Long-term neurologic outcome of hemilaminectomy and disk fenestration for treatment of dogs with thoracolumbar intervertebral disk herniation: 831 cases (2000–2007)

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Objective—To determine the proportion of dogs with thoracolumbar intervertebral disk herniation (IVDH) that successfully recovered following hemilaminectomy and fenestration, the time to ambulation (TTA) in affected dogs after surgery, and the frequency of urinary and fecal incontinence in recovered dogs and to document long-term complications.

Design—Retrospective case series.

Animals—831 dogs with thoracolumbar IVDH treated by hemilaminectomy and concomitant disk fenestration by the same surgeon.

Procedures—For all dogs, neurologic deficits before surgery had been assessed with a modified grading system. Dogs were reexamined after surgery over a period of 3 to 6 months, and follow-up evaluation was performed at > 12 months. The proportion of dogs that neurologically improved after surgery, TTA, and incidence of fecal or urinary incontinence in recovered dogs were compared among dogs with various grades of neurologic dysfunction before surgery.

Results—Of 831 dogs, 122 had unsuccessful outcomes and 709 had successful outcomes. Of 620 dogs with intact deep nociception before surgery, 606 (97.7%) were ambulatory after surgery. Despite maintaining the ability to walk, 7 dogs were judged to have an unsuccessful outcome because the severity of ataxia did not improve. Of 211 paraplegic dogs with loss of deep nociception, 110 (52.1%) dogs became ambulatory after surgery. Long-term complications included incontinence, permanent neurologic deterioration, and self-mutilation. Dogs with paraplegia before surgery had a higher frequency of urinary or fecal incontinence, compared with dogs that were ambulatory.

Conclusions and Clinical Relevance—Prognosis for dogs with thoracolumbar IVDH that retain deep nociception in at least 1 of the pelvic limbs or tail before surgery was good. (*J Am Vet Med Assoc* 2012;241:1617–1626)

Thoracolumbar IVDH is a common disorder of the vertebral column mainly seen in chondrodystrophic dogs.[1–23] Surgical decompression and removal of the extruded disk material is a widely accepted treatment modality, especially for disk extrusion. Disk protrusion typically occurs in nonchondrodystrophic larger dogs at older ages with chronic onset, but either type of disk herniation can occur in any breed of dog.[3,7–9] Clinical signs of thoracolumbar IVDH range from back pain, ambulatory or nonambulatory paraparesis, paraplegia, urinary dysfunction, or loss of deep nociception in the most severe cases. Mild clinical signs can be intermittent, slowly progressive, or chronic, but acute onset of paraplegia with loss of deep nociception can also occur as the first sign. Information regarding the proportion of dogs that recover from surgery, estimated time for recovery, and perioperative and long-term complications associated with surgical treatment can influence decision-making about whether to perform the expensive surgical intervention. Prognostic indicators for functional recovery include severity and clinical course of the neurologic deficit at preoperative examination,[8,9,13,14,21] findings on diagnostic imaging,[22,23]

Abbreviations

CI	Confidence interval
DM	Degenerative myelopathy
FI	Fecal incontinence
IVDH	Intervertebral disk herniation
PF	Prophylactic fenestration
PHM	Progressive hemorrhagic myelomalacia
TTA	Time to ambulation
UI	Urinary incontinence

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serum and CSF biomarkers,[24] and postoperative neurologic status.[13,20] As a possible preoperative prognostic indicator, the rate of onset of clinical signs in paraplegic dogs without deep nociception has been analyzed and the results suggested that rapid loss of motor function is a poor prognostic indicator, compared with slow progressive loss of ability to ambulate.[13] However, another study[14] in paraplegic dogs with intact deep nociception demonstrated conflicting results. Thus, it is inconclusive whether the rate of onset of clinical signs is a reliable prognostic indicator in paraplegic dogs.

Studies[8–10,15–19] have suggested that prognosis for voluntary ambulation in dogs with thoracolumbar IVDH in which deep nociception remains intact is good following medical or surgical treatment. In contrast, loss of deep nociception is associated with a worse outcome.[20–22] The duration of deep nociception loss has been one of the predictable factors, and early surgical intervention via spinal cord decompression within the set period of time ranging from within 12 hours[13,22] to 48 hours[22] has been advocated. However, other studies[13,25] have found that dogs with loss of deep nociception for > 72 hours still had a good functional outcome.

Clinical outcome after surgical intervention in dogs that lack deep nociception ranges from death because of PHM to successful functional recovery. Reported percentage of functional recovery in dogs without deep nociception varies from 38% to 76%, and the risk of PHM is thought to be approximately 10%.[9,13,20,22,23,26,27] The variability in reported outcomes might be a reflection of the small sample size in several of the studies, differences in the imaging and surgical approaches used, differences in duration of deep nociception loss, and the accuracy of assessing deep nociception.[28] Deep nociception is assessed by observing the dog's reaction to a toe pinch,[7,13,29] but some paraplegic dogs do not have the immediate, typical reaction to these stimuli, suggesting the deep nociception is present but partially or incompletely lost. Although deep nociception status in paraplegic dogs is the most important prognostic indicator, the prognostic value of incomplete loss of deep nociception, unilateral loss of deep nociception, or bilateral loss of deep nociception in the limbs but retention in the tail has not been determined.

Dogs with intact deep nociception have excellent to good functional outcome after surgery, but TTA varies.[30] Recurrent urinary tract infections and residual paraparesis have been extensively reported, but the risk of mild UI and FI in dogs that recovered from surgery or other long-term complications such as persistent postoperative neurologic deterioration or self-mutilation have not been reported until recently.[20,31] Micturition dysfunction is a well-known neurologic disorder in paraplegic dogs with spinal cord injury, especially for dogs with absent deep nociception. However, only a few studies[20,32] have assessed the prevalence of UI and FI in dogs that recovered from severe spinal cord injury. Urinary incontinence or FI can be a daily concern for owners, even though their dogs regain the ability to walk, especially for indoor dogs, and it can be the primary reason for owners to bring their dog to a veterinary hospital.[33]

Although thoracolumbar IVDH in dogs is one of the extensively documented diseases in veterinary neurology, information regarding long-term outcome is still insufficient.[20] The goal of the study reported here was to evaluate a modified preoperative grading system for dogs with thoracolumbar IVDH that underwent hemilaminectomy and concomitant fenestration. Our hypothesis was that the proportion of dogs that recover from hemilaminectomy and fenestration, TTA, and incidence of postoperative UI or FI in dogs would correlate with the preoperative neurologic grades.

Materials and Methods

Case selection—Medical records of dogs admitted to the Aikawa Veterinary Medical Center between August 2000 and December 2007 with myelographic or MRI evidence and definitive confirmation of thoracolumbar IVDH by hemilaminectomy by 1 surgeon (TA) were reviewed. Records for 883 dogs were identified and reviewed. Criteria for inclusion in this study were complete medical records, including history, initial neurologic grade, surgical report including fenestrated sites if done, clinical outcome, and noted complications, and complete diagnostic imaging reports including survey radiographs and myelography or MRI. Dogs that had follow-up until death or > 12 months were included. Fifty-two dogs without follow-up information (> 12 months) were excluded. Eight hundred thirty-one cases met the inclusion criteria.

Neurologic grading—On the basis of the severity of the neurologic dysfunction, dogs were assigned a grade of 0 to 5, where 0 represents a clinically normal dog,[34] grade 1 represents thoracolumbar paraspinal pain without neurologic deficits, and grade 2 represents ambulatory paraparesis. Grade 2 was further classified as mild, moderate, and severe on the basis of the subjective assessment of the severity of ataxia[34]; mild represents ambulatory paraparesis in which the dog can walk and run but the clinician can detect a slight to mild ataxia or conscious proprioception deficit, moderate represents ambulatory paraparesis with ataxia, and severe represents ambulatory paraparesis with obvious ataxia where the dog has the ability to stand up and take several steps (> 5 m) without support. Grade 3 represents nonambulatory paraparesis. Grade 4 represents paraplegia with intact or positive deep nociception in pelvic limbs and tail and was further classified as 4a or 4b on the basis of deep nociception status in both pelvic limbs and tail; grade 4a represents paraplegia with intact deep nociception in both pelvic limbs and tail, and grade 4b represents paraplegia with intact or decreased deep nociception in at least 1 of the pelvic limbs or tail. Decreased deep nociception was used to describe the response in dogs without a typical reaction to the toe pinch, such as immediate vocalization or turning of the head, but where a minimal or questionable reaction was observed. Grade 5 represents paraplegia with absent deep nociception in both pelvic limbs and tail. The assessment of the tail was conducted for dogs with a tail.

Diagnostic imaging—All dogs underwent lumbar myelography or MRI as the diagnostic imaging modality. Myelography was performed with the guide of

fluoroscopy and standard aseptic technique. The actual amount of contrast medium, iohexol,[a] to be administered was determined on the basis of fluoroscopic evaluation during the procedure. Iohexol was first prepared as a standard dose (0.3 to 0.5 mL/kg [0.13 to 0.22 mL/lb]) and cautiously added until the entire thoracolumbar subarachnoid space was filled with the contrast medium. The first lateral projection was taken during the injection. After removing the spinal needle, the ventrodorsal and 30° and 60° oblique projections were taken immediately. For dogs that were referred after MRI had already been performed, MRI images were reviewed and additional myelography was performed if the dogs had multiple IVDH compressive lesions, if > 2 days had lapsed between the time of MRI and referral, or if the dog had worsened neurologically after the MRI and prior to referral. Additional myelography was not performed in dogs that had conclusive MRI diagnosis of IVDH at the time of referral. For dogs that had grade 4b neurologic dysfunction before surgery, the correlation between the side of the lesion assessed by neurologic findings and actual lesion on myelography in dogs with asymmetry of deep nociception status was assessed.

Hemilaminectomy and fenestration—A dorsolateral hemilaminectomy was performed in all dogs immediately after myelography.[16,17,35] In some dogs, intraoperative modification to extend the site of hemilaminectomy was made if diffuse spinal cord compression due to the residual disk material or epidural hematoma was observed. For dogs that had diffuse or multiple spinal cord compressions that were thought to be responsible for the clinical signs, multiple hemilaminectomies on 1 or both sides were performed. For dogs that had bilateral spinal cord compression at the same intervertebral disk space, unilateral hemilaminectomy was performed on the more severe side as assessed by myelography. No dogs underwent bilateral hemilaminectomy at the same intervertebral disk space. At a minimum, PF was performed at the affected disk in all dogs. Most chondrodystrophic dogs had PF at T11-12 through L2-3 intervertebral disk spaces.[36] A thin (2- to 3-mm) layer of subcutaneous fat graft was placed on the hemilaminectomy site before closure.

Postoperative care—After surgery, all dogs were hospitalized for 4 to 7 days. Postoperative neurologic status was evaluated daily for any evidence of neurologic deterioration, compared with the preoperative neurologic grade. For dogs that had difficulty urinating, manual bladder expression 3 to 4 times daily was performed until the dog regained voluntary urination. Owners were instructed to perform manual bladder expression at least 3 to 4 times daily for dogs that did not regain the ability to voluntarily urinate by the time of discharge. For owners who could not perform daily complete bladder expression, the referral veterinarians were asked to hospitalize the dog or instruct the owner until the owner felt comfortable performing daily complete bladder expression. Diazepam[b] (0.5 mg/kg], PO, 20 minutes prior to bladder expression) and prazosin hydrochloride[c] (0.5 mg/kg, PO, q 12 h) were used to aid with bladder expression when indicated. For paraplegic dogs, owners were instructed to perform massage, range of motion exercise in the pelvic limbs, and assisted standing with support. For paraparetic dogs, walking on a nonslick surface with support if needed was encouraged.

Follow-up—Outcome was considered successful if the dog recovered deep nociception, voluntary urinary function, and the ability to walk for dogs with preoperative grades 3 to 5; if the dog had improved subjectively in severity of ataxia after surgery for dogs with preoperative grade 2; and if there was relief of paraspinal signs of pain for dogs with preoperative grade 1. Time to ambulation was defined as the number of days from surgical decompression until the dog was able to stand and take a series of steps without support.[15] If the dog had recovered between recheck examinations, the owner was questioned about the first day (or week) that the dog started walking between recheck examinations. Time to ambulation was categorized into the following groups: 0 to 14, 15 to 30, and 31 to 60 days and 2 to 6 and 6 to 12 months. Early recovery was defined as regaining the ability to ambulate unassisted within 14 days.

All dogs were reexamined 10 to 14 days after surgery at the time of suture removal. Monthly or bimonthly reexaminations were conducted by the authors for 3 to 6 months to evaluate the dog's recovery. Annual follow-up was made by means of reexamination by the authors throughout the study period (2005 to 2010). For dogs that were not reexamined by the authors for > 12 months from the time of previous examination, annual follow-up was performed by telephone interview with the owner or the referring veterinarian. On telephone interviews, to evaluate the signs of UI, FI, and other neurologic problems, owners or referring veterinarians were asked whether the dog had UI (retained voluntary urinary function but dribbled urine in the house, could not get outside in time, started walking before completely voiding, or urinating when excited) or FI (defecated in an inappropriate place, did not seem aware of the need to defecate, could not get outside in time or defecated when excited, or started walking before completing defecation). Respondents were also asked to describe the severity and frequency of the problems as well as any other neurologic problems that the dog has encountered (eg, self-mutilation).[20]

The incidence of prolonged neurologic deterioration or lack of neurologic improvement due to various reasons as well as persistent UI or FI and any episodes of self-mutilation were recorded. For each grade, the incidence of persistent postoperative UI or FI in dogs was evaluated. In dogs that did not regain deep nociception over the long term, the proportion of dogs that became ambulatory and their urinary and fecal function were evaluated.

Statistical analysis—Because TTA (number of days) in each grade were nonnormally distributed, the median TTA of each grade was calculated, and multiple comparisons were performed with a Kruskal-Wallis test, followed by a Steel-Dwass test. Additionally, the TTA was transformed into a logarithmic value (log [X + 1]) in base 10, and data were analyzed via a 1-way ANOVA, followed by a Tukey-Kramer test. The means of 95% confidence limits were calculated with trans-

formed data for TTA (days).[37] The mean TTA and its 95% CIs were expressed with the original data unit by antilogarithmic transformation. On the basis of the grades of neurologic dysfunction before surgery, multiple comparisons were made by use of a χ^2 test or Fisher exact test on the proportion of dogs that recovered from surgery, had early surgical recovery, and had FI or UI after surgery. All analyses were performed with standard software[d]; values of $P < 0.05$ were considered significant.

Results

Eight hundred thirty-one dogs met the inclusion criteria with a median follow-up period of 35 months (range, 1 to 123 months). Dogs of 9 chondrodystrophic breeds and 10 nonchondrodystrophic breeds were represented in this study. Seven hundred ninety-three of 831 (95.4%) dogs were chondrodystrophic and included the following breeds: Dachshund (n = 671 [80.7%] dogs; 666 Miniature Dachshunds and 5 Standard Dachshunds), Pembroke Welsh Corgis (25 [3.0%]), French Bulldog (24 [2.9%]), Shih Tzu (18 [2.2%]), Beagle (17 [2.0%]), Cocker Spaniel (12 [1.4%]), Pekinese (11 [1.3%]), Toy Poodle (6 [0.7%]), Bassett Hound (2 [0.2%]), and chondrodystrophic crossbreeds (7 [0.8%]). Thirty-eight (4.6%) nonchondrodystrophic dogs included the following breeds: Papillon (n = 10 [1.2%] dogs), Maltese (6 [0.7%]), Chihuahua (5 [0.6%]), Schnauzer (4 [0.5%]), Shetland Sheepdog (3 [0.4%]), Siberian Husky (2 [0.2%]), Cavalier King Charles Spaniel (2 [0.2%]), Labrador Retriever (1 [0.1%]), Miniature Pinscher (1 [0.1%]), Pug (1 [0.1%]), and nonchondrodystrophic crossbreeds (3 [0.4%]). Affected dogs were a median of 6.0 years old (range, 1 to 15 years). Five hundred (60.2%) dogs were male (133 were castrated), and 331 (39.8%) dogs were female (109 were spayed). Dogs had a median body weight of 5.6 kg (12.32 lb; range, 1.5 to 40.0 kg [3.3 to 88 lb]).

At the time of initial evaluation, 126 (15.2%) dogs had a history of at least 1 episode of clinical signs compatible with thoracolumbar IVDH that had been treated conservatively. Seven dogs had histories of hemilaminectomies performed by other veterinarians 4 to 37 months before initial evaluation. The remaining 698 (84.0%) dogs had no known past history suggesting thoracolumbar IVDH. The median duration of neurologic deficit prior to referral was 3 days (range, 1.5 hours to 10 months).

Preoperative neurologic status—Preoperative neurologic grade in 831 dogs was as follows: 5 dogs (0.6%) had grade 1, 274 (33.0%) had grade 2, 180 (21.7%) had grade 3, 84 (10.1%) had grade 4a, 77 (9.3%) had grade 4b, and 211 (25.4%) had grade 5 neurologic function. In dogs that had grade 4b neurologic dysfunction before surgery, 23 had asymmetry of the deep nociception status and 5 had bilateral deep nociception loss in the pelvic limbs with positive deep nociception in the tail.

Diagnostic imaging—Spinal cord compression was assessed with lumbar myelography in 829 dogs. Eight dogs were referred with the MRI diagnosis of IVDH in which additional myelography was performed in 6 dogs. Myelography revealed conclusive evidence of a spinal cord compressive lesion in all dogs.

Distribution of thoracolumbar IVDH sites—The most frequent site of surgically confirmed disk herniation was T12-13 (n = 218 [26.2%]), followed by T13-L1 (177 [21.3%]), T11-12 (120 [14.4%]), L2-3 (108 [13.0%]), L1-2 (105 [12.6%]), L3-4 (49 [5.9%]), L4-5 (15 [1.8%]), T10-11 (11 [1.3%]), L5-6 (2 [0.2%]), and T9-10 (1 [0.1%]). Fourteen (1.7%) dogs had multiple disk extrusions. In 11 (1.3%) dogs, the disk material spread out extensively in the vertebral column, which prevented the surgeon from being able to confirm the exact site of extrusion.

Twenty-three dogs that had grade 4b neurologic dysfunction before surgery had asymmetry in deep nociception. In 18 (78.2%) of these dogs, the worst side in terms of lack of deep nociception correlated with the side of actual compression. In 5 (21.8%) dogs, the lesion was located opposite the worst side of a deep nociception deficit.

Clinical outcome—Overall, 122 of 831 (14.7%) dogs were considered to have an unsuccessful outcome. Of 831 dogs, 25 (3.0%) died or were euthanatized because of the development of PHM after surgery. Seven (0.8%) other dogs died around the time of surgery from the following: respiratory arrest after anesthesia, possible colonic perforation, sepsis secondary to severe gastrointestinal disturbance, status epilepticus as a suspected myelographic complication, rectal perforation, acute pancreatitis, and unknown cause after hospital discharge. A diagnosis of PHM was made from clinical signs of an abnormal cutaneous trunci reflex, development of lower motor neuron signs in the pelvic limbs, and subsequent development of tetraparesis with partial Horner syndrome.

One dog that had grade 4b neurologic dysfunction and 3 dogs that had grade 5 dysfunction before surgery regained voluntary motor function after surgery but did not become ambulatory. Of 831 dogs, 71 (8.5%) dogs that had grade 5 dysfunction before surgery survived but did not regain deep nociception after surgery; 15 (1.8%) dogs that had grades 1 to 4a dysfunction before surgery did not have postoperative neurologic improvement or had deteriorated neurologic status for various reasons and were considered to have not recovered on the basis of our criteria. The remaining 709 of 831 (85.3%) dogs were considered to have recovered.

Grade 1 or 2—Two hundred seventy-nine dogs had grade 1 or grade 2 neurologic dysfunction before surgery. One dog died 2 days after surgery because of refractory status epilepticus that developed 1 day after surgery, and another dog permanently lost the ability to walk after surgery. Two dogs, both Pembroke Welsh Corgis, initially improved after surgery but then gradually worsened and progressed to have grade 4a dysfunction during a 14-month period. An additional 5 dogs did not improve after surgery in terms of the severity of ataxia. The remaining 270 of 279 (96.8%) dogs that had grade 1 or 2 dysfunction before surgery were considered to have a successful outcome; 234 of 270 (86.7%) dogs became ambulatory within 14 days, 23 (8.5%) within

15 to 30 days, and 5 (1.9%) within 31 to 60 days. Eight of 270 (3.0%) dogs took > 61 days to become ambulatory following a prolonged neurologic deterioration after surgery. The mean (95% CI) and median (25th to 75th percentile) TTA were 4.8 days (4.4 to 5.5 days) and 4 days (1 to 10 days), respectively.

Grade 3—One hundred eighty dogs had grade 3 neurologic dysfunction before surgery. Four dogs died shortly after the surgery. In these 4 dogs, death was related to anesthesia, possible sepsis, PHM caused by excessive intraoperative manipulation of the hard disk material that had lodged into the spinal cord, and rectal perforation secondary to severe rectal diverticulum related to perineal hernia. Two dogs permanently lost deep nociception after surgery, and 3 additional dogs did not become ambulatory after surgery. The remaining 171 of 180 (95.0%) dogs that had grade 3 dysfunction before surgery were considered to have a successful outcome; 140 of 171 (81.9%) dogs became ambulatory within 14 days, 20 (11.7%) within 15 to 30 days, and 7 (4.1%) within 31 to 60 days. Four of 171 (2.3%) dogs took > 61 days to become ambulatory after surgery. The mean (95% CI) and median (25th to 75th percentile) TTA were 7.7 days (6.6 to 9.0 days) and 7 days (4 to 13 days), respectively.

Grade 4a—Eighty-four dogs had grade 4a neurologic dysfunction before surgery. One dog lost deep nociception after surgery. Another dog gradually deteriorated neurologically to a grade 4a status during a 1-year period after the surgery. The remaining 82 of 84 (97.6%) dogs that had grade 4a dysfunction before surgery were considered to have a successful outcome; 61 of 82 (74.4%) dogs became ambulatory within 14 days, 10 (12.2%) within 15 to 30 days, and 5 (6.1%) within 31 to 60 days. Six of 82 (7.3%) dogs took > 61 days to become ambulatory after surgery. The mean (95% CI) and median (25th to 75th percentile) TTA were 10.5 days (8.4 to 13.2 days) and 10 days (6 to 16 days), respectively.

Grade 4b—Seventy-seven dogs had grade 4b neurologic dysfunction before surgery. One dog did not become ambulatory. The remaining 76 of 77 (98.7%) dogs that had grade 4b neurologic dysfunction before surgery recovered to be ambulatory and were considered to have a successful outcome; 53 of 76 (69.7%) dogs became ambulatory within 14 days, 8 (10.5%) within 15 to 30 days, and 7 (9.2%) within 31 to 60 days. Seven of 76 (9.2%) dogs took > 61 days to become ambulatory after surgery. Exact TTA was not recorded for 1 dog. For 75 dogs, the mean (95% CI) and median (25th to 75th percentile) TTA were 12.1 days (9.6 to 15.3 days) and 10 days (6 to 21 days), respectively.

Grade 5—Two hundred eleven dogs had grade 5 neurologic dysfunction before surgery. Twenty-four of 211 (11.4%) dogs died or were euthanized because of PHM. One dog died of pancreatitis 6 days after surgery. One dog died of a suspected colonic perforation 6 days after the surgery. One dog died of unknown cause shortly after hospital discharge. Seventy-one of 211 (33.6%) dogs survived, but neurologic status remained unchanged. Three dogs regained voluntary movement in both pelvic limbs but did not become ambulatory. The remaining 110 of 211 (52.1%) dogs that had grade 5 neurologic dysfunction before surgery recovered to regain deep nociception and to be ambulatory and were considered to have a successful outcome; 40 of 110 (36.4%) dogs became ambulatory within 14 days, 28 (25.5%) within 15 to 30 days, 20 (18.2%) within 31 to 60 days, 12 (10.9%) between 2 and 6 months, and 3 (2.7%) between 6 and 12 months. Exact TTA was not recorded for 7 (6.4%) dogs. For 103 dogs, the mean (95% CI) and median (25th to 75th percentile) TTA were 21 days (17.3 to 25.5 days) and 21 days (10 to 45 days), respectively.

Comparison of outcomes—No significant difference was found in the proportion (95.0% to 98.7%) of dogs that had a successful surgical outcome among those that had grade 1 or grade 2 through grade 4b neurologic dysfunction before surgery. The proportion (52.1%) of dogs that had grade 5 dysfunction before surgery and recovered from surgery was significantly ($P < 0.001$) lower than for dogs that had the other 4 grades of dysfunction.

The proportion of dogs that had an early recovery from surgery was significantly greater in dogs that had grade 1 or grade 2 (86.7%) neurologic dysfunction before surgery, compared with that of dogs that had grades 4a (74.4%; $P = 0.008$), 4b (69.7%; $P < 0.001$), and 5 (36.4%; $P < 0.001$) dysfunction. The proportion of dogs that had an early recovery from surgery was significantly greater in dogs that had grade 3 (81.9%) dysfunction, compared with that of dogs that had grades 4b ($P = 0.033$) and 5 ($P < 0.001$) dysfunction. The same proportion was significantly ($P < 0.001$) lower in dogs that had grade 5 dysfunction, compared with that of dogs that had the other 4 grades of dysfunction.

TTA—For dogs that successfully recovered from surgery, those that had grade 1 and grade 2 neurologic dysfunction before surgery had a significantly ($P < 0.001$) shorter mean TTA than did dogs that had other grades of dysfunction. Dogs that had grade 3 dysfunction before surgery had significantly shorter mean TTA than dogs that had grades 4b ($P = 0.014$) and 5 ($P < 0.001$) dysfunction, and the mean TTA was significantly longer in dogs that had grade 5 dysfunction before surgery, compared with dogs that had grades 4a ($P < 0.001$) and 4b ($P = 0.002$) dysfunction.

Outcomes in dogs that had persistent deep nociception loss—Seventy-five dogs continued to have persistent loss of deep nociception after surgical decompression; 20 dogs became ambulatory. Six of the 20 dogs appeared to walk with their pelvic limbs coordinated with the thoracic limbs and wagged their tails purposefully. They could also urinate and defecate voluntarily but had some degree of UI or FI. The remaining 14 dogs appeared to develop spinal walking and required daily assistance with urination. The TTA was recorded for 15 of the dogs; median TTA was 9 months (range, 2 to 28 months).

Neurologic deterioration—Fifteen dogs (7 Miniature Dachshunds, 7 Pembroke Welsh Corgis, and 1 Siberian Husky) deteriorated neurologically after surgery.

The 7 Miniature Dachshunds had a median follow-up time of 40 months (range, 30 to 64 months). Four had grade 5 neurologic dysfunction at last follow-up (27 months after surgery). Two of the 4 dogs had extremely severe spinal cord compression with disk material lodged deeply into the spinal cord at initial surgery. One dog with moderate compression had contrast uptake in a distended central canal throughout the thoracolumbar region noted on myelography. One dog developed grade 5 neurologic dysfunction shortly after the surgery due to iatrogenic disk extrusion into the spinal canal by the PF procedure. The remaining 3 dogs with a preoperative grade 2 neurologic dysfunction maintained the ability to walk, but were considered to have an unsuccessful outcome on the basis of the study criteria.

One dog, a Pembroke Welsh Corgi that had grade 2 neurologic dysfunction before surgery, had suspected aseptic necrosis or infection of the fat graft at 4 days after surgery. The dog developed hyperthermia (40.1°C [104.1°F]), continuous serosanguineous discharge from the surgical site, and anemia (PCV, 23%). At 5 days after surgery, neurologic function had deteriorated to grade 5. Treatment with cefazolin[e] (20 mg/kg [9.0 mg/lb], IV, q 8 h) was initiated, but the clinical signs did not improve. A massive hemorrhagic effusion around the hemilaminectomy site was noticed, and the previously placed fat graft was removed at a second exploratory surgery 9 days after the initial surgery. Bacterial culture results (aerobic and anaerobic) were negative. Histologic examination was not performed. The dog regained the ability to walk at 27 months but was considered a treatment failure on the basis of the study criteria.

Seven other dogs (6 Pembroke Welsh Corgis and 1 Siberian Husky) with preoperative status of grade 2, 3, or 4a neurologic dysfunction did not have improvement of paraparesis or paraplegia after the surgical decompression for chronic thoracolumbar IVDH. All of these dogs had 6- to 24-month histories of chronically progressive paraparesis or paraplegia before surgery. Myelography had revealed mild extradural spinal cord compression; mild disk protrusions were surgically confirmed in all dogs. Two of these dogs remained ambulatory, but gradual worsening of paraparesis was noted until the dogs died of unknown causes, one at 4 months after surgery and the other at 4 years after surgery. One dog progressed to develop grade 5 dysfunction over 7 months and died 15 months after surgery of an unknown cause. Two dogs had chronically progressive paraparesis and developed grade 4a dysfunction within 14 months after the surgery. The remaining 2 dogs had not improved at 22 and 24 months of follow-up.

Persistent UI and FI—In 709 dogs that had a successful surgical outcome, 92 (13.0%) had UI and 48 (6.8%) had FI. Thirty-five of 709 (4.9%) dogs had both UI and FI. Of the 270 dogs that had grade 1 or 2 neurologic dysfunction before surgery with a successful surgical outcome, 15 (5.6%) developed postoperative persistent UI and 9 (3.3%) developed persistent FI; 6 (2.2%) dogs had both UI and FI, of which 1 had a lower motor neuron lesion affecting the bladder and FI caused by severe spinal cord damage (sensorimotor function loss of the tail and absence of anal sphincter tone) with severe focal intramedullary hemorrhage associated with IVDH at L5-6. Lesion distributions in the 18 dogs were as follows: T11-L2 (n = 13), L2-3 (2), L3-4 (2), and L5-6 (1). Of the 171 dogs that had grade 3 dysfunction, 9 (5.3%) developed postoperative persistent UI and 5 (2.9%) developed persistent FI; 3 (1.8%) dogs had both UI and FI. Lesion distributions in the 11 dogs were as follows: T10-L2 (n = 10) and L2-3 (1). Of the 82 dogs that had grade 4a dysfunction, 12 (14.6%) developed postoperative persistent UI and 6 (7.3%) developed persistent FI; 4 (4.8%) dogs had both UI and FI. Lesion distributions in the 14 dogs were as follows: T11-L2 (n = 13) and L2-3 (1). Of the 76 dogs that had grade 4b dysfunction, 14 (18.4%) developed postoperative persistent UI and 8 (10.5%) developed persistent FI; 6 (7.9%) dogs had both UI and FI. Lesion distributions in the 16 dogs were as follows: T10-L1 (n = 15) and L4-5 (1). Of the 110 dogs that had grade 5 dysfunction, 42 (38.2%) developed postoperative persistent UI and 20 (18.2%) developed persistent FI; 16 (14.5%) dogs had both UI and FI. Lesion distributions in the 46 dogs were as follows: T10-L2 (n = 39), L2-3 (4), and L3-4 (2) or multiple lesions in T10-11 and T12-13 (1).

For dogs that successfully recovered from surgery, those that had grade 1 or 2 neurologic dysfunction before surgery had a significantly lower rate of postoperative UI (5.6%), compared with dogs with grades 4a, (14.6%; $P = 0.006$), 4b (18.4%; $P < 0.001$), and 5 (38.2%; $P = 0.006$) dysfunction. Dogs that had grade 3 neurologic dysfunction before surgery had a significantly lower rate of postoperative UI (5.3%), compared with dogs with grades 4a ($P = 0.011$), 4b ($P = 0.001$), and 5 ($P < 0.001$) dysfunction. Dogs that had grade 5 dysfunction had significantly higher rates of postoperative UI, compared with grades 4a ($P < 0.001$) and 4b ($P = 0.003$). Dogs that had grade 1 or 2 neurologic dysfunction before surgery had a significantly lower rate of postoperative FI (3.3%), compared with dogs with grades 4a (7.3%; $P < 0.001$), 4b (10.5%; $P = 0.016$), and 5 (18.2%; $P < 0.001$) dysfunction. Dogs that had grade 3 dysfunction had a significantly lower rate of FI (2.9%), compared with dogs that had grades 4b ($P < 0.001$) and 5 ($P < 0.001$) dysfunction. Dogs that had grade 4a dysfunction had a significantly ($P = 0.029$) lower rate of FI, compared with dogs that had grade 5 dysfunction.

Urinary incontinence manifested as a daily problem in 7 dogs. These dogs dribbled urine in the house or could not wait until able to go to the appropriate place to void. Some owners also reported that dogs with FI appeared to be aware of the need to defecate but could not go to the appropriate place in time, and some dogs did not appear to be aware that they were defecating. There was no case of constipation that required persistent enema or daily evacuation.

Self-mutilation—Five dogs that had grade 5 neurologic dysfunction before surgery developed episodic self-mutilations on their pelvic limbs, tail, penis, vulva, or back that started 1 to 12 months after surgery. One dog mutilated its penis severely 12 months after surgery and required penile amputation and scrotal urethrotomy; the dog had no further self-mutilating problems. One dog mutilated its vulva during estrus 8 months after surgery and was euthanized.

Discussion

The data collected in this study were similar to those in reports[4–7,10–12,14,16,30] on IVDH with respect to breed, body weight, age, and lesion distributions. The Dachshund was the most commonly affected breed in this study (80.7%), as consistently reported.[5,7,10–12,16,32,35]

Many reports[13,14,20–22] have cited the presence or absence of deep nociception as the most important physical examination–based parameter with regard to return of motor function in paraplegic dogs. However, incomplete loss of deep nociception and an asymmetric deep nociception status, such as unilateral loss of deep nociception, have not been well evaluated. The deep nociception status in the tail has not been assessed in some studies.[13,22,25] The objective of our grading system was to create a grading scheme that incorporates incomplete loss of deep nociception or asymmetric loss of deep nociception to minimize preoperative grading errors in dogs that have equivocal or questionable deep nociception status.

In this study, ambulatory status was regained after surgery in 606 of 620 (97.7%) dogs that had grades 1 to 4b neurologic dysfunction before surgery and in 110 of 211 (52.1%) dogs that had grade 5 dysfunction. This suggests that the preoperative neurologic grades 1 to 4b are good prognostic indicators for return of ambulation. Our definition of recovery in preoperative neurologic grade 2 resulted in 7 dogs that did become ambulatory after surgery being considered unsuccessful surgical outcomes because the severity of ataxia did not improve. This definition of recovery gives us more realistic information regarding possible deterioration in neurologic function for ambulatory dogs when considering surgical intervention.

Meticulous preoperative deep nociception assessment in both pelvic limbs and the tail should be performed in paraplegic dogs. In domestic animals, axons that conduct deep nociception course in the ipsilateral and contralateral ventral portions of the lateral funiculus medial to the spinocerebellar tract and form the lateral spinothalamic tract.[38,39] This pathway is interrupted frequently by axons leaving the path, entering the gray matter to synapse on another neuron whose axon rejoins the spinothalamic pathway on the same or opposite side. Thus, animals have a diffuse, bilaterally represented multisynaptic pathway for the conduction of impulses stimulated by noxious stimuli.[38,39] Although one might expect this diffuse conduction pathway system to theoretically prevent dogs from having asymmetry in deep nociception, this does not appear to be the case clinically. Our grading system distinguished grades 4a and 4b in an attempt to help determine the surgical side when diagnostic imaging was not conclusive and to provide a more detailed preoperative prognostic indicator. For dogs with grade 4b dysfunction, correlation between the side of the lesion and the worse side in terms of lack of deep nociception was seen in 18 of 23 (78.3%) dogs. Although deep nociception assessment has a degree of subjectivity by nature, this result suggests that if a paraplegic dog has asymmetry in deep nociception, it can be used to determine the side of decompression when the diagnostic imaging is not conclusive.

Myelography was primarily used as the diagnostic tool in this study. Although severe epidural leakage made accurate evaluation difficult in some cases, the site of spinal compressive was adequately determined in all cases by myelography, suggesting that myelography is an effective means of diagnosing spinal cord compression due to IVDH. There were 6 cases of postmyelographic seizures or neurologic deterioration, resulting in death in 1 dog and prolonged neurologic deterioration in 2 dogs, presumably because of either irritation by the contrast media or intracranial pressure elevation.[40,41] Although the incidence of complications associated with myelography appears to be low, owners should be informed of the potential risks.

We assessed whether this grading system can be used as a prognostic indicator for TTA in dogs that had a successful outcome. The proportion of dogs with early recovery (TTA within 14 days) was lower, and the mean and median TTAs were longer with more severe grades of neurologic dysfunction before surgery. Multiple statistical comparisons among neurologic grades demonstrated that preoperative grade can be used as a prognostic indicator for TTA.

The evaluation of deep nociception 1 month after surgery has been proposed as a potential prognostic indicator for late recovery in dogs.[20] Consistent with results of the study,[20] the majority of dogs (89.1%) in our study that had grade 5 neurologic dysfunction and successfully recovered from surgery regained deep nociception within 1 month. Recovery of motor function in dogs with persistent absence of deep nociception has been reported,[20] and it could suggest either survival of axons crossing the injury site or development of spinal reflex walking. Spinal reflex walking is believed to originate from local spinal circuits and has been shown to develop in adult dogs, cats, and rodents following spinal cord transection.[20,42–47] In our study, 6 dogs with persistent absence of deep nociception regained the ability to walk and their pelvic limbs' movements were coordinated with the thoracic limbs. They would also wag their tail when seeing their owner and regained voluntary urinary function but did have intermittent incontinence. Dogs might be able to regain the ability to walk despite persistent absence of deep nociception several months after the injury, and they might regain the ability to voluntarily urinate as well, even though they nearly always have some degree of incontinence.[11,20,22] This result suggests that even though it took longer than the ordinary recovery, the dogs with permanent loss of deep nociception after thoracolumbar IVDH still may become ambulatory. To promote recovery, further therapeutic options such as a rehabilitation program might have some beneficial effect.

Even though good outcomes should be expected in dogs with intact deep nociception in pelvic limbs or tail after surgical treatment, 15 dogs had permanent deterioration of neurologic status after surgery, as did 1 dog that had grade 3 dysfunction before surgery that developed PHM after surgery. The possible causes for this deterioration in neurologic status include myelography, impaired segmental spinal cord blood supply, persistent secondary spinal cord damage after disk extrusion, reperfusion injury, additional spinal cord dam-

age by patient handling under general anesthesia, excessive surgical manipulation of the spinal cord with poor functional reserve, possible PF complication, or possible undiagnosed underlying diseases such as infection, inflammation, hemorrhage, neoplasia, or DM in some cases.[48,49]

Surgical manipulation of the spinal cord during removal of the adhered disk material was the most likely cause of the permanent severe neurologic deterioration in 3 dogs in the present study. A recent report[50] describes lateral corpectomy for chronic thoracolumbar IVDH in which none of 15 treated dogs had transient worsening of their neurologic status in the immediate postoperative period. Further investigation to evaluate the indication, potential risk, and efficacy of this procedure will be necessary. One dog had possible aseptic fat graft necrosis of an excessively thick fat graft that is recognized as a rare complication after spinal cord decompression to cause deterioration within a few days of surgery.[51] Such adverse reactions have also been reported after placement of absorbable gelatin sponges.[35] Although results of conventional bacterial culture of this fat graft were negative, infectious epidural empyema and extradural hematoma could not be ruled out completely.

Older Pembroke Welsh Corgis with a chronic history of thoracolumbar IVDH seemed more at risk to have permanent deterioration after surgery in this study. Six of 8 Pembroke Welsh Corgis (> 9 years old; grade 1 to 4a neurologic dysfunction) did not recover fully after the surgery without any known cause for additional spinal cord damage. Clinical signs prior to surgery of all 6 dogs were sufficient to suspect DM in this breed, and the possibility of DM was discussed with the owner at the initial evaluation.[48,49] Degenerative myelopathy is usually diagnosed clinically, radiographically, myelographically, or with the use of CT or MRI, but definitive diagnosis requires histologic evaluation. Characteristic clinical course of DM is usually continuously progressive paraparesis without spinal hyperpathia for approximately 6 months or more. The myelographic results for dogs with DM are typically normal, but some dogs may have concurrent mild disk protrusions, which actually are not responsible for the clinical sign.[48,49] In retrospect, surgically confirmed mild disk protrusions in the 6 Pembroke Welsh Corgis in this study might have been an incidental finding.

Results of 1 study[32] on hemilaminectomy in dogs that were nonambulatory with intact deep nociception before surgery revealed that the prevalence of postoperative UI and FI was 7.8% and 3.9%, respectively. Our results of the prevalence of UI and FI in dogs that had a comparable grade of neurologic dysfunction before surgery (grades 3 to 4a) were similar (8.2% and 4.3%, respectively).[32] Another study[20] assessed the prevalence of postoperative UI and FI in dogs that regained deep nociception and the ability to walk after surgery, which were 12 (37%) and 15 (41%) of 37 dogs, respectively. In comparison, postoperative incontinence in neurologic grade 5–affected dogs in the present study were similar (38.2% of dogs had daily or intermittent UI and 18.2% had FI). The present study demonstrated that more severe grades of neurologic dysfunction before surgery were associated with a greater risk of UI or FI after surgery in recovered dogs.

Defecation and bladder voiding are the result of simultaneous relaxation of striated muscle and contraction of smooth muscles. For a coordinated defecation process, relaxation of the external anal sphincter and contraction of the colon, rectum, and abdominal muscles are consciously modulated by cortical input.[52,53] For coordinated micturition, contraction of the bladder and relaxation of the sphincter are maintained until voiding is complete and is modulated by the brainstem micturition center.[54] These pathways can be disturbed following spinal cord injury and result in contraction of both bladder and urethral muscle leading to incomplete evacuation or obstruction of urine flow.[52,53,55] The brainstem micturition center is also considered the upper motor neuron system for normal urination that coordinates the spinal reflex arcs involved in bladder filling. Therefore, dogs with spinal cord injury can have problems in bladder filling and have prolonged urine storage. Cystometrography in human spinal cord injury patients demonstrates sustained detrusor contraction with constant urethral resistance or detrusor contraction with fluctuating spasms in the dyssynergic urethral sphincter resulting in high intravesical pressures.[52,53] Studies[52,53] of human spinal cord injury patients found a relationship between ureterovesical and anorectal dysfunction. In such patients, anorectal manometry demonstrates the patterns of sphincter activity similar to that recorded in the patients' cystometrograms.

In humans with incomplete upper motor neuron spinal cord injury and FI, there is impaired rectal perception, increased rectal contraction, and anal relaxation in response to lower volumes of rectal stimulation, and reduced voluntary control of the external anal sphincter is also noted.[55] Human spinal cord injury patients often have no sensation of rectal fullness and are unable to consciously initiate reflex defecation. Therefore, defecation is planned on a regular basis to avoid FI.[52,53] The clinical signs of UI and FI in dogs with spinal cord injury are similar to what is described in human patients. Further investigation in dogs via electrophysiological tests (anorectal manometry, cystometrograms, and electromyography) will be needed to definitively diagnose these complications more fully.[52,53] The determination of UI and FI were primarily on the basis of the owner's description at recheck examination or on the telephone conversations with the owner in some cases. Similar to the human spinal cord injury patients, the incontinence in dogs with spinal cord injury can be managed successfully by taking dogs outside frequently. As these long-term complications become more understood, we will be able to inform dog owners more effectively.

Self-mutilation is considered to occur in animals with a complete lack of sensation as a result of paraesthesia and in bored or stressed animals as a stereotypical behavior.[56] This type of complication is rare, but owners who manage a paraplegic dog should be informed of the risk of this because it can be quite detrimental.

Limitations of this study include the fact that data was collected retrospectively and that a validated physical examination–based spinal cord injury score[29,57] was

not used. In addition, the severity of paraparesis in dogs with grade 2 neurologic dysfunction was subjectively assessed and outcome was compared with the preoperative neurologic status. It is recognized that this subdivision is highly subjective; however, from an owner's perspective, differentiating dogs with mild paraparesis from those with severe paraparesis may be vitally important.[34] Use of the grading scale used in this study has been reported,[34,58] but this modified clinical grading system has not been validated by assessing interobserver and intraobserver variability associated with its use.[29,57] Furthermore, because of the retrospective nature of the study, individuals abstracting information from the medical records were not blinded to outcome.[59] These factors may have resulted in clinician bias when assessing recovery, especially for dogs with grade 2 neurologic dysfunction before surgery.[59] Although improvement in the severity of ambulatory paraparesis with residual mild to moderate deficits may not be a satisfactory outcome in some respects, this criterion was used in the present study. One of the aims of the present study was to evaluate the TTA and incidence of UI or FI in dogs that recovered or maintained the ability to walk after surgery. In addition, some owners elect for their dog to have surgery not only to treat the degree of spinal cord injury but also to stop the progression of IVDH and prevent the possibility of IVDH recurrence. These owners seem to consider a recovery with residual mild to moderate paraparesis acceptable, as long as the surgery minimized the risk of IVDH recurrence.

The heterogenous follow-up times may have affected the outcome of long-term evaluations. The minimum follow-up time of 12 months in the present study may not have been long enough to analyze the true incidence of developing spinal reflex walking or the possibility of self-mutilation, and these incidences may have increased if all dogs were followed for a longer period. Finally, the noted TTA might have some inaccuracy, especially for dogs that took longer (> 6 months) to recover, considering that these data were on the basis of the owner's memory. To help overcome this, analysis of the proportion of dogs with early recovery was performed by grouping dogs according to ranges of dates (ie, 0 to 14, 15 to 30, and 31 to 60 days and 2 to 6 and 6 to 12 months). Furthermore, the proportion of dogs with a TTA > 6 months (0.4%) was considered to be small, but inaccuracies in this group might have affected the reported mean TTA.

Results suggested that the prognosis for thoracolumbar IVDH after surgical treatment with deep nociception intact in at least 1 of the hind limbs or tail is excellent. Determination of grade of neurologic dysfunction can be used as a prognostic indicator for recovery, TTA, and risk of incontinence. A small percentage of dogs without deep nociception may still regain their motor function regardless of recovery of deep nociception.

a. Omnipaque 240, Daiichi Pharmaceutical Co, Tokyo, Japan.
b. Horizon, 5-mg tablet, Astellas Co, Tokyo, Japan.
c. Minipress, 0.5-mg tablet, Pfizer Co, Tokyo, Japan.
d. SAS, version 9.1, SAS Institute Inc, Cary, NC.
e. Cefazolin injection, Fujita Pharmaceutical Co, Tokyo, Japan.
f. Gelfoam, Pharmacia, Kalamazoo, Mich.

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