to be overrepresented. [3,4,6,34]

Work-related stress and some breed susceptibility seem to be contributing factors to the development of canine DLSS, since police and military working dogs seem to be overrepresented. [4] Working dogs frequently practice climbing high surfaces, explore while standing on their posterior limbs, and bite and hold an individual by one of their arms. [4] These actions demand great lumbosacral flexion, making this population greatly exposed to repetitive strain and stress of the LS junction. [4,10] Besides, GSDs' predisposition to DLSS may also involve a smaller vertebral canal at the LS junction than other dog breeds, accordingly to a study from Germany. [35] A recent study intended to evaluate the prevalence of LS deformities in brachycephalic breeds presenting for problems unrelated to spinal disease demonstrated that French Bulldogs, English Bulldogs, and Pugs are overrepresented on LS congenital vertebral abnormalities and IVD herniation, although they may present neurologically normal. [36]
Non-chondrodystrophic breeds are more susceptible to AF protrusion (Hansen type II) of degenerated LS and caudal cervical IVDs at the age of 6-8 years old. [5–7,22,30]

1.4. Diagnosis

1.4.1. Clinical signs and physical examination

Gathering the history and clinical signs, followed by thorough neurologic and orthopedic examinations is the first approach to the diagnosis of DLSS.

Background of caudal lumbar or LS pain are the most common clinical signs in dogs presenting DLSS. [6,23,37] These signs are often reported by owners mentioning unilateral or bilateral pelvic limb lameness, hyperesthesia, or self-mutilation of the LS region or posterior limbs, struggle with rising, sitting or lying down, unwillingness for jumping or climbing, dragging of toes, a dropped tail, and urinary or fecal incontinence in severely affected dogs. [6,10,23] Since many of these affections may only be noticed following intensive exercise or play, [37] it may be advisable to have the owner bringing a video recorded at home where it might be easier to spot these manifestations. [6]

During clinical examination, it's important to assess the lumbosacral region by applying direct digital pressure on the LS space while observing for any signs of pain, since this is the most consistent finding in dogs with DLSS. [6,10] Back pain may be present if the dog shows hyperesthesia over the dorsal LS junction region, evasive behavior, vocalization, or even a violent response. [4] It is important to distinguish when the pain is induced by hyperextension of the hip joints and when it is induced by hyperextension of the LS segment, although both may be present. [6]

To investigate if LS pain tends to be lateralized to the left or right, each pelvic limb should be hyperextended while applying lumbosacral pressure simultaneously. [6] When the vertebral canal or the nerve root canal is narrowed due to herniation of an IVD, the motion range of the nerve root amid movement of the lower extremities is reduced. [38] Therefore, compression and traction on the nerve root originate radicular pain, prompting more evident clinical signs. In some cases of DLSS, where the stenosis affects the left or right L7/S1 nerves with radiating nerve root pain, the affected dogs present a nonweight-bearing pelvic limb as their most obvious symptom. [6] In such cases, lameness may be induced by both hyperextension of the affected limb and LS pressure. [6]

While DLSS has a neurologic component, some authors defend that affected dogs are usually more orthopedic patients than neurologic patients. [6] Since *cauda equina* is the affected segment in DLSS cases and it is less susceptible to compressive forces caused by stenotic alterations than the spinal cord itself, severe neurologic deficits are rare in these dogs. [6] Most of presumed situations of DLSS don't show neurological deficits but

rather tend to show clinical signs related to low back pain only. [4] Being so, canine patients with DLSS presenting ataxia and proprioceptive deficits should be further examined to rule out other serious pathological situations, such as degenerative myelopathy, herniation of a thoracolumbar IVD, discospondylitis, or neoplasia. [4,6]

The neurological signs, which can be observed in patients with DLSS, are related to lower motor neuron affection presented as pelvic limbs paresis, atrophy of muscles innervated by the sciatic nerve, diminished withdrawal or cranial tibial reflex. [6] Pseudo-hyperreflexia of the patellar reflex is also a possible neurologic alteration. [6,10,23] This reflex is not disturbed by LS disease, so this "patellar override" occurs because the muscle tone of the stifle extensors (femoral nerve, L4-L6) outcomes that of the flexors (sciatic nerve). [6]

1.4.2. Imaging diagnosis

To confirm the diagnosis, imaging techniques are necessary. Simple radiography is often the first approach, even if it only allows to rule out bone-associated pathologies, such as neoplasia, discospondylitis, traumatic injuries, or vertebral anomalies. [4]

Computed tomography (CT) and magnetic resonance imaging (MRI) may both be classified as gold standard diagnostic tools for the identification of DLSS. [6,26] Even though these tools have become broadly available, there are still many veterinarians that rely on routine radiographic techniques, which do not have the required sensitivity for the diagnosis of DLSS and therefore cannot exclude the existence of DLSS. [6]

Advanced diagnostic imaging techniques have given a huge contribute to the knowledge on DLSS, allowing veterinary teams to tailor different treatment approaches to each patient individually. [6]

Though imaging techniques are essential to reach a diagnosis of DLSS, several studies in veterinary medicine as well as in human medicine, point out that some individuals live with multiple disc protrusions while never presenting any clinical signs. [34] Therefore, when addressing DLSS, there's often a low correlation between clinical signs, pathology,

and imaging findings. [28,34] This seems to reinforce the hypothesis that inflammation itself plays a critical role in the manifestation of clinical signs in dogs with DLSS.

1.4.2.1. X-ray

This conventional radiography technique implies the patient to be placed in lateral recumbency to obtain the most information of the lumbosacral canal. [6]

Common findings include narrowing of the IVD space (Fig. 6), sclerosis of the joining facets of L7 and S1 (Fig. 7), LS step formation with ventral subluxation of the first sacral vertebra, ventral or lateral spondylosis, and the vacuum phenomenon. [6] The vacuum phenomenon, which consists in accumulation of nitrogen gas in a ruptured degenerated disc, is a commonly observed radiologic evidence of degenerative IVD disease often reported in human patients, although more rarely in animals. [4,6,39]

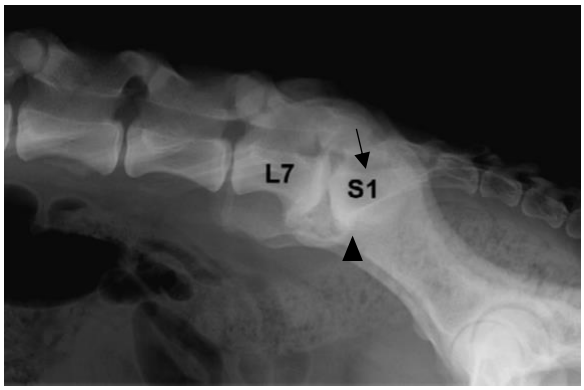


Fig. 6 Lateral radiographic view of the LS section in a canine patient with DLSS showing the collapse of the IVD space (black arrow) and end plate sclerosis (arrowhead) (adapted from Meij & Bergknut, 2010).

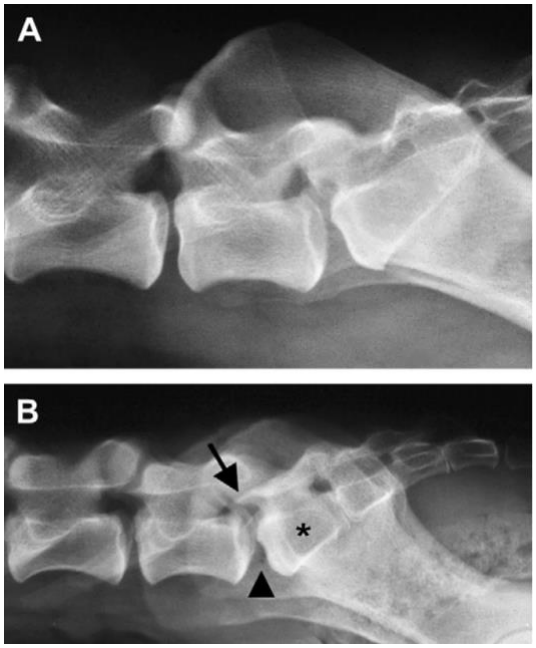


Fig. 7 Lateral radiographic view of the LS section. (A) Healthy dog. (B) Dog with DLSS and a transitional vertebra (asterisk), projection of the S1 lamina into the vertebral canal of the last lumbar vertebra (black arrow), and vacuum phenomenon between the last lumbar and first sacral vertebra (arrowhead) (adapted from Meij & Bergknut, 2010).

Simple radiography is an important and simple way to exclude bone neoplasia such as metastases from prostate carcinoma, and other anomalies like traumatic luxation or discospondylitis. [6]

To increase the sensibility of conventional x-rays, a radiography of the lumbosacral region taken in dynamic flexion/extension of the lumbosacral joint may be helpful to improve the lumbosacral step formation. [6] However, simple radiography lacks sensitivity for the diagnosis of DLSS whether because it is incapable of providing detail on soft-tissues, [23] therefore, delivering false-negative results. Also, degenerative patterns may be noted even when clinical signs are absent, delivering false-positive results. [40] Furthermore, a report on German-Shepherd working dogs with and without DLSS, showed evidence regarding small to no correlation between simple radiographic findings and the progression of DLSS. [41]

1.4.2.2. Myelography

The injection of a nonionic contrast medium is used for injection into one of two possible sites: the subarachnoid space at the cerebellomedullary cistern or between L5 and L6. [6,23] Even though the efficacy of myelography to diagnose DLSS is more reliable than conventional radiography, its usefulness is still questionable since it relies on the projection of the dural sac into the LS junction. [6,23] The spinal cord usually extends to L6 and the dural sac may extend even more caudally. [6] Still, this technique's sensitivity can be improved by applying both flexion/extension at the lumbosacral joint during the radiographic study, making myelography an effective technique in the diagnosis of DLSS. [6]

1.4.2.3. Epidurography

Epidurography is known for being an easier technique when compared to a myelography. [6,23] To obtain an epidurography, a contrast solution must be applied by injection inside the epidural space either at the LS or sacrococcygeal (SCo) site. [6,23]

Placing the patient in lateral recumbency to obtain a lateral view is ideal to obtain a more informative epidurogram. [6] Expectable findings in epidurograms in canine patients with DLSS include narrowing, arise, eccentricity, or blocking of the epidural contrast-medium marks. [6] These are more evident once an associated flexion and/or extension motion is made. However, epidurography lacks sensitivity in the diagnosis of lateral compressive lesions. [6,42] Besides, overlaying of structures, presence of adipose tissue, deficient filling, and leaking by the intervertebral foramina make the analysis of the epidurogram challenging and that's why it is rarely used, especially when there is an increased availability of CT and MRI. [6]

1.4.2.4. Discography

This technique is performed by injecting the contrast medium into the NP through the dorsal aspect of the AF. [6,23] Leaking into the degenerated disc may indicate a diagnosis of DLSS. [6,23] However, disc puncture itself can induce a disc degeneration [23] and that's why discography is controversial and a rarely used procedure, since CT and MRI have become broadly available. [6]

1.4.2.5. Computed tomography

CT-scan offers greater soft-tissue contrast resolution when compared to regular radiography as well as allowing transverse (axial) image orientation. [4,6,23] CT's possibility of obtaining reconstructed images to assess sagittal, dorsal, or oblique sections as well as 3-dimensional reconstructions make it great tool for the diagnosis of DLSS. [6,23] Findings in conventional radiography and CT are similar but the last allows the identification of Hansen type II disc herniation, interarcuate ligament hypertrophy, and joint capsules' integrity. [6] As in conventional radiography, it is important to position the animal with an extended LS junction, so that the dynamic element of disc protrusion and telescoping of S1 won't be unnoticed. [4] Worth *et al* published a study whereby applying dynamic extension on a flexed LS junction from an initial flexed position, a reduction of the mean foraminal volume was detected by 79% in GSDs where DLSS was absent and by 85% in GSDs where DLSS was present. [15] When extending the LS joint, the foraminal volumes decreased in GSDs in which DLSS had been identified when compared to GSDs that did not present clinical signs. [15]

Transverse views allow the identification of entrapped and bulged nerve roots and distension of the dural sac exactly before and after the stenotic defect. [6] The intervertebral foramina between the last lumbar and the first sacral vertebra can be properly evaluated in the parasagittal, dorsoplanar, and transverse views. [6] When present, disc protrusion can be noticed from the center or more eccentric and it can present as a moderately affected IVD (less than 50% of the vertebral canal's diameter) to a severely affected IVD (more than 50%). When IVD protrusion and hypertrophy of the interarcuate ligament are

present, epidural fat may lose its protective role of the dural sac and local nerves. [6] Dogs presenting symptoms of DLSS, frequently display a reduced amount of epidural fat protecting the nerve roots, as well as a more evident soft tissue opacity obliterating the intervertebral foramina, a bulged AF, spondylosis intruding the foramina, dural sac dislocation, reduced L7-S1 lateral intervertebral foramen diameter, a tightened LS spinal canal, thickened articular processes, and osteophytosis of articular bone structures on CT images. [4,23]

Even though CT is a high standard diagnostic tool, it's still considered less sensitive than magnetic resonance image (MRI) for discriminating conditions that do not involve mineralization of extradural material or lysis of bone, as well as other soft tissues within the spinal canal. [6,26,43] CT has greater sensitivity for soft-tissue calcifications, cortical bone spurs, and degeneration processes of the facet joints, when compared to MRI. [6] Other advantages of CT over MRI are lower cost, simpler maintenance, associated expenses, and faster imaging. [43]

1.4.2.6. Magnetic Resonance Image (MRI)

Correct positioning is particularly important in MRI to improve specificity by avoiding artifacts. Positioning the dog in dorsal recumbency, as standardized for spinal segments, while extending the LS segment with help from positioning foam holders and sandbags allows an increased diagnostic sensitivity. [44]

MRI and CT findings are similar in canine DLSS. [6] However, MRI delivers further comprehensive information on IVD degenerative changes, as well as alterations at the dural sac and spinal cord in general. Nerve root displacement and defective epidural adipose tissue are also more evident on MRI. [6,23] Signal intensity varies accordingly to the concentration of hyaluronic acid and glycosaminoglycans (GAG) which draw and retain water. [6]

Degeneration of the NP is more evident on MRI than on CT images and it is perceived as a less brighter signal in T2-weighted images. [4,20] Epidural fat is easily identified in MRI

since it acquires a high signal intensity appearing as bright white. [6] So when epidural fat is lost, there is an attenuation of the normal signal, possibly indicating compression of the nerve root at its respective foramen.

Nerve root compression is more evident in MRI than in CT and it was observed in 68% of the population in a study by Mayhew *et al*, about MRI findings in dogs with DLSS. [45]

1.4.3. Differential diagnosis

When addressing canine DLSS, pathognomonic features lack, and frequently a presumptive diagnosis is made centered on clinical signs, advanced imaging findings, and ruling out other causes that may promote compression of the *cauda equina*. [4,10] The condition known as "*cauda equina* syndrome" indicates the manifestation of clinical signs that are triggered by a damage concerning the *cauda equina*'s nerve roots, or a lesion distressing the L5-L7, sacral or coccygeal vertebrae, or surrounding soft tissues that result in compression of the *cauda equina*. [4] Therefore, when *cauda equina* syndrome is detected, DLSS must be included in a differentials list. [10] Several conditions such as neoplasia, discospondylitis, epidural empyema, and other compressive formations must be ruled out as a cause when addressing *cauda equina* syndrome or a suspected DLSS. [4,6,10] Other conditions may suggest the presence of DLSS by showing similar clinical signs, typical age range, or a predisposed breed and must be included in the list of differentials. [6] Some examples are cranial cruciate ligament rupture, hip dysplasia, psoas muscle injury, and contracture of the gracilis, and semitendinosus muscles. [6]

Apart of orthopedic conditions and if neurologic deficits are evident, other differentials such as degenerative myelopathy, thoracolumbar IVD disease, severe discospondylitis, and neoplasia, such as peripheral nerve sheath tumor, must be considered. [6]

1.5. Therapeutic

There's little evidence on which to support the correct treatment approach for dogs suffering from DLSS. In dogs presenting mild symptoms, a conservative approach is usually the first step, by means of analgesic and anti-inflammatory medication and by adjusting the animal's routine to prevent aggravation of clinical signs. [4]
Momentary pain relief can be achieved by administering either non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroids. However, clinical signs often relapse when/if these drugs are discontinued. [4]

1.5.1. Conservative treatment

Conservative treatment is considered when clinical signs are mild and pain control is considered a sufficient option. However, this approach does not eliminate the underlying problems such as IVD disease or other predisposing factors to DLSS. Studies of conservative treatment approaches lack representative numbers of dogs, and the existing ones report unexceptional and temporary results. [34,46–48] Effects are reported to last only while medication is administered, and exercise restrain is maintained. [34]

A conservative therapy for DLSS usually includes the usage of NSAIDs combined with an adjustment in exercise routine and body weight drop. [6,34] The success of conservative treatment seems to support the theory that the inflammatory process behind DLSS may have an important responsibility in the progression of clinical signs since the mechanical compression is not affected by this therapeutical approach. [34]

Systemic use of corticosteroids is frequently avoided since it is possible to obtain a similar degree of analgesia using NSAIDs, which are related to fewer side effects. [6] Alongside any DLSS conservative treatment, it is recommended to restrict the patients' exercise pattern. Restrictions involve frequent but brief leash walks to avoid muscle mass loss and therapeutic regular walking on an underwater treadmill may also benefit recovery.

In a more recent retrospective study published by De Decker *et al* 55% of dogs with DLSS were successfully managed with modification of lifestyle to avoid strenuous exercise and use of anti-inflammatory and analgesic drugs. [48] However, in 32% of the dogs, conservative treatment failed, and surgical decompression was followed, while euthanasia was carried on in 10% due to further intensification of clinical signs. [48] Epidural injection of corticosteroids was not tested in this study.

1.5.1.1. Epidural steroid injections

While in human medicine the designation "epidural steroid injection" (ESI) is broadly used, in veterinary medicine the term has not been established yet, although it seems appropriate for veterinary patients. [21] In human medicine, the data firmly indicates that ESIs can offer short temporary relief of radicular symptoms, while being less consistent for long-term relief. [46,49] Still, they are used to diminish inflammatory processes, relief pain, and reduce additional medications or avoid a surgical approach. [25,49]

The reasoning behind this therapy seems to rely on the mitigation of the effects of local inflammatory substances such as cytokines, 70 heat shock protein, nitric oxide and others, however, since the exact mechanism behind the response remains unexplained, a multifactorial effect seems most likely. [21,25,34,50] Epidural administrations of corticosteroids allow the veterinary surgeon to apply the anti-inflammatory drugs right onto the inflamed region, theoretically avoiding systemic effects and superior local doses than oral medication. [21,51] Besides providing an immediate result post treatment, these advantages offer the possibility of ESI to be implemented as a treatment, a diagnostic assessment and even as a prognostic factor of later outcome post-surgical management of DLSS. [21]

Epidural injections can be administered at the LS junction or at the SCo junction. [52] In human medicine, three routes are described for epidural steroid administration: transforaminal, interlaminar, and caudal approach. [49,53]

Although epidural injection of drugs is considered a simple procedure, [34,54,55] precise needle position in the epidural space depends on the performer's experience [56] and there are several methods reported in the literature to assist in epidural space identification. [57] Ultrasonography, [58] electrical nerve stimulation, loss of resistance (LOR), and the "hanging drop" technique are considered less intrusive than epidurography which makes their practice more appropriate for clinical patients. [59–61] Although the "hanging drop" technique and LOR are among the most used methods, controversies regarding their specificity and sensitivity exist. [9,59] Epidurography is considered one of the most useful procedures to confirm correct needle placement in dogs, but side effects after injection of contrast medium may somehow constraint its use in some routine practices. [59] Ultrasound-guided parasagittal epidural access may be a viable method in regular dogs, but also in dogs with LS radiographic irregularities, and in overweighted dogs. [9,62] The observation of epidural pressure waves confirmation technique may be considered for dogs participating in clinical trials, since it is less intrusive than epidurography and has been studied demonstrating acceptable success rates. [59] Electrical nerve stimulation has been used as a confirmation technique in previous clinical trials and a specificity of 93% and a sensitivity of 74% in the LS location has been reported. [21,56]

SCo epidural injections of corticosteroids for treatment of DLSS in dogs are not widely reported but this location is also used for epidural injections and may be a better alternative than LS injections. [9,63] The most important advantage of this approach relies on the less likely chance of spinal puncture. [9] Ultrasound-guided technique for SCo epidural injections has been previously reported and considered promising. [63]

A recent study from the University of Sydney, Australia, verified that there was no significant change in the cranial extent of epidural injectates when using either SCo or LS approach. [18] The study then suggests that the SCo approach does not look to demand higher volumes to achieve an identical cranial range to that of the LS approach, and identical volume guidelines may be considered for both approaches. [18] Reports on how the migration of anesthetics in the epidural space is influenced in bovine and feline populations mention four major factors: physical individualities of each specie, technical

aspects, inherent anatomic factors, such as the distribution of epidural adipose tissue or configuration of the epidural veins, and epidural pressure. [64–66]

When epidural injections are administered, either methylprednisolone, betamethasone or triamcinolone is used. [34] The success rates of ESI in human medicine have encouraged further investigation in the veterinary field.

A 2009 retrospective study reported an improvement of clinical signs of 18.4% of the dog population after the first epidural injection of methylprednisolone acetate and an overall long-term reduction of clinical signs in 79% of dogs after more than one ESI. [34] Janssens *et al* also validated that ESI had only a transitory effect, often requiring several subsequent infiltrations to obtain longer effects. [34] In this study, the duration of treatment effects was 4-14 days (median of 11 days) in animals that underwent only one infiltration, 4-6 weeks (median of 32 days) in those who had a second infiltration, and one week to 46 months (median of 4.5 months) in animals with a third infiltration. [34]

A recent clinical trial assessed the usefulness of a single epidural instillation of methylprednisolone acetate for treatment of DLSS, while comparing the results of both ESI and decompressive surgery. [21] Gomes *et al* achieved similar results than previous studies whereas ESI appears to be an inferior option when compared to the surgical approach. [21] While surgical decompression seems to be superior to relief long-term clinical signs of DLSS, ESI seemed to produce a more consistent attenuation of clinical signs in the long-term in a subgroup of canine patients. [21] This supports ESI's relevant function in the management of DLSS once surgery is not a viable choice, or as a first approach treatment when the diagnosis is obtained. [21] Additional research is required to create a protocol that helps in the recognition of veterinary patients that may benefit in the long term to ESI alone. [21] For these injections to be effective, dogs must be cleared of any proprioceptive deficits in the pelvic limbs and not presenting urinary or fecal incontinence. Some reported side effects include a lower immune response with the surge of discospondylitis. [6]

Worth *et al* suggest epidural infiltrations are indicated when dogs present with clinical signs consistent with DLSS due to focal neuritis of L7/S1 roots and other differentials have been ruled out, and diagnostic imaging findings do not support diagnosis of DLSS (no or minimal compression). [4]

Because of potential collection of epidural fat in the epidural space of obese dogs, lean body weight or vertebral column length (from occipital condyle to Ca1) should be assessed when calculating the epidural dose. [1,47] The same applies to those presenting anomalous spinal column patterns. [47]

1.5.2. Surgical treatment

When it comes to surgery, there are two main approaches to address DLSS: decompressing the L7, sacral and/or caudal nerve roots; and distracting/stabilizing the LS segment.

Generally, surgery is recommended when dogs present with moderate to severe LS pain nonresponsive to medical therapy and in dogs where neurologic deficits are present. [4,6,34] Surgery may also be an option for working dogs in which DLSS limits their activity and restricts their lifestyle and well-being. [4,6] The purpose of the surgical approach is to decompress the *cauda equina* and relieve the entrapped nerve roots. Thanks to the broad availability of CT and MRI, many surgeons and neurologists can rely on cross-sectional imaging as the exclusive diagnostic tool for surgical planning. [43]

Accordingly to literature, decompressive surgery is the most broadly used technique for treatment of DLSS. The technique includes an incomplete dorsal laminectomy of L7 and S1 plus a dorsal annulectomy of the prolapsed L7-S1 AF. [4,34,67–69] Other studies report the use of dorsal decompression alone, with no annulectomy being performed, or a dorsal decompression and disc fenestration with partial discectomy of the L7-S1 IVD. [67,70] Worse outcomes in patients undergoing both incomplete discectomy and dorsal laminectomy compared to dorsal laminectomy alone still make discussable the validity

of the double technique. [31] Facetectomy is a different surgical option, and it implies the removal of the whole L7 caudal articular process and for that reason it is not recommended without concurrent stabilization of the LS junction. [4] Surgical stabilization of the LS junction alone is an alternative for the management of DLSS, using screws, pins, or bars. [4] Foraminotomy is a technique that aims for the decompression of the entire segment of the L7-S1 neurovascular foramen by bone removal. Though challenging approach technique and difficulty in controlling hemorrhage around the L7 nerve root resulting in fibrosis and consequent residual pain have all been reported, [4] some studies indicate good to excellent outcome in most of the dogs that underwent the lateral foraminotomy. [71,72]

2) Pain

2.1. Definition

The International Association for the Study of Pain (IASP) describes pain as "*an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage*" which is accepted for both human individuals and non-humans (Raja, Srinivasa N., et al. "The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises." Pain 161.9 (2020): 1976-1982).

Pain is a normal sensory function that helps an individual preventing from constant or acute harm. Nevertheless, as the sensation progresses into a predominantly chronic condition, it changes into a non-functional sensation that handicaps the individual, strongly reducing their quality of life (QOL). [73] Differently from pain, QOL underlies a concept that lacks a physiological source or any particular behavioral features and can be evaluated by analyzing different interrelating factors, either intrinsic or extrinsic to a single individual. [74] Some QOL features that vary with an illness/health status or with

medical treatment constitute may be understood as health-related quality of life (HRQL). [75]

Pain is commonly caused after a noxious stimulus occurs. However, some anomalies can originate an aberrant processing of neuronal signals, prompting an amplified and pathological pain condition, such as hyperesthesia (an exaggerated sensitivity to nonnoxious stimuli) and hyperalgesia (an increased painful reaction to mildly noxious stimuli). [76]

2.2. Pain pathophysiology

Pain happens when specific peripheral afferent neurons, known as nociceptors, are stimulated. Pain is processed through three distinct phases: transduction, transmission, and modulation. [76] Transduction is generated by an action potential once the nociceptors are stimulated by noxious stimuli of either mechanic, thermic, or chemical source, and is conducted essentially via myelinated A delta fibers and unmyelinated C fibers. [76,77] Both of these types of fibers are largely dispersed all over the body and allow different structures, such as the skin, subcutaneous tissues, periosteum, joints, muscles, and viscera to exhibit nociception functions. [76] Pain is conducted through A delta and C fibers which connect to the spinal cord's dorsal horn through the dorsal root ganglia, where the neurotransmitters travel by synapse with second-order neurons. [76,77] Following transmission, pain is modulated in the dorsal horn, by means of interactions with excitatory and inhibitory interneurons. [76] Modulation of pain occurs by several routes, often involving inflammatory mediators, such as phospholipase A2. [46] Phospholipase A2 plays a role in the conversion of membrane phospholipids into arachidonic acid [28] , a major substrate for lipoxygenase and cyclooxygenase pathways. [46,78] Phospholipase A2 exists in large amounts in human IVD, which seems to help justify why pressure, inflammatory processes, and extrusion of disc contents can lead to chronic back pain and radiculopathy. [22,79] The subsequent nociceptive stimulus travels through the spinothalamic tracts to the many areas of the brain, where integration takes place and where it is then processed and acknowledged as pain. [76]

2.3. Pain classification

Although pain is a universal sensation, classification is possible depending on its temporal extension (acute, chronic, or intermittent), intensity (mild, moderate, severe, or excruciating), and anatomic source (somatic, visceral, or neuropathic). [76]

2.3.1. Chronic pain

Chronic pain usually indicates a pain lasting longer than three months (WSAVA Global Pain Council, 2014), [52] persisting beyond healing or even when healing doesn't even occur. [74] Yet, it has more recently been presented a definition for *chronic primary pain* as "*pain in one or more anatomical regions that persists or recurs for longer than three months and is associated with significant emotional distress or functional disability and that cannot be better accounted for by another chronic pain condition*". [80]

While acute pain is useful for the individual to avoid a painful stimulus, chronic pain doesn't seem to attend a specific purpose (with the possible exception of minimizing additional damage where healing has not yet taken place) and behavior recognition may be challenging to identify. [74] This is particularly true when speaking of maladaptive pain, which can be stated as "*dysfunctional pain that neither protects nor supports healing or repair*". [81,82]

As dogs' lives last longer, an upsurge of incidence of painful chronic pain forms such as osteoarthritis and cancer has been noted. [52] Evidence of chronic pain in dogs is usually hard to recognize and reactions to treatment vary from each individual. [52]

The source of LS pain in dogs suffering from DLSS is often associated to direct nerve compression, where concurrent inflammation or injury to surrounding soft tissues, such as the AF, *ligamentum flavum*, dorsal longitudinal ligament and synovial membrane, occurs. [4] DLSS has been presumed to induce neuropathic pain, [28] and its clinical significance relies on an exaggerated presence of calcium channel (receptor) subunit alpha-2-delta, [4] substance P, [83] and calcitonin gene-related peptide [38] in dorsal root

ganglion of canine patients with DLSS. [4] Besides, the clinical outcome in canine patients with LS pain receiving gabapentinoid drugs, which modulate the receptor channel subunit alpha-2-delta, encourages the theory supporting the presence of neuropathic pain in DLSS. [4,84]

2.3.2. Chronic pain assessment

Recognizing pain is paramount to effectively manage pain. [52] Chronic pain and QOL are intimately connected, and their assessment is vital for delineating adequate therapeutical options and counselling owners. [74] Chronic pain has a broad range of effects which affect the QOL of the sufferer, whether it is a human or an animal. [75,85] Since pets cannot directly express a sensation of pain, it is important to develop alternate and trustworthy techniques to identify pain, such as structured questionnaires with formal scoring methodology. Currently, some tools have been developed to assess and classify chronic pain in canine patients and they have delivered knowledge regarding the range of variations in the demeanor, mood, and behavior of dogs due to chronic pain. [52] These often contemplate: (1) vitality and mobility (how energic, happy, active/lethargic, contended, playful is the dog; ease of lying, sitting, jumping, tolerance to exercise), (2) mood and demeanor (including states of alertness, anxiety), (3) levels of distress (e.g., vocalization, demeanor, and reaction to other animals and humans), and (4) indicator of pain (e.g., comfort levels, stiffness, lameness). [52]

Communication with the pet owner is a valuable aspect of pain recognition, especially when addressing chronic pain since behavioral variations may be very subtle and gradual. [52,75,76,85] Because pet owners usually recognize their pets' regular behavior better than anyone else, they are able to more easily identify subtle differences that might indicate pain or discomfort. [75,76,85] To simplify the perception of pain and its classification based on behavioral interpretations, a few certified observer pain scales have been applied to better classify pain in animals. [52,75]

2.4. Pain scales

Specialized queries with proper scoring methodology have been developed to assess pain in both people and animals, measuring the affective (emotional) element of the pain sensation. [75]

Up to date, literature doesn't mention any specific pain scale to address chronic pain associated to DLSS in dogs. However, several clinical metrology instruments are still being developed in veterinary medicine to assess chronic pain mostly related to osteoarthritis (OA) in dogs, such as the Canine Brief Pain Inventory (CBPI), the Helsinki Chronic Pain Index (HCPI), the Liverpool Osteoarthritis in Dogs questionnaire (LOAD), and the Canine Orthopedic Index (COI). [52,75]

The CBPI is mainly applied to evaluate increases/decreases in pain scores in dogs with either OA [86] and osteosarcoma. [52] The HCPI is an owner-applied query which has been employed to estimate chronic pain in dogs suffering from OA and, like the CBPI, it has been assessed for content validity, reliability, [87,88] and responsiveness. [52,87,89]

The LOAD has been certified in dogs with chronic elbow OA as well as both forelimb and hind limb OA and has been indicated to be reliable with acceptable sensitivity. [52,90,91] The COI was developed by the University of Pennsylvania with the objective of measuring owners' assessment of outcome in dogs with orthopedic disease. [92]

Although pain scores can help us to understand the severity of the pain, there are limitations on their use, such as respondent bias. [75] It is suggested that when an individual scores a patient's pain with a particular condition that the individual him/herself has experienced, more severely would he/she score the pain associated to that specific condition. [75] Another example may include a consciously or unconsciously owners that may bias their response for numerous reasons, such as the fear of euthanasia being suggested by the veterinarian. [75]

The use of force plates gait analysis (FPGA) has been reported as a more objective pain assessment method in some studies. [93,94] However, besides being a time-consuming method and not being commonly available in general practice, the combination of FPGA and kinematic gait analysis (KGA) is considered a more objective than visual gait examination or FPGA alone. [95]

II) SACROCOCCYGEAL EPIDURAL INJECTION FOR CHRONIC PAIN MANAGEMENT IN DOGS WITH LUMBOSACRAL STENOSIS: A RESTROSPECTIVE STUDY

1. Introduction and Objectives

Although better described in human medicine literature, epidural injections of corticosteroids in veterinary medicine are still poorly reported [4,21,34] and more studies with larger populations are necessary to determine their efficacy and when to prescribe them. This retrospective study aims to evaluate the mitigation of clinical signs in dogs with DLSS that underwent a SCo epidural infiltration of triamcinolone, morphine, lidocaine, and iodine dye.

To assess treatment efficacy, the HCPI was applied at three moments. The first was applied before the epidural injection to establish a basis clinical condition of each animal. Then, for a short-term follow-up, the HPCI was applied one month after the procedure, and lastly, for a long-term follow-up, it was applied three months after the procedure.

The main objective of the study was to verify if an epidural SCo injection of triamcinolone is effective in the improvement of the quality of life in canine patients with DLSS, for one and three months. Furthermore, in the attempt of excluding the possibility of systemic absorption of corticosteroids after epidural administration and misinterpretation of the results, an assessment of related side effects from corticosteroids administration was made. Owners were asked to evaluate water and food consumption,

urine production and frequency, and feces consistency. Lastly, owners answered whether they considered the treatment was useful in the improvement of the QOL of their dog or not.

2. Materials and Methods

2.1. Data collection

Epidural steroid injections were already administered as conservative treatment for DLSS at *HVCLC* as routine procedure. For purpose of the current study, the HCPI was applied in all dogs who presented at the hospital with a diagnosis of DLSS and in which the owners consented and complied with the follow-up queries, in addition to the habitual authorization for sedation and medical procedure.

Information regarding the sex, age, breed, and bodyweight of each patient was collected from the software used at HVCLC, Magnisoft® | OranGest v22 VET. Besides a Complete Blood Count (CBC) and a basis biochemistry panel (to assess renal and liver function), prior to the procedure, a list of clinical signs was listed for each animal in three different moments (pre-treatment, one month after treatment, and three months after treatment). The assessment of the clinical signs was made through the application of the HCPI which was adapted from English to Portuguese (Appendix I). The data of each patient was stored in a Microsoft® Excel (Microsoft Office, Version 16.54) data base after being collected by Google Forms (Google®). The queries were applied in person or sent by e-mail to the dogs' tutors.

2.2. Inclusion criteria

For the present study, all dogs presented at *HVCLC* between March 1st, 2021, and May 30th, 2021, with a confirmed diagnosis of DLSS were included. All dogs admitted with

compatible clinical with DLSS went through a neuro-orthopedic examination followed by a CT scan for the purpose of diagnosis confirmation (Fig. 8). Clinical signs included unilateral or bilateral pelvic limb lameness and at least one of the following: hyperesthesia, or self-mutilation of the LS area or pelvic limbs, difficulty with rising, sitting or lying down, reluctance to jump or climb, dragging of toes, a low carriage of the tail.

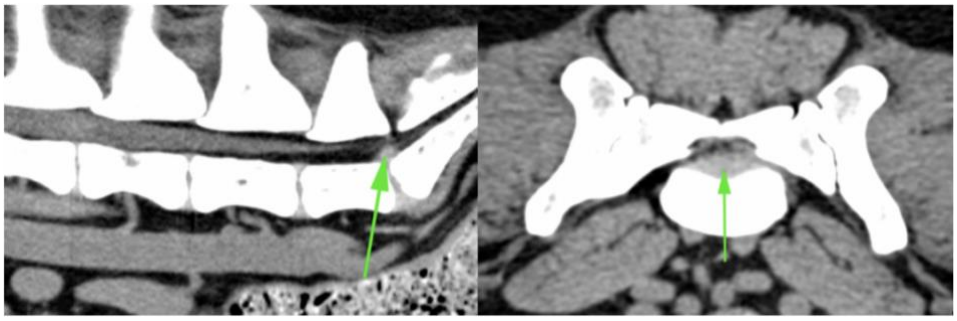


Fig. 8 CT image showing a herniated lumbosacral IVD (green arrow) in lateral view (A) and cranio-caudal view (B); (image kindly provided by José Diogo dos Santos – HVCLC)

Once the diagnosis of DLSS was obtained, the best therapeutic approach was discussed for each dog and the animals which were suited for conservative treatment by epidural injection of corticosteroids were considered for this study.

2.3. Exclusion Criteria

Animals with severe neurologic deficits or with urinary and/or fecal incontinence were excluded from this trial. Animals that were previously taking pain control medication or the ones who needed to introduce any of these drugs were not considered for this study. Dogs whose owners did not answer follow-up questionnaires were also excluded from the study.

2.4. Methods

The patients were admitted at the hospital in the same day in which they were being infiltrated after a six-hour food and water fasting period. Physical exam was performed, and weight of each patient was registered. All patients were classified as ASA I (American Society of Anesthesiologists). Occipital-coccygeal length (OCL) of each patient was calculated (Fig. 9) for dose calculation.

The sedation protocol included a 2-5 μg/kg dose of intramuscular dexmedetomidine. A peripheral venous catheter was placed in one of the anterior limbs and a disposable medical intravenous injection catheter cap was attached. Induction was attained with intravenous propofol that was administered whenever necessary for anesthesia maintenance.

Fig. 9 Measurement of vertebral column's length from occipital condyle to the first coccygeal vertebra (image kindly provided by José Diogo dos Santos – HVCLC).

Every infiltration was performed by the same veterinary surgeon, José Diogo dos Santos, Head of anesthesia department at *HVCLC*.

The patients were placed on the procedure's platform in sternal recumbency with the pelvic limbs pointed cranially (Fig. 10) to hyperflex the SCo junction and thus allow a better access to the epidural space. All patients were oxygenated with a mask and five liters of fraction of inspired oxygen (FiO_2) 100%. Vital parameters, such as heart beats per minute, electrocardiogram, respiratory rate, peripheral capillary oxygen saturation (SpO2), and temperature were monitored throughout the whole procedure.

Fig. 10 Patient positioned in sternal recumbency with pelvic limbs pointed cranially (image kindly provided by José Diogo dos Santos – HVCLC).

The patient's hair was clipped from over the last lumbar spinous process to the tail (Fig. 11). The Sco epidural space was detected after palpating the last sacral vertebra and the first caudal vertebra (Fig. 12).

Fig. 11 Patient in sternal recumbency displaying the clipped area (caudal view); (image kindly provided by José Diogo dos Santos – HVCLC).

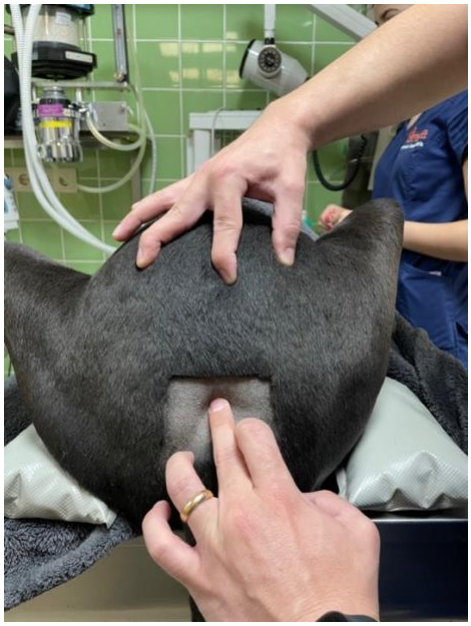


Fig. 12 Identification of the sacrococcygeal intervertebral space location (caudal to the last spinous process of the median sacral crest); (image kindly provided by José Diogo dos Santos – HVCLC).

Proper aseptic technique was performed with a 1% chlorhexidine dihydrochloride and 70% of alcoholic solution. A disposable sheet was used to cover a Mayo table where the

instruments were placed on. The clipped and properly prepared area was covered with a sterile cover field sheet.

The doses of each drug were calculated accordingly to their concentration and patient's weight: 0.1 mg/kg of triamcinolone (Retardoesteroide®, 2mg/mL, Calier, Barcelona, Spain), 0.1 mg/kg of morphine (Morphine 1%, 10 mg/ml, B. Braun Melsungen AG, Germany), 0.05 mL/OCL of lidocaine (Lidocaine 2%, 20 mg/ml, B. Braun Melsungen AG, Germany) and 0.05 ml of iodine (350 mg/mL, GE Healthcare, County Cork, Ireland) for 1 mL of total solution (Fig. 13).

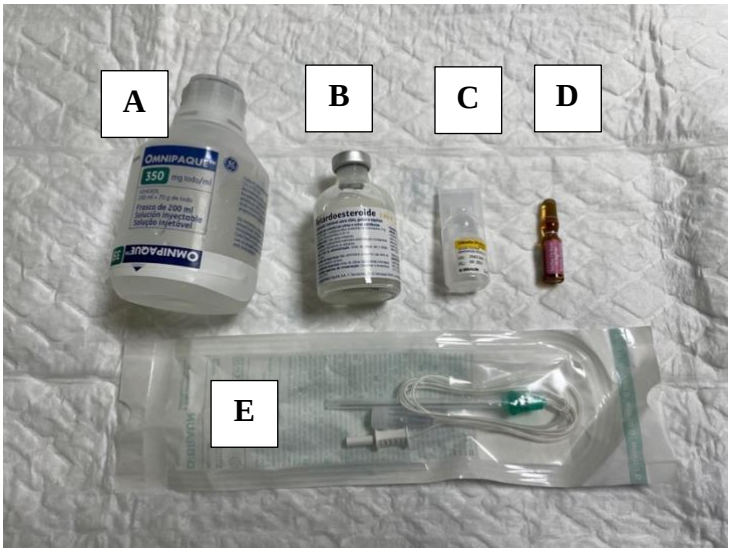


Fig. 12 (A) Omnipaque® (GE Healthcare, County Cork, Ireland), (B) Retardoesteroide ® (Calier, Barcelona, Spain), (C) Morphine 1% (B. Braun Melsungen AG, Germany), (D) Lidocaine 2% (B. Braun Melsungen AG, Germany), and (E) Stimuplex® needle (B. Braun Melsungen AG, Germany); (image kindly provided by José Diogo dos Santos – HVCLC).

Aseptically, and wearing sterile gloves, a 22-gauge needle (Stimuplex® Ultra 360 ® needle 0.7 x 80 mm, B. Braun Melsungen AG, Germany) for peripheral nerve blocks was connected to the negative lead to a nerve stimulator (Stimuplex® HNS 12, B. Braun Melsungen AG, Germany) and the positive lead was connected to a proximal area of the left pelvic limb (Fig. 14) after draining the solution through an extensor.

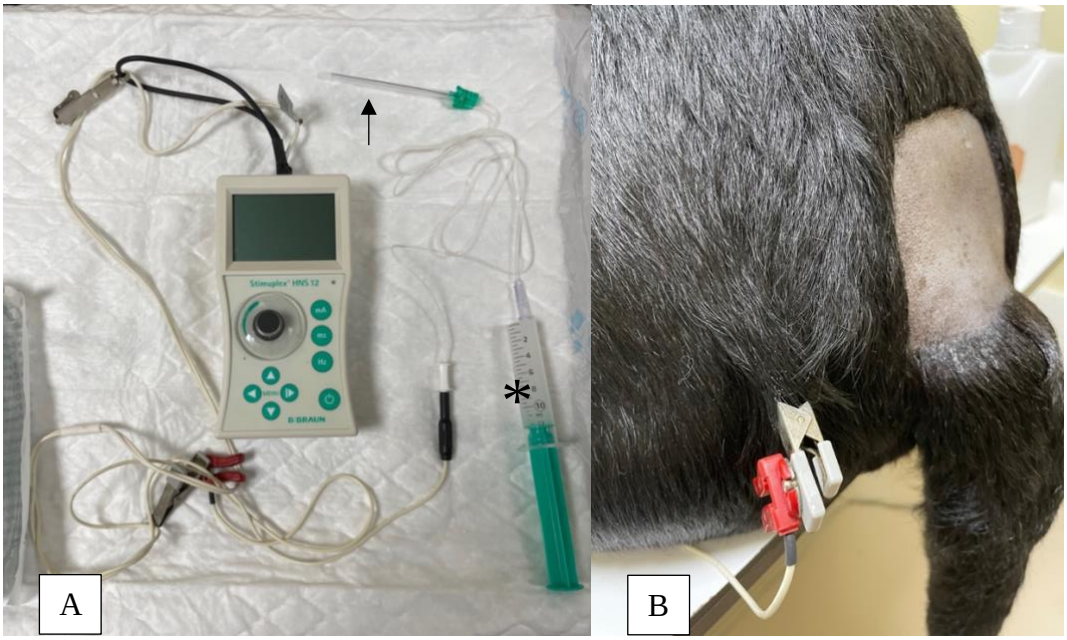


Fig. 13 (A) Stimuplex® HNS 12, B. Braun Melsungen AG, Germany device connected to electric impulse wires and leads, Stimuplex® needle (arrow), syringe with corticosteroid, morphine, lidocaine, and Omnipaque® solution (asterisk); (B) positive lead was connected to a proximal area of the left pelvic limb (image kindly provided by José Diogo dos Santos – HVCLC).

Correct needle positioning was confirmed using a neurostimulator with 0.04 milliamperes (mA) (Fig. 14) and epidurography (Fig. 15). The nerve stimulation test was considered positive when a motor response of the tail's muscles was produced.

After the correct placement of the needle in the epidural space was confirmed, the solution was slowly administered for a one-minute period. After administration, confirmation of correct location of the injectate was obtained by lateral lumbocaudal spinal radiographs, and the contrast was identified in the epidural space, by epidurography (Fig. 16).

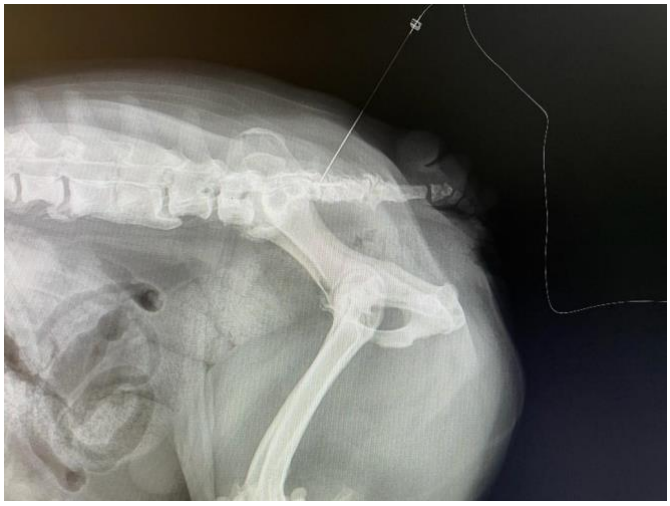


Fig. 14 X-ray confirmation of Stimuplex (B. Braun Melsungen AG, Germany) needle correctly inserted in sacrococcygeal epidural space (image kindly provided by José Diogo dos Santos – HVCLC).

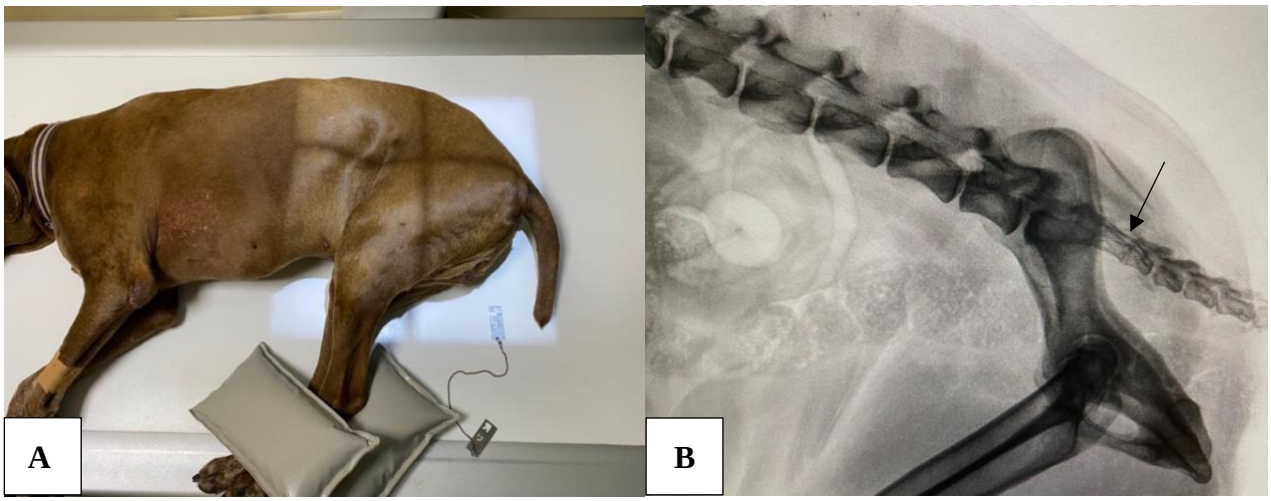


Fig. 15 Confirmation of correct administration in the epidural space by conventional radiographs. (A) Patient in lateral recumbency; (B) Correct location of the injectate in the epidural space (black arrow), identified by the presence of an iodine contrast; (image kindly provided by José Diogo dos Santos – HVCLC).

The patients were kept in observation for an average of four hours for identification of any possible side effects and were then discharged. All injections were successful, and no complications were registered in the 4-hour recovery period.

2.5. Statistical analysis

Statistical analysis for this study was assessed with IBM® SPSS® V.25 program. A descriptive and inferential analysis was performed.

The descriptive analysis was presented graphically by evaluation's domain for each dog through their three moments of evaluation: pre-treatment, one month after treatment, and three months after treatment. [96]

Inferentially, to compare the three moments of evaluation in the several assessed domains and considering the ordinal qualitative nature of the applied scores (HCPI and systemic effects of corticosteroids absorption query) and the small sample (n=6), a non-parametric test (Friedman's test) was used, with multiple Dunn-Bonferroni comparisons. [96]

A p-value < 0.05 was considered statistically significant.

3. Results

After all exclusion criteria was applied, a total of six (n=6) dogs were admitted to this retrospective study. Table 1 summarizes the results of sex, age, breed, and bodyweight observed for the clinical patients in this study.

Table 1. Results of sex, age, breed, and bodyweight for the studied population.

	C1	**C2**	**C3**	**C4**	**C5**	**C6**
Sex	Female	Female	Male	Female	Male	Male
Age	7 y.o.	10 y.o.	10 y.o.	6 y.o.	14 y.o.	12 y.o.
Breed	Labrador Retriever	Grand Danois	Labrador Retriever	French Bulldog	German Shorthaired Pointer	Labrador Retriever
BW	31.5 kg	44.0 kg	35.0 kg	11.7 kg	31.7 kg	44.0 kg

C – canine patients; *BW* – bodyweight; *y.o.* – years old.

The patients' clinical signs were assessed before the epidural injection, and then one month and three months after the procedure by application of the HCPI. Clinical signs at both follow-ups were compared to pre-treatment status.

Since the HCPI represents a numeric qualitative scale and the studied population is small ($n<30$), parametric statistics were used to organize the data. Friedman's test was used to analyze the three paired samples (evaluation assessments pre-treatment, one month after treatment, and three months after treatment). Data was organized in Table 2 and Table 3. In Table 2, a p-value < 0.05, indicates that a difference was observed between the three moments of evaluation. The mean rank is used to rate each domain, in which the lowest value represents a better score.

In Table 3, arrows were used when the status had changed at any moment in both follow-ups. Upper arrows represent an improvement of clinical signs, where downer arrows represent a worsening of clinical sigs. When no alteration was observed, the letter "M" was used. It can be noticed that patient 5 improved its condition on several assessed parameters, while patient 6 improved its condition in most of them. However, relative to patients 1 – 4, they showed improvements one month after treatment but relapsed at the 3-month follow-up. Also, regarding a few clinical signs, patients 1 and 3 showed

decreased function at the 1-month follow-up, while returning to the initial status at the 3-month follow-up.

Table 2. Statistical comparison between pre-treatment and 1 and 3 months after treatment.

Evaluation domain	Treatment phases Mean Rank			$Fr_{(2)}$	*p*
	Before	1month after	3months after		
1. Dogs' attitude and/or mood	1.92	2.17	1.92	2.000	*0.368*
2. Dogs' willingness to participate in play or interact	2.00	2.00	2.00	0.000	1.000
3. Dogs' frequency in vocalization or discomfort behavior (audible whining, grunting, yelping, or unusual licking)	2.25	1.75	2.00	2.000	*0.368*
4. Dogs' eagerness to walk	2.00	2.00	2.00	0.000	*1.000*
5. Dogs' ability and/or willingness to walk up and/or downstairs	2.25	1.75	2.00	1.500	*0.472*
6. Dogs' ability and/or willingness to run	2.17	2.17	1.67	2.000	*0.368*
7. Dogs' ability and/or willingness to jump (onto bed, couch, vehicle, etc.)	2.50	1.50	2.00	6.000	*0.050*
8. Dogs' easiness in lying down	2.25	1.92	1.83	1.400	*0.497*
9. Dogs' rising from a down position	2.42	1.58	2.00	5.000	*0.082*
10. Dogs' ease of movement after a long rest	2.42	1.42	2.17	6.500	*0.039*
11. Dogs' ease of movement during and/or after exercise/walks (tired, dragging feet, scuffing nails, lying down)	2.50	1.42	2.08	6.615	*0.037*

Fr – relative frequency; *p* – p-value

Table 3. Summary comparison between pre-treatment and both 1-month and 3-months follow-up.

Behavior	Dogs					
	C1	C2	C3	C4	C5	C6
Q1. attitude and/or mood	M	M	1m ↘ 3m ↗	M	M	M
Q2. willingness to participate in play or interact	M	M	1m ↘ 3m ↗	M	M	1m ↗ 3m ↘
Q3. frequency in vocalization or discomfort behavior (audible whining, grunting, yelping, or unusual licking)	1m ↗ 3m ↘	1m ↗ 3m ↘	M	M	M	1m = 3m ↗
Q4. eagerness to walk	M	M	M	M	M	M
Q5. ability and/or willingness to walk up and/or downstairs	1m ↗ 3m ↘	M	1m ↘ 3m ↗	1m ↗ 3m ↘	M	1m ↗↗ 3m =
Q6. ability and/or willingness to run	1m ↘ 3m ↗	M	1m ↘ 3m ↗	M	1m ↗↗ 3m =	1m ↗ 3m =
Q7. ability and/or willingness to jump (onto bed, couch, vehicle, etc.)	M	M	1m ↗ 3m ↘	1m ↗ 3m ↘	1m ↗↗ 3m =	1m ↗↗ 3m =
Q8. easiness in lying down	1m ↘ 3m ↗	M	M	M	1m ↗ 3m =	1m ↗↗ 3m ↘
Q9. rising from a down position	M	1m ↗ 3m ↘	M	M	1m ↗ 3m =	1m ↗↗ 3m ↘
Q10. ease of movement after a long rest	1m ↗ 3m ↘	1m ↗ 3m ↘	M	1m ↗ 3m ↘	M	1m ↗ 3m =
Q11. ease of movement during and/or after exercise/walks (tired, dragging feet, scuffing nails, lying down)	M	1m ↗ 3m ↘	1m ↗↗ 3m ↘↘	M	1m ↗ 3m =	1m ↗↗ 3m ↘

ID – identification; *C* – canine patients; *Q* – question; – month(s); *M* – maintains at both follow-ups; *upper arrows* – improvement of clinical signs; *down arrows* – worsening of clinical signs; = - maintains at one follow-up.

3.1. Pre-treatment and follow-ups

It appeared that the treatment had none or little impact on the dogs' *attitude and/or mood*, *willingness to participate in plays or interactions*, *frequency of vocalization or discomfort behavior*, *eagerness to walk*, *ability to walk up and/or downstairs*, *ability and/or*

willingness to run, *easiness in lying down*, and *easiness on rising from a down position* (results individually displayed in graphics 1, 2, 3, 4, 5, 6, 8, and 9 – Appendix III).

Results showed significant improvement one month after treatment on the dogs' *ability and/or willingness to jump (onto bed, couch, vehicle, etc.)*, *easiness of movement after a long rest*, and *easiness of movement during and/or after exercise/walk* (results individually displayed in graphics 7, 10, and 11– Appendix II). However, when an improvement was registered at the one-month follow-up, three months after the epidural injection the clinical signs usually relapsed to the initial status.

3.2. Ruling out of systemic effect of corticosteroid

In the attempt of trying to exclude the hypothesis of systemic absorption of the triamcinolone injected in the epidural space, owners were asked to answer a group of questions concerning some possible side effects of systemic use of corticosteroids (Table 4. and Table 5.).

Five owners reported normal parameters with no changes in the pre-treatment period and in both follow-ups, thus the triamcinolone effect was assumed to act successfully *in locum*. In one animal (C1) an increased water intake was reported both at the one-month and 3-month follow-up (Graphic 12. – Appendix III), as well as an increase in urine production and/or frequency (Graphic 14. – Appendix III).

Table 4. Statistical comparison between pre-treatment and 1- and 3-months follow-ups.

Evaluation domain	Treatment phases Mean Rank			$Fr_{(2)}$	*p*
	Before	1m after	3m after		
Q1. Water consumption	1.83	2.08	2.08	2.000	*0.368*

Q2. Appetite/food consumption	2.00	2.00	2.00	0.000	1.000
Q3. Urine production (quantity and/or frequency)	1.83	2.08	2.08	2.000	*0.368*
Q4. Feces macroscopic aspect	2.00	2.00	2.00	0.000	*1.000*

Fr – relative frequency; *p* – p-value

Table 5. Comparative synthesis between physiological behavior between pre-treatment and 1- and 3-months follow-ups.

Behavior	Dogs ID					
	C1	C2	C3	C4	C5	C6
Q1. Water consumption	1m ↗ 3m =	M	M	M	M	M
Q2. Appetite/food consumption	M	M	M	M	M	M
Q3. Urine production (quantity and/or frequency)	1m ↗ 3m =	M	M	M	M	M
Q4. Feces macroscopic aspect	M	M	M	M	M	M

ID – identification; *C* – canine patients; *Q* – question; *m* – month(s); *M* – maintains at both follow-ups; *upper arrows* – improvement of clinical signs; = - maintains at one follow-up.

3.3. Owner feedback

At the last follow-up (three months after treatment), owners were asked whether they considered the treatment had contributed to a better QOL of their dogs, in which the answer was positive in all six cases.

4. Discussion

While the lumbosacral access has been reported in only a few studies [21,34,48] , the SCo approach doesn't seem to be described in veterinary medicine for this purpose. Although not exceptional, these results were particularly relevant since some improvements of clinical signs were noticed after the SCo epidural injection, specially one month after treatment.

Although, to the best knowledge of the author, the administration of a mixture of triamcinolone, morphine, lidocaine, and a radiopaque contrast is not reported in the literature, it was decided to associate these drugs. The injection of a corticosteroid aimed to actively reduce the local inflammation associated to DLSS, while the association of morphine aimed to contribute to the modulation of pain and to extend the analgesic effect. The use of lidocaine allowed the reduction of other analgesics as it acts as a local anesthetic. It was also added an iodine contrast to confirm the correct application of the solution in the epidural space, through epidurography. This study showed that the SCo epidural injection was successful in the cranial spreading through the epidural space producing an effective action on the inflamed area at the LS junction. Although the population lacks representability, during this study's procedures, no medulla puncture was detected and that would be a strength of the SCo injection when compared to the LS approach, in which is more likely to occur. [12,97] A literature review of human ESIs pointed out that accidental dural puncture still represents 0.33% to 1% (lumbar) of complications. [98] The author didn't find any similar review in veterinary literature, but the percentage is expected to be higher than what is described in human medicine and that's why the SCo approach would be a better option for reduction of dural punctions.

The technique showed most effectiveness in the improvement of three parameters: *dogs' ability and/or willingness to jump (onto bed, couch, vehicle, etc.)*, *dogs' ease of movement after a long rest*, and *dogs' ease of movement during and/or after exercise/walks (tired, dragging feet, scuffing nails, lying down)* since p-value was beneath 0.05 in all these categories at the first follow-up. However, in all other eight categories, no significant improvements were noted. Still, in a 0 to 5 index (HCPI), where 0 represents a better

clinical status and 5 a more severely affected patient, an overall review of the results (displayed in Appendix II) allows us to perceive that one month after the treatment, in any category there are no dogs scoring a condition worse than 3. In other words, even though after the treatment not all dogs had significantly improved their clinical signs (especially those which already had better initial statuses), most of those in initial worse conditions were able to achieve a lower score (better clinical status) at the one-month follow-up.

As previously reported in veterinary medicine and human medicine, this conservative treatment approach lacks efficacy in the long-term. [99] Janssens *et al* and Gomes *et al* studies showed a relapse of clinical signs with a median of 11 days and two months, respectively, after the first epidural injection of methylprednisolone. [21,34] These results are somehow compatible to what was observed in the present study in means that most of the improvements in the clinical signs of the dogs infiltrated didn't last in the long term, even though the used corticosteroid and administration route were different. Contrariwise, a 2016 study in humans reported satisfactory results with epidural methylprednisolone injection (caudal route in 74.3%, transforaminal in 90% and interlaminar in 77.7% after both six months and one year after treatment). [100]

Although HCPI was developed to assess chronic pain in dogs with OA and not specifically for DLSS, this seemed like the most reliable evaluation index to apply in cases of DLSS, since to the best knowledge of the author, no specific pain scale has been developed for the assessment of DLSS.

Another aspect that should have been approached was the quantification and classification of the severity of each lesion in CT-scans analysis as well as in clinical signs.

Although no systemic analgesics were associated to the treatment, since it could affect the results, it is the author's belief that in current practice the concurrent oral administration of gabapentinoids, or other pain modulators such as NSAIDs could greatly improve the efficacy of this treatment.

If more studies with larger populations are made in this regard, potentially it would be possible to prove that SCo injections represent a safer practice in epidural injections with the same degree of efficacy, as shown by the present results. Also, it would be interesting to test a different corticosteroid or whether the administration of higher doses of corticosteroids would result in a higher efficacy of this treatment. Furthermore, it could be useful to investigate whether a repetition of the treatment could result in a better outcome, instead of a single injection.

In the author's opinion, a conservative treatment could be a valid approach in dogs with DLSS, given the results of this and other previous studies. [21,34] Although some surgical treatments are reported to have a higher level of success, [70,71] and epidural injections of corticosteroids have limited time effect and appear to not last longer than three months (on a single injection treatment approach), they may be a therapeutic option when surgery is not. This may apply to patients in which general anesthesia, or a more invasive procedure, represent a higher risk to their health. In this regard, the author believes that a SCo epidural injection for the treatment of DLSS is a safe and well-accepted therapeutic approach. It is important to do more research in order to establish when to prescribe this treatment instead of others, which has been a challenge in human medicine, as well.

All six owners considered that the treatment helped to improve the QOL of their dogs, which is already a positive aspect for this treatment and encourages further investigation on this conservative treatment approach.

5. Conclusion

The present study intended to show the feasibility of ESI for pain management in dogs with DLSS and improvement of QOL.

It was possible to withdraw some conclusions from this study. The used technique – epidural SCo injection – though requiring expertise, is considered safe and a valid alternative to LS injections. The administration of a corticosteroid in the epidural SCo

space was possible to perform in dogs with DLSS which may be useful in some cases with better results, lower risk of dural punction, and without apparent systemic absorption of corticosteroids. This conservative approach represents an acceptable alternative to surgery, essentially in animals where surgery represents a greater risk. An important result was the positive response from the owners on the improvement of their pets' QOL.

Due to the short period in which the patients presented to the hospital and to insufficient adherence from the patients' tutors, the case load was severely reduced from the original number of dogs diagnosed with DLSS. Therefore, it was not possible to achieve the intended statistical relevance for this study due to the small sample obtained. The inclusion of a control group would also be an aspect to improve in future studies with a larger population.

Also, queries based on the owner's observation may be subjective and may have associated biases such as interpretation variability. Therefore, ideally, all follow-ups should have been made by the same clinician to uniformize the assessment of the population's clinical signs' evolution or, ideally, using FPGA and KGA.

Although the obtained results were not exceptional, they do not close the door for this approach in the treatment of dogs with DLSS. Believing that the efficacy of this treatment is not yet to be ruled out, more studies with less weaknesses are necessary to confirm whether this could be a viable option to treat DLSS and improve the QOL of these patients.

Appendix I – Helsinki Chronic Pain Index's adaptation to Portuguese.

Nome do animal: Data:

Índice de Dor Crónica de Helsínquia

Hielm-Bjorkman HK, Rita H, Tulamo R-M. Psychometric testing of the Helsinki chronic pain index by completion of a questionnaire in Finnish by owners of dogs with chronic signs of pain caused by osteoarthritis. Am J Vet Res. 70: 727-734, 2009.

(Traduzido do Finlandês para o Inglês, e do Inglês para o Português)

Selecione a descrição de dor e funcionalidade que melhor representa o comportamento do seu cão:

Avalie a atitude e/ou disposição do seu cão:

0	1	2	3	4
Muito alerta	Alerta	Nem alerta nem desinteressado	Desinteressado	Muito desinteressado / letárgico

Avalie a disposição do seu cão para interagir ou participar numa brincadeira:

0	1	2	3	4
Muito disposto	Disposto	Relutante	Muito relutante	Não participa ou não interage de todo

Avalie a frequência com que o seu cão vocaliza ou demonstra comportamento de desconforto (choro audível, gemidos, lambedura fora do normal):

0	1	2	3	4
Nunca	Quase nunca	Às vezes	Frequentemente	Muito frequentemente

Avalie a avidez/vontade do seu cão para caminhar:

0	1	2	3	4
Muita vontade para caminhar	Capaz de caminhar	Relutante	Muito relutante	Não tem qualquer vontade para caminhar

Avalie a capacidade e/ou disposição do seu cão para subir e/ou descer degraus:

0	1	2	3	4
Muito disposto / capaz	Disposto / capaz	Relutante	Muito relutante	Não sobe nem desce degraus de todo

Índice de Dor Crónica de Helsínquia

Avalie a capacidade do seu cão e/ou disposição para correr:

0	1	2	3	4
Muito disposto / capaz	Disposto / capaz	Relutante	Muito relutante	Não corre de todo

Avalie a capacidade e/ou disposição do seu cão para saltar (para a cama, sofá, veículo, etc.):

0	1	2	3	4
Muito disposto / capaz	Disposto / capaz	Relutante	Muito relutante	Não salta de todo

Avalie a facilidade com que o seu cão se deita:

0	1	2	3	4
Com muita facilidade	Com facilidade	Nem com facilidade nem com dificuldade	Com dificuldade	Com muita dificuldade

Avalie a facilidade com que o seu cão se levanta:

0	1	2	3	4
Com muita facilidade	Com facilidade	Nem com facilidade nem com dificuldade	Com dificuldade	Com muita dificuldade

Avalie a facilidade com que o seu cão se movimenta após um longo período de descanso:

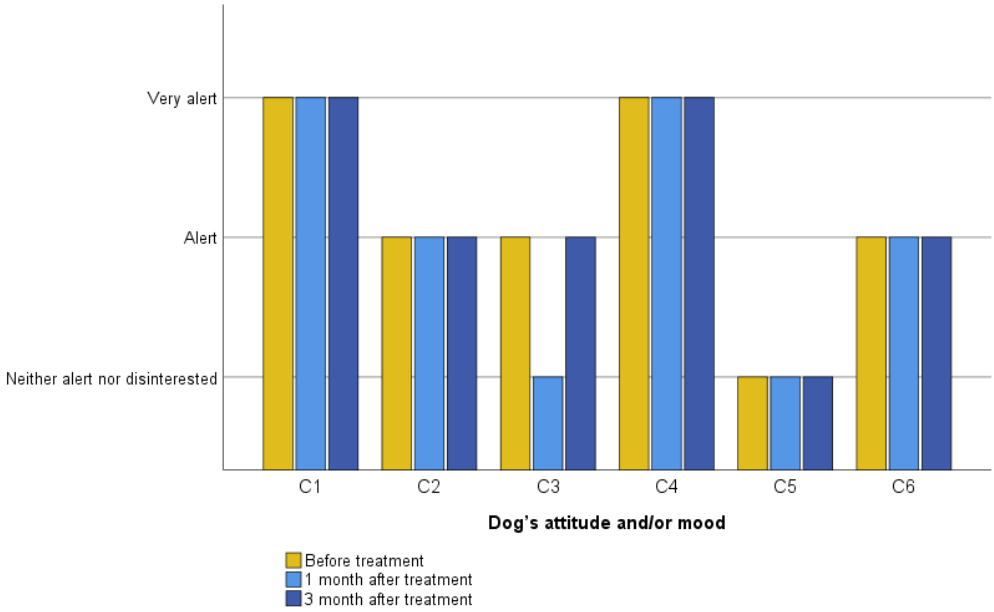
0	1	2	3	4
Com muita facilidade	Com facilidade	Nem com facilidade nem com dificuldade	Com dificuldade	Com muita dificuldade

Avalie a facilidade com que o seu cão se movimenta durante e/ou após exercício/passeios (cansaço, arrastar dos membros, raspar as unhas no chão, deitar-se no chão):

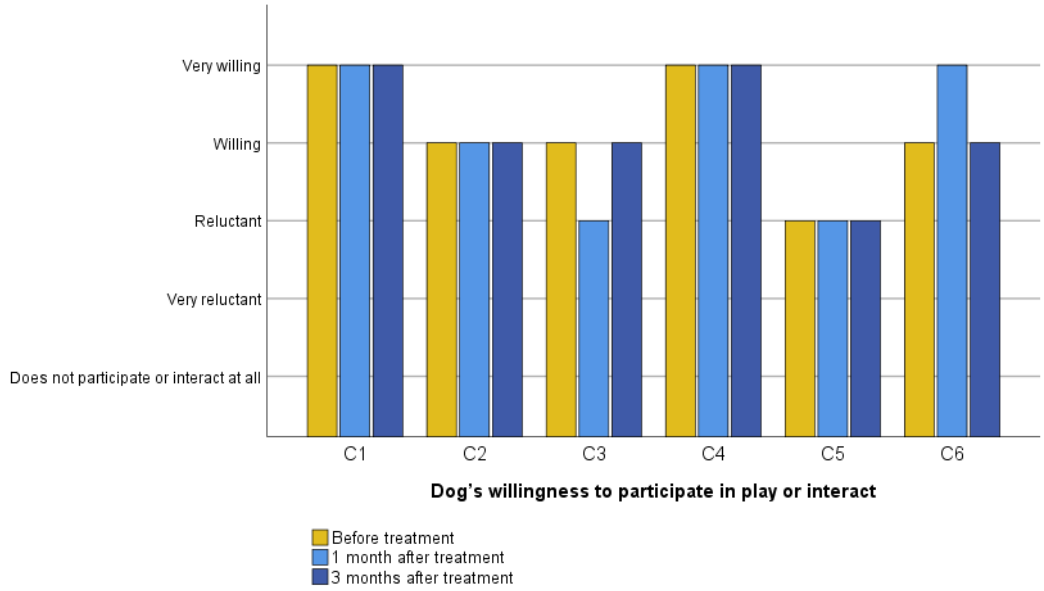
0	1	2	3	4
Com muita facilidade	Com facilidade	Nem com facilidade nem com dificuldade	Com dificuldade	Com muita dificuldade

Appendix II – Pre-treatment and follow-ups' results.

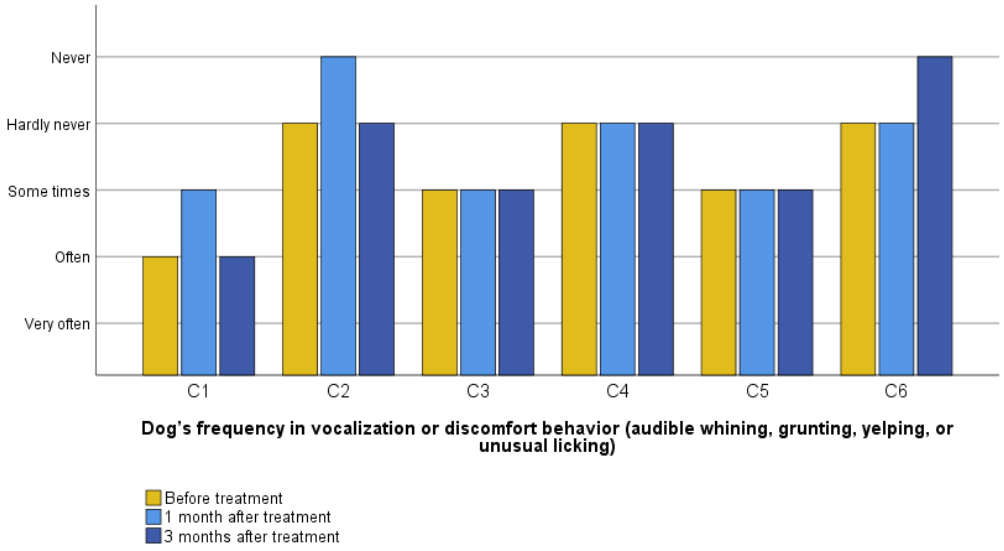
Graphic 1. Dogs' attitude and/or mood between pre-treatment and the 1- and 3-month follow-ups.



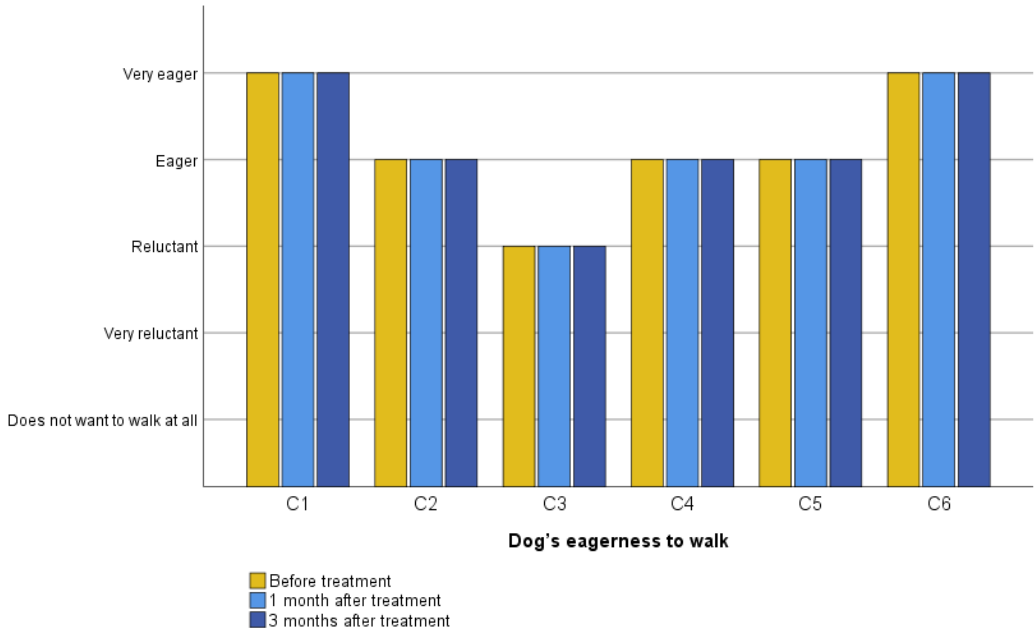
Graphic 2. Dogs' willingness to participate in play or interact between pre-treatment and the 1- and 3-month follow-ups.



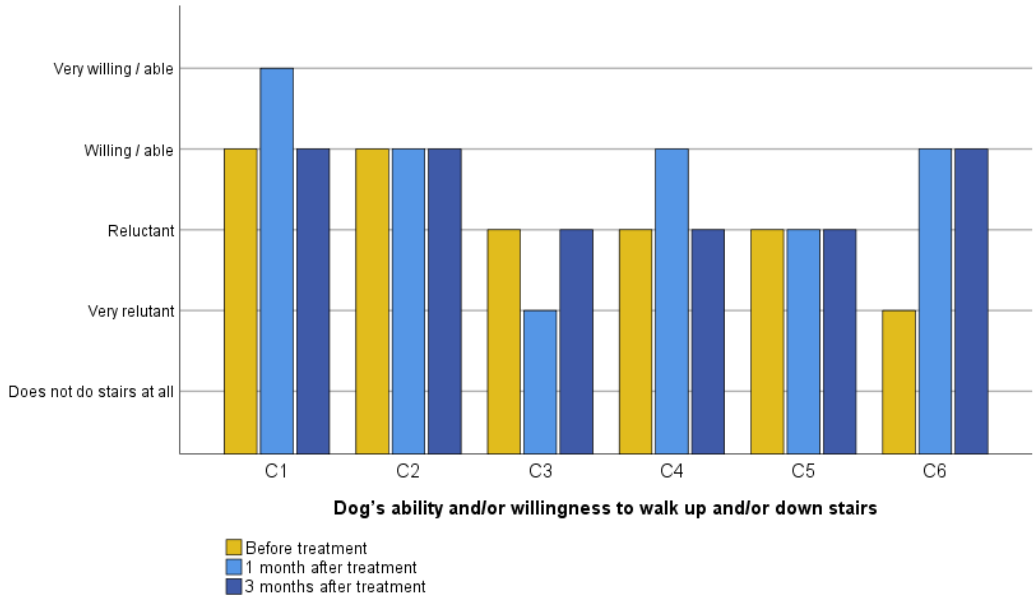
Graphic 3. Dogs' frequency in vocalization or discomfort behavior (audible whining, grunting, yelping, or unusual licking) between pre-treatment and the 1- and 3-month follow-ups.



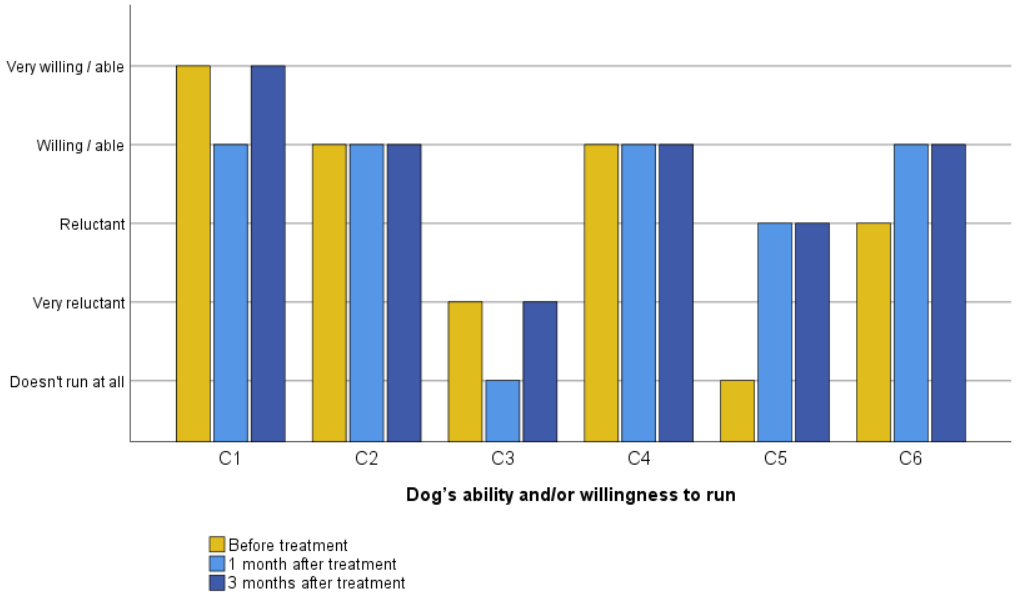
Graphic 4. Dogs' eagerness to walk between pre-treatment and the 1- and 3-month follow-ups.



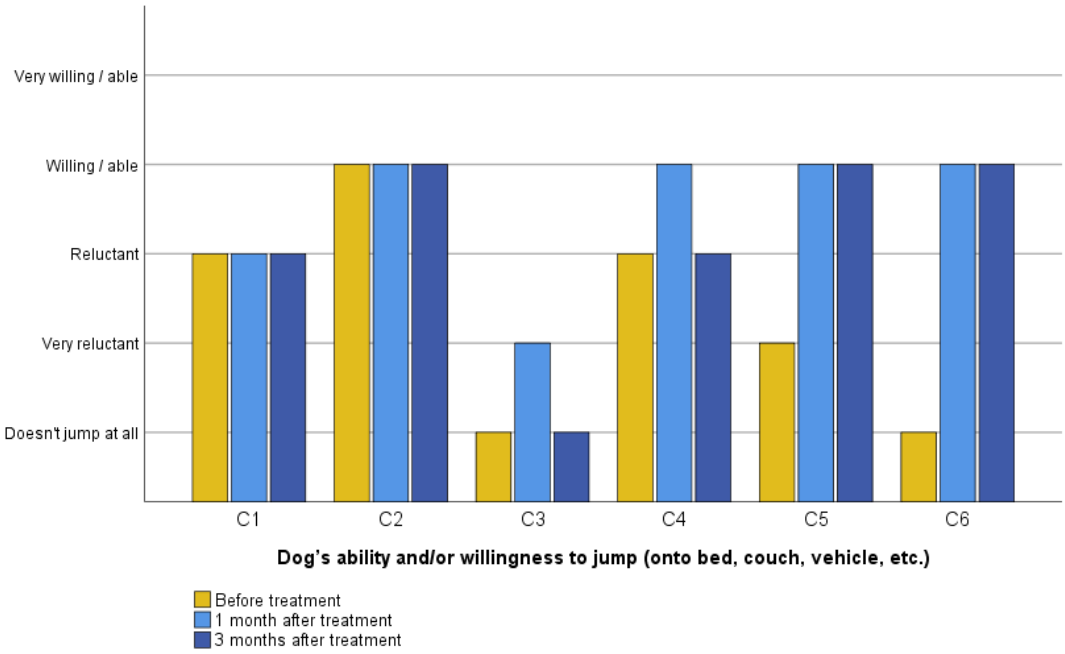
Graphic 5. Dogs' ability and/or willingness to walk up and/or downstairs between pre-treatment and the 1- and 3-month follow-ups.



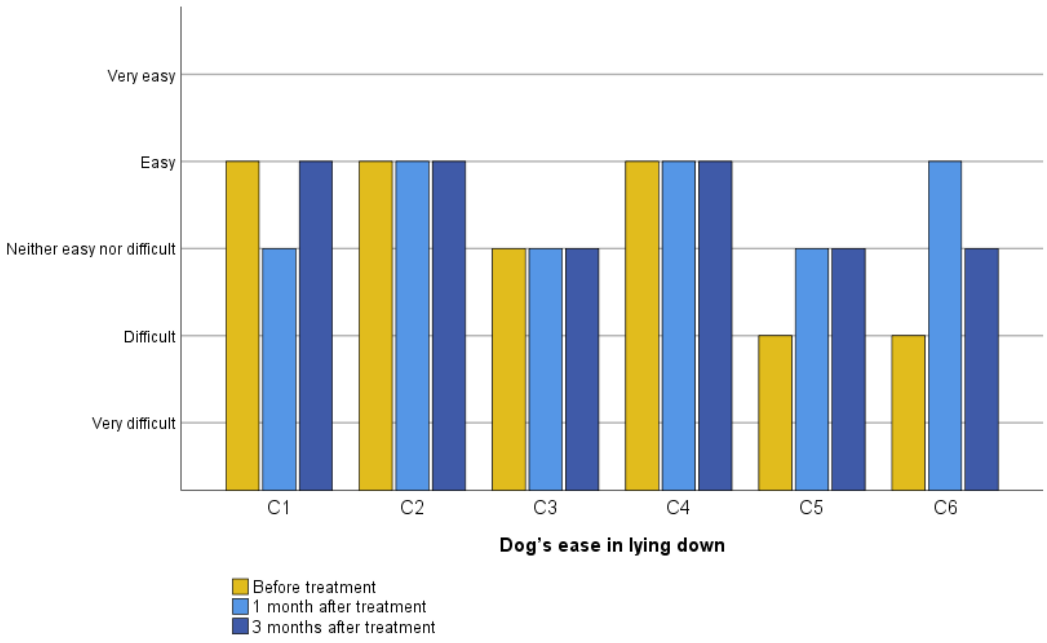
Graphic 6. Dogs' ability and/or willingness to run between pre-treatment and the 1- and 3-month follow-ups.



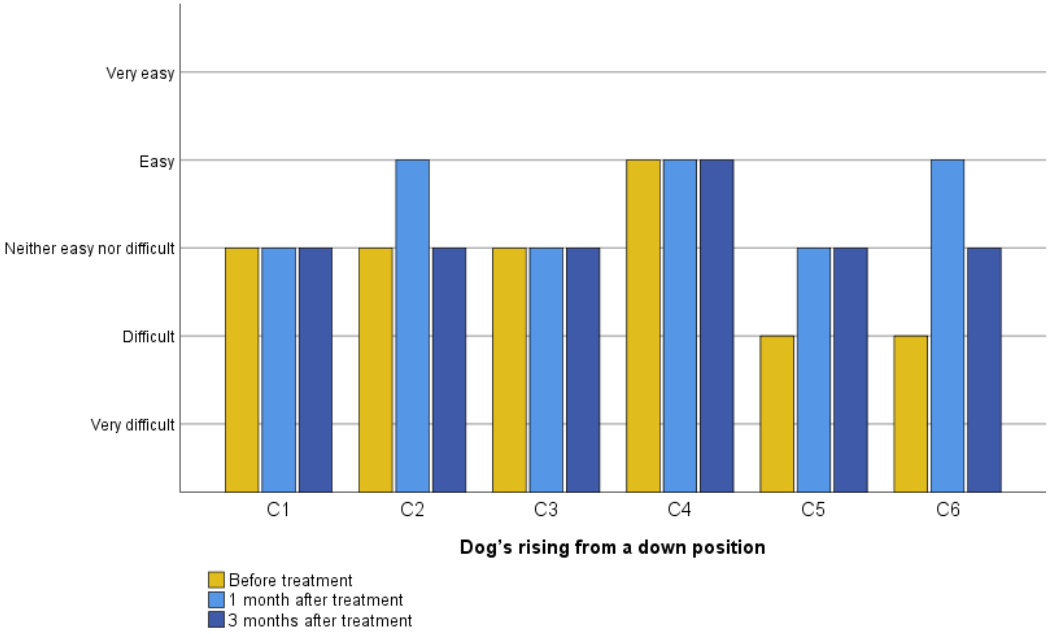
Graphic 7 – Dogs' ability and/or willingness to jump (onto bed, couch, vehicle, etc.) between pre-treatment and the 1- and 3-month follow-ups.



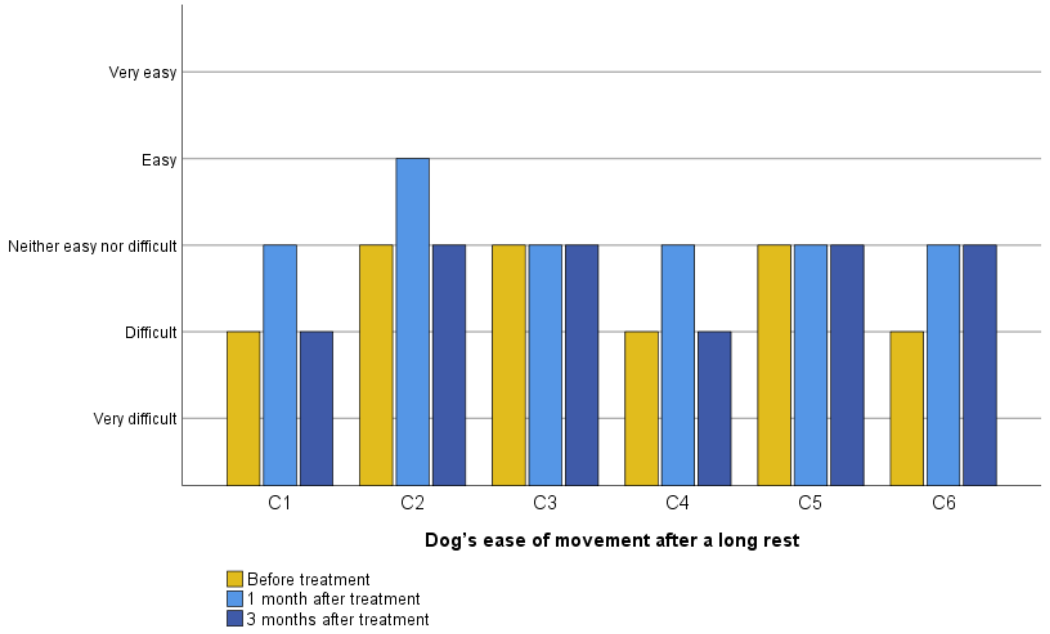
Graphic 8. Dogs' easiness in lying down between pre-treatment and the 1- and 3-month follow-ups.



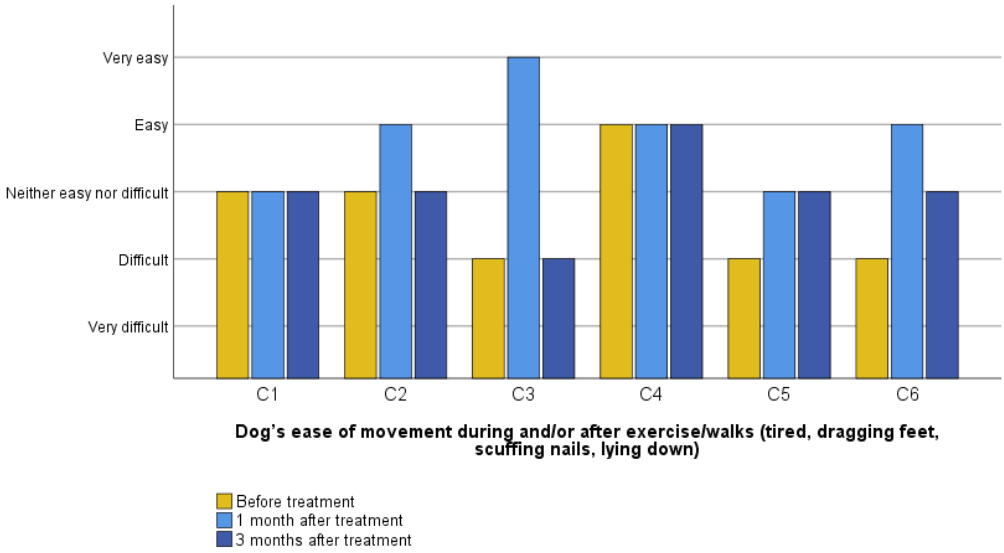
Graphic 9. Dogs' rising from a down position between pre-treatment and the 1- and 3-month follow-ups.



Graphic 10. Dogs' ease of movement after a long rest between pre-treatment and the 1- and 3-month after treatment

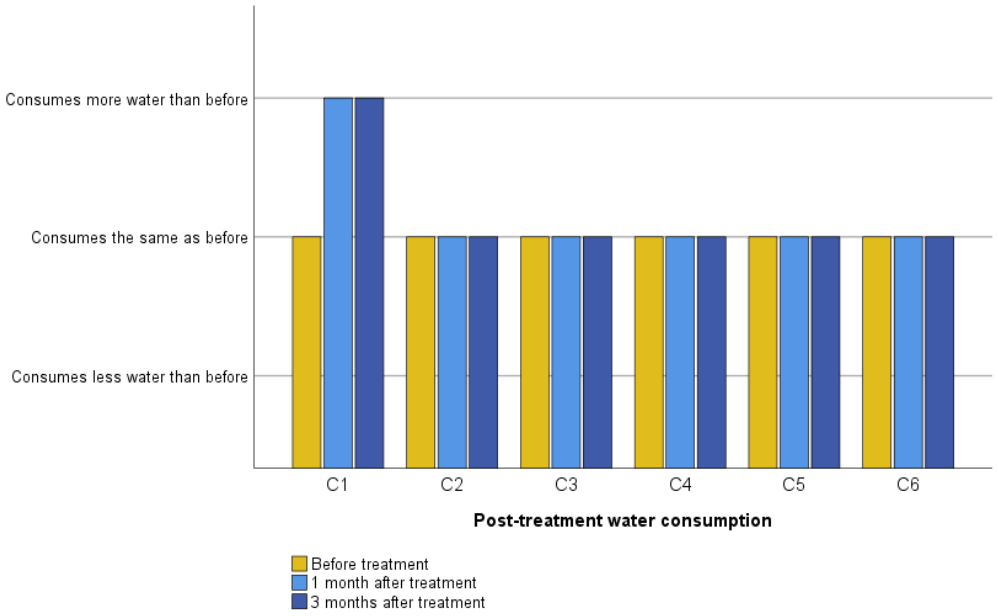


Graphic 11. Dogs' ease of movement during and/or after exercise/walks (tired, dragging feet, scuffing nails, lying down) between pre-treatment and the 1- and 3-month follow-ups.

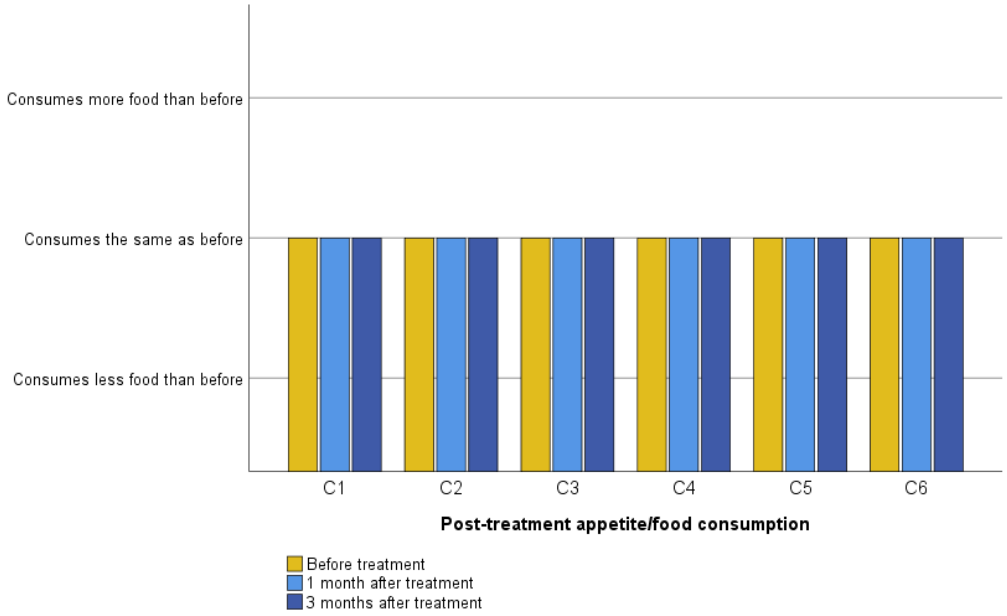


Appendix III – Corticosteroid systemic effect query

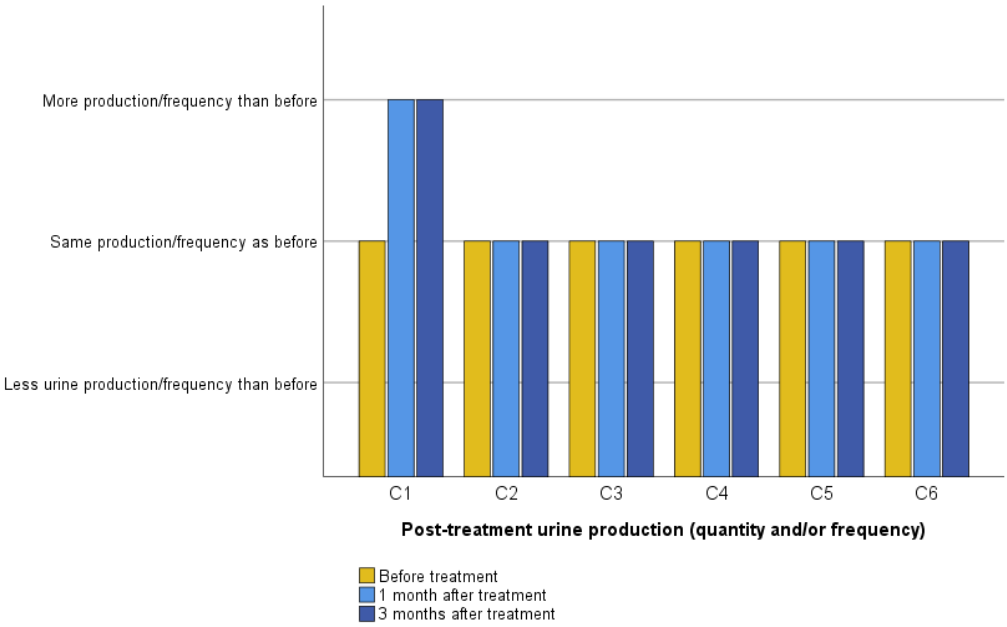
Graphic 12. Water consumption between pre-treatment and the 1- and 3-month follow-ups.



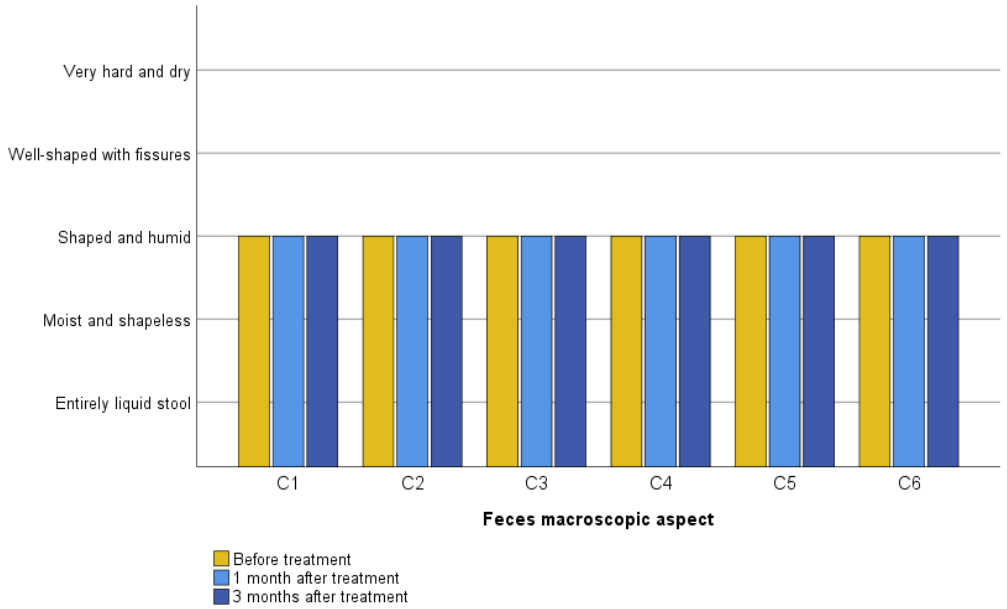
Graphic 13. Appetite/food consumption between pre-treatment and the 1- and 3-month follow-ups.



Graphic 14. Urine production between pre-treatment and the 1- and 3-month follow-ups.



Graphic 15. Feces macroscopic aspect between pre-treatment and the 1- and 3-month follow-ups.



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