# “How Stella Got Her Groove Back”

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## Introduction

Intervertebral Disc Disease (IVDD) is a neurologic syndrome that is characterized by degenerative changes in the intervertebral disc. IVDD is a common occurrence in dogs, thought to occur in around 2% of patients, and occurs less frequently in cats, horses, and food animals.[6] IVDD can occur in 2 major ways – Hansen Type 1 and Hansen Type 2. Hansen Type 1 is an acute form that is characterized by extrusion of the nucleus pulposus into the spinal canal. Hansen Type 2 is a chronic form that is characterized by protrusion of the anulus fibrosus and nucleus pulposus into the spinal canal.[8] The following case will focus on Hansen Type 1 IVDE in a chocolate Labrador and her recovery after treatment.

## History & Presentation

Stella is a 7-year-old, female spayed, Labrador who presented to MSU-CVM for being acutely down in her pelvic limbs. Her owners heard a yelp around 4 am and woke to find Stella in severe pain and unable to ambulate.

Upon presentation, Stella was anxious and alert. She weighed 41.6 kgs with a BCS of 8/9 (overconditioned). She had a temperature of 103.3 F, a pulse of 200 bpm, and a respiration of panting. Her mucous membranes were pink and moist with a capillary refill time of <2 seconds. Her heart and lungs auscultated normally – no crackles, wheezes, murmurs, or arrhythmias heard. Her blood pressure was 198/129 (132), 211/58 (175).

A full neurological exam revealed Stella appeared normal in mentation and all of her cranial nerves were intact. She had normal muscle tone in all limbs, but absent motor in her pelvic limbs. Her pelvic limbs had absent proprioception; her thoracic limbs were normal. Her segmental reflexes were intact in all limbs. Upon palpation of her spine, hyperpathia was noted

along L2-L3. Due to absence of motor in the pelvic limbs, nociception was tested and was present. Based on the loss of motor function to the pelvic limbs and present nociception, Stella was diagnosed as paraplegic (deep pain positive).

To guide diagnostic approach and differentials, neurologic lesions are localized to 3 main areas – brain (forebrain, brainstem), spinal cord, and neuromuscular system. Due to Stella's normal mentation and intact cranial nerves, forebrain and brainstem lesions were ruled out and a neuromuscular disorder was deemed less likely because of the intact reflexes. Therefore the lesion was in the spinal cord. There are 4 spinal cord segments, cervical (C1-C5), cervical intumescence (C6-T2), thoracolumbar (T3-L3), and pelvic intumescence (L4-S2) lesions. Each segment, when dysfunctional, will result in unique neurologic signs. Stella's normal thoracic limbs ruled out cervical and cervical intumescence lesions. A pelvic intumescence lesion was ruled out because of Stella's intact segmental reflexes in her pelvic limbs and UMN bladder. Therefore, based on normal mentation, normal thoracic limbs, and UMN pelvic limbs, Stella's lesion was localized to the thoracolumbar spine (T3-L3).

**Diagnostic Approach**

Prior to advanced imaging, a blood sample was collected for a complete blood count (CBC) and serum chemistry panel. The CBC showed a mild stress leukogram; mature neutrophilia (87%) and lymphopenia (869 /ul). The chemistry panel showed a moderate hypercapnia (17.7 mEq/L) due to her panting, a mildly increased ALP (158 U/L) likely due to stress, and a mild hyperphosphatemia (5.8 mg/dl) and severely increased CK (5791 U/L) both due to muscle damage sustained during the IVDE inciting event. Radiographs were performed to assess for underlying pulmonary issues and neoplastic metastasis. An age-related bronchial pulmonary pattern was seen

with no evidence of metastatic neoplasia. Once any underlying pathologies were ruled out, Stella was sent for further imaging at VSC.

The top differentials for an acute, focal myelopathy in the thoracolumbar spine include IVDD, fibrocartilaginous embolism (FCE), and acute non-compressive nucleus pulposus extrusion (ANNPE). The diagnostics of choice are an MRI or a CT; with MRI being the gold standard test. Due to the spinal hyperpathia, IVDD was the top different for Stella and an MRI was performed. The MRI showed a disc herniation with mineralization at L2-L3 and an anomalous 8$^{th}$ lumbar vertebra. Based on the history, clinical signs, and MRI results, Stella was diagnosed with Hansen Type 1 IVDE at L2-L3.

**Pathophysiology**

The spine consists of vertebrae separated by intervertebral discs that lie just ventral to the vertebral canal which houses the spinal cord. The discs act as shock absorbers and stabilizers for the spine. Each disc has an outer annulus fibrosus surrounding an inner nucleus pulposus. The annulus fibrosus consists of distinct, parallel lamellae that arise from the cartilaginous endplates and adjacent vertebrae. The lamellae are mostly composed of type 1 collagen and is innervated with nociceptive fibers which play a role in the discogenic pain and paraspinal hyperesthesia seen with disc degeneration.[1] The nucleus pulposus is an embryologic remnant of the notochord made primarily of water and proteoglycans. The most important proteoglycan is glycosaminoglycan (GAG). It is made of chondroitin sulfate, dermatan sulfate, keratan sulfate, and hyaluronic acid.[6] The nucleus has an abundance of notochordal cells which produce and assemble glycosaminoglycans. In type 1 IVDD, the glycosaminoglycans begin to lose their water content, shorten, and increase in the ratio of keratan sulfate to chondroitin sulfate leading to a weakened

anulus and a mineralized nucleus. It is termed type 1 chondroid metaplasia and is primarily associated with early intervertebral disc degeneration (IVDD).[6]

Hansen Type 1 is defined as complete rupture of the annulus fibrosus and extrusion of the nucleus pulposus into the vertebral canal. It most commonly occurs in chondrodystrophic breeds (Dachshunds, Beagles, Basset Hounds, Corgis, etc.) between the ages of 3 and 5 years old.[8] The weakened anulus and mineralized nucleus results in a fragile disc that ruptures easily due to an inciting event that creates excess pressure such as jumping off the couch or even a cough. Interestingly, a study done on Dachshunds with Hansen type 1 IVDD found that body weight and body condition score did not affect disc calcification, however, shorter vertebral column length and shorter distance from tuber calcaneus to midpatellar tendon did predispose to acute disc herniation.[5] It has been noted that nonchondrodystrophic dogs maintain their notochordal cells into adulthood, whereas chondrodystrophic dogs do not. In young chondrodystrophic dogs, notochordal cells made up 13% of the nucleus cell population which fell to only 0.4% in adults.[9] This suggests that notochordal cells play a key role in disc homeostasis.[3] Hansen Type II is defined as rupture of the inner annulus fibrosus with partial displacement of the nucleus pulposus into the disrupted annulus. This results in a protrusion of the materials into the canal. Hansen type II most commonly occurs in large breed dogs, such as German Shepherd dogs and Labrador retrievers, between 5 and 12 years of age.[6] The patients usually present with chronic, progressive ataxia and are non-painful.[8] While these differences can be helpful in differentiating and guiding treatment, there are exceptions which makes imaging vital. Based on Stella's age and breed, Hansen type II was predicted, but the acute, painful nature and MRI findings diagnosed her with Hansen type I. Making the distinction is necessary prior to treatment because Hansen type II is usually less responsive to intervention as opposed to Hansen type 1.

Micturition problems are very common with spinal cord injuries and disease. In Stella's case, she was unable to urinate which is a common finding in dogs with lesions from T3-L3 due to loss of coordination of the micturition cycle. The lesion interrupts cranially projecting sensory pathways, caudally projecting motor pathways, and conscious recognition of a full bladder.[7] The 3 nerves involved in micturition include the pudendal nerve, hypogastric nerve, and pelvic nerve. Loss of coordination from thoracolumbar lesions results in sustained neural activity of somatic and parasympathetic nerves and loss of inhibition to the pudendal nerve which results in increased external urethral sphincter tone.[7,8] This condition is characterized by a distended bladder that is hard to express, also known as an upper motor neuron bladder.

With an injury to the spinal cord, the ascending and descending pathways can become dysfunctional. The 3 main pathways affected include proprioception, motor, and nociception. These pathways are lost in a certain order and return in the opposite order due to their structure (myelination and size) with larger, heavily myelinated fibers affected first. The proprioceptive pathway has large, heavily myelinated fibers that are highly susceptible to damage and therefore it is the first function lost and the last to return. The motor pathway has smaller, less myelinated fibers and is the second function lost. Loss of the nociceptive pathway signifies significant spinal cord damage. It is the last function lost but the first to return due to the small, unmyelinated fibers. In Stella's case, paraplegic deep pain positive, nociception was intact and motor and proprioception were absent. Based on how somatosensory pathways recover, Stella's motor function will come back prior to proprioception.

**Treatment and Management**

IVDD can be treated surgically or conservatively. The decision between the two is based on the clinical picture. Conservative therapy is best for patients with pain and/or minimal

neurologic deficits (i.e. ambulatory or only mild GP ataxia). It involves strict cage rest for 4 to 6 weeks and pain medications, if needed. Corticosteroids are usually contraindicated due to owner compliance with cage rest. Unfortunately, recurrence after medical management commonly occurs in 6 months to 1 year in 30 to 50% of cases.[6, 10] Surgery is indicated in patients with severe neurologic deficits (paraplegia, spinal hyperesthesia, and loss of nociception), recurrent episodes, and when medical management isn't working. The most common surgeries include hemilaminectomy, dorsal laminectomy, and pediculectomy. While there is little difference between the surgeries and recovery, hemilaminectomies provide minimal spinal cord manipulation, allow for more disc retrieval, and are less frequently associated with post-surgical constrictive laminectomy.[6] It is common for surgeons to prophylactically fenestrate various disc spaces to reduce recurrence from 2.7 to 42% to 0 to 24%. Fenestration involves making a window in the anulus and removing accessible nucleus pulposus. It is thought to incite an inflammatory response and result in phagocytosis of necrotic disc material. Surgical complications from any of the aforementioned surgical options include seroma formation, hemorrhage, incomplete removal of disc material, constrictive fibrosis, and in severe cases, progressive myelomalacia.[6]

Recovery to full *functional* potential following surgery is seen in 85 to 90% of cases. The recovery rate drops drastically when the patient is deep pain negative, usually around 50%, and lowers even more when they are deep pain negative for more than 24 to 48 hours, usually around 5% recovery rate.[8] A study done in 2018 found that 42% of dogs were ambulatory by 2 weeks and 79% were ambulatory by 4 weeks. The recovery rate was affected by the age and the weight of the patients, with larger and older patients taking longer to recover.[6]

Based on Stella's neurologic status, she underwent a left hemilaminectomy and fenestration at L2-L3. Surgery was routine and recovery from anesthesia was uneventful. Prior to

anesthetic recovery, a urinary catheter was placed due to her inability to urinate. Stella remained in the hospital for 10 days where she was evaluated daily for neurologic changes, started in a rehabilitation program, and monitored for bladder control and UTIs due to prolonged urinary catheter placement. Studies have shown that catheterization over 3 days was directly related to an increased incidence of UTIs, around 50%.[2] Post-hemilaminectomy, Stella was on placed on LRS fluids above maintenance due to methemoglobinuria from muscle damage, maropitant for hypersalivation and nausea, and a fentanyl CRI for pain control. She was then switched to Tylenol 4 and gabapentin for pain control, carprofen for IVC edema, and trazodone for anxiety. For management of Stella's bladder dysfunction, she was started on diazepam, a skeletal muscle relaxant that relaxes the external urethral sphincter, prazosin, a sympatholytic that relaxes the internal urethral sphincter, and bethanechol, a parasympathomimetic that contracts the detrusor muscle. She was closely monitored for hypotension due to prazosin and parasympathetic signs such as salivation, lacrimation, urination, and defecation due to bethanechol. Midway through her hospital stay, Stella began showing parasympathetic signs such as diarrhea, inappetence, and hypersalivation likely due to bethanechol. Her bethanechol dose was halved and she was started on metronidazole. She was also presumed to have a UTI, based on debris seen on ultrasound, and was started on amoxicillin. A urine culture was performed to ensure Stella was on the right antibiotic. The culture came back with *Enterococcus faecalis* and *Proteus mirabilis* which were susceptible to amoxicillin. One-week post-hemilaminectomy, Stella's neurologic status was upgraded to "non-ambulatory paraparetic" as she was regaining motor in her pelvic limbs.

**Case Outcome**

Stella was discharged 10 days post-hemilaminectomy. At this time, she was non-ambulatory paraparetic with voluntary bladder control and required a Help 'em Up® harness

and/or cart to get around. She was confined to strict cage rest for 4 to 6 weeks and was allowed to be cart or leash-walked for 10 minutes 2 to 3 times a day. She was sent home with gabapentin, trazodone, prazosin, bethanechol, amoxicillin, and metronidazole. She continued rehab on an outpatient basis in Flowood. While home, Stella was dribbling urine constantly and leaking diarrhea. Based on the fact that she was posturing to urinate and completely voiding her bladder, Stella's bethanechol was discontinued and clinical signs resolved.

At her 4 week recheck, Stella was still non-ambulatory paraparetic and required a cart and/or help-em-up harness. Strict cage rest and rehab was continued for 4 more weeks along with gabapentin and trazodone. At her 8 week recheck, Stella's neurologic status had upgraded to "ambulatory paraparetic". She was able to walk without assistance and was taken off strict cage rest. At this time, Stella continues outpatient rehab and is no longer on any medications.

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