Foramen magnum decompression for treatment of caudal occipital malformation syndrome in dogs

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Caudal occipital malformation syndrome (COMS) is a recently described disorder in dogs that is believed to be analogous to Chiari type I malformation of human beings.[1–3] The disorder has been recognized in a number of small-dog breeds, with Cavalier King Charles Spaniels being most commonly reported, and there is strong evidence to suggest that COMS is a heritable condition in Cavalier King Charles Spaniels.[1–5]

The underlying abnormality in dogs with COMS is a malformation of the caudal portion of the occiput, resulting in overcrowding of the caudal fossa. The diagnosis of COMS in dogs and Chiari type I malformation in people is most often made by means of **magnetic resonance imaging (MRI)**.[1,3,6,7] Affected individuals typically have some degree of cerebellar compression as a result of the occipital malformation, as well as constriction of the cervicomedullary junction in the vicinity of the foramen magnum.[1–3,6,7] It is thought that the bony compression at the cervicomedullary junction, in conjunction with turbulent CSF flow and pressure changes in the region, results in hypertrophy of the underlying meninges over time, and dural fibrosis has been documented histologically in humans and dogs with this malformation.[1,8] Focal meningeal hypertrophy at the level of the foramen magnum is believed to be responsible for progressive constriction at the cervicomedullary junction, which in turn leads to increased CSF pressure in the intracranial and spinal compartments over time.[1,8]

Similar to people with Chiari type I malformation, dogs with COMS can have a wide array of clinical signs, including cerebellovestibular dysfunction, myelopathy (usually cervical), and seizure activity. Syringohydromyelia, primarily involving the cervical portion of the spinal cord, is common in dogs with COMS, and a unique clinical sign in dogs with syringohydromyelia is persistent scratching activity directed toward the head, neck, and shoulder regions.[1–5]

The preferred treatment for people with symptomatic Chiari type I malformation is **foramen magnum decompression (FMD)**.[7,9,10] Most human patients that undergo FMD experience either a halt in progression or a sustained improvement in clinical signs.[7,9–11] In contrast, the efficacy of FMD in dogs with COMS has not been established, although a recent study[1] of 16 dogs with COMS reported that clinical signs resolved in 3 of 5 dogs that underwent FMD and improved in the remaining 2. By comparison, 5 of 10 dogs treated with medical therapy alone (eg, prednisone) were euthanatized because of a poor response to treatment, and the 1 dog that was not treated had no change in clinical status.[1] The purpose of the present report was to describe a method for FMD in dogs with COMS and results in 16 clinical cases.

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Surgical Procedure

Client-owned dogs with MRI evidence of COMS and no other identifiable concurrent neurologic disorders that could contribute to clinical signs of neurologic dysfunction were considered candidates for the FMD

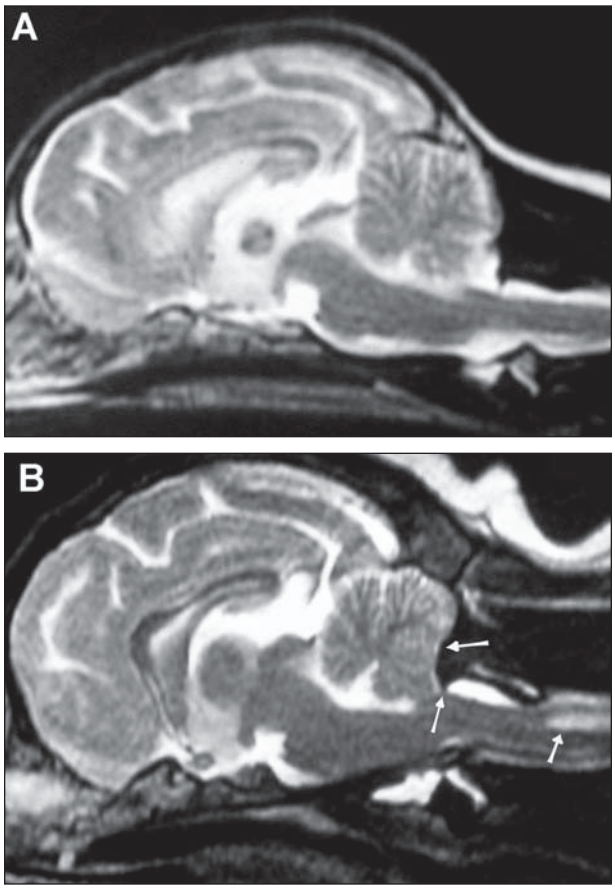

Figure 1—Midsagittal T2-weighted magnetic resonance images of a healthy small-breed dog (A) and a dog with caudal occipital malformation syndrome (COMS;B). Notice that in the dog with COMS, there is rostral displacement of the cerebellum by the occiput, obliteration of the dorsal subarachnoid space at the cervicomedullary junction, and cervical syringohydromyelia (arrows). (Reprinted with permission from Dewey CW. Myelopathies: disorders of the spinal cord. In: Dewey CW, ed. *A practical guide to canine and feline neurology.* Ames, Iowa: Iowa State Press [Blackwell Publishing], 2003;277–336.)

procedure. Dogs were considered to have COMS only if compression of the caudal aspect of the cerebellum by the caudal occipital bone and attenuation or obliteration of the dorsal subarachnoid space at the cervicomedullary junction were both evident on midsagittal magnetic resonance images of the brain (**Figure 1**).

For FMD, dogs were anesthetized, with the anesthetic protocol selected by the attending clinician. Cefazolin (22 mg/kg [10 mg/lb]) was administered IV at the onset of surgery and every 2 hours thereafter while the procedure continued. Mannitol (0.5 g/kg [0.23 mg/lb], IV) and prednisone (30 mg/kg [13.6 mg/lb], IV) were administered prior to surgery, according to the surgeon's discretion.

Dogs were placed in sternal recumbency, with the neck ventroflexed (**Figure 2**), and the dorsal aspect of the head and neck was shaved from the level of the bregma to the level of the third or fourth cervical vertebra, with a width approximately equal to that of the atlas. A dorsal midline incision was made extending from approximately 1 cm rostral to the external occipital protuberance cranially to the middle of the second cervical vertebra caudally. The superficial dorsal cervical musculature (**Figure 3**) was separated at the median raphe, exposing the underlying biventer cervicis muscles. The paired biventer cervicis muscles were separated on the midline, exposing the rectus capitis dorsalis muscles (**Figure 4**). The caudal aspects of the rectus capitis dorsalis muscles were removed from the cranial half of the second cervical vertebra with a combination of sharp dissection and periosteal elevation, and the muscle bellies were split on the midline. The cranial aspects of the rectus capitis dorsalis muscles were then sharply incised from the nuchal crest, exposing the caudal portion of the occiput and the arch of the atlas. Hemorrhage was controlled with electrocautery.

A high-speed air drill with a 3- to 4-mm-diameter round drill bit and Lempert rongeurs were used to remove a portion of the occiput and the dorsal aspect of the first cervical vertebra (**Figure 5**). The lateral limits of the bony defect were the atlanto-occipital joints and the lateral vertebral foramina of the atlas. The rostral extent of the defect was approximately halfway between the external occipital protuberance and the dorsal aspect of the foramen magnum. The caudal limit of the defect was between half and three-quarters the length of the arch of the atlas.

The meninges that were exposed and the dorsal atlanto-occipital membrane were incised longitudinally with a scalpel blade. This tissue was then removed from the circumference of the defect or marsupialized (**Figure 6**) to surrounding musculature with 5-0 absorbable suture in a simple interrupted pattern. Absorbable gelatin sponge was placed in the foramen magnum defect at the discretion of the attending surgeon. Closure was routine.

Figure 2—Photograph illustrating positioning of the head and neck for foramen magnum decompression (FMD) in a dog with COMS.

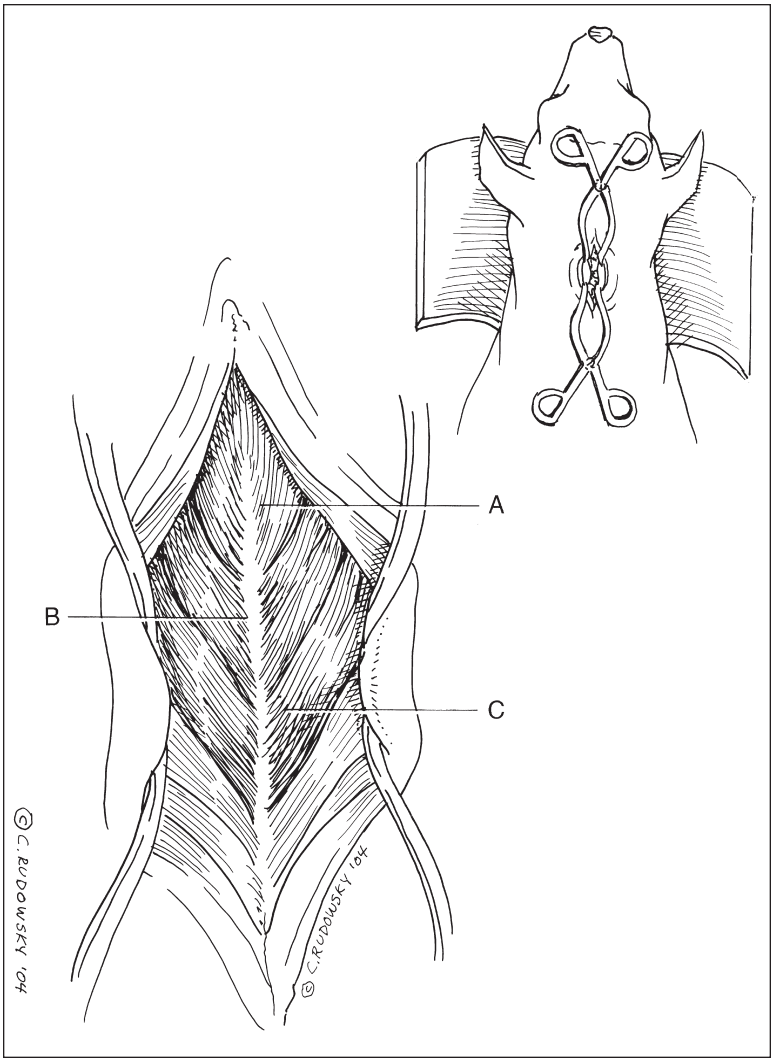

Figure 3—Illustration of the initial surgical approach for FMD in dogs with COMS. A = Occipitalis muscle. B = Median raphe. C = Cervicoscutularis and cervicoauricularis superficialis muscles.

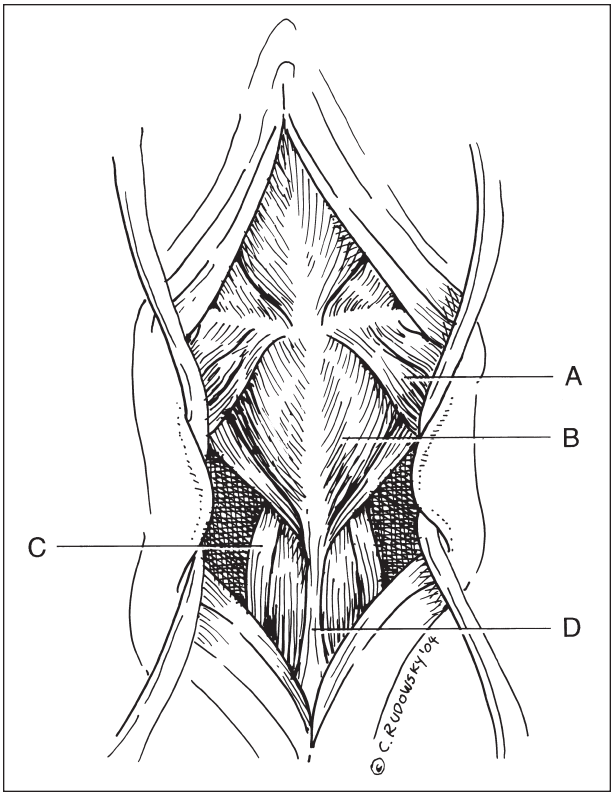

Figure 4—Illustration of deeper structures encountered during FMD in dogs with COMS. A = Biventer cervicis muscle. B = Rectus capitis dorsalis muscle. C = Multifidus muscles. D = Nuchal ligament.

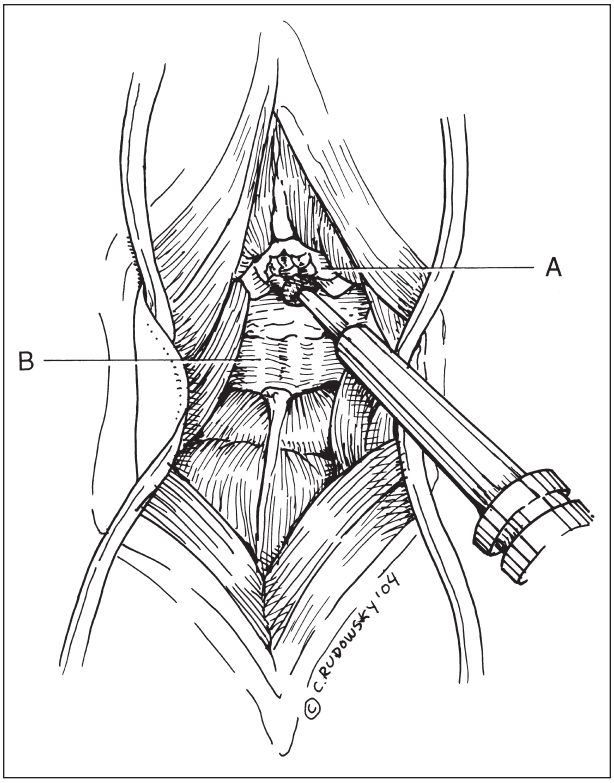

Figure 5—Illustration demonstrating removal of a portion of the occiput in the region of the foramen magnum and the dorsal aspect of C1 for FMD in dogs with COMS. A = Caudal portion of the occiput. B = Dorsal arch of C1.

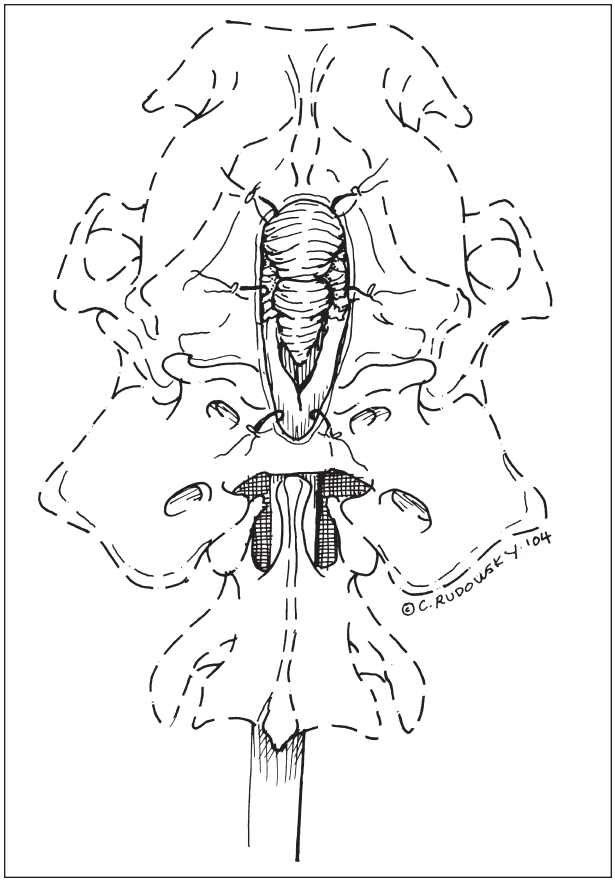

Figure 6—Schematic illustration of the completed FMD procedure in dogs with COMS. Exposed meninges have been incised longitudinally and marsupialized to the surrounding musculature.

All dogs were treated with prednisone (0.5 mg/kg, PO, q 12 h) for 3 to 5 days after surgery. The dosage of prednisone was progressively decreased over the succeeding 1 to 4 weeks, and administration was discontinued if possible. In all dogs, opioid analgesics were administered every 4 to 6 hours for the first 48 hours after surgery; additional analgesics were given after this period if necessary. Depending on the attending clinician's preference, cephalexin (22 mg/kg, PO, q 8 h) was administered for 10 days after surgery. All dogs were discharged from the hospital between 3 and 5 days after surgery, and owners were instructed to strictly confine their dogs for 7 to 10 days after surgery, after which dogs were allowed to gradually return to their normal activities. Dogs were reevaluated between 2 and 4 weeks after discharge from the hospital, with additional follow-up examinations performed as necessary. Additional follow-up information was obtained through telephone consultations with owners and referring veterinarians.

Results

The FMD procedure was performed on 16 dogs with COMS. Nine dogs underwent surgery at the Long Island Veterinary Specialists, and 7 dogs underwent surgery at the County Animal Specialty Group. All surgical procedures were performed by a board-certified neurologist or board-certified surgeon. Details for 4 of these dogs have been published previously.[1]

The 16 dogs consisted of 9 Cavalier King Charles Spaniels, 2 Maltese, 2 Pugs, a Miniature Poodle, a Yorkshire Terrier, and a Pomeranian. Mean age at the time of surgery was 3.9 years (range, 10 months to 9 years). Mean body weight was 6.7 kg (14 lb; range, 1.7 to 12.9 kg [3.8 to 28.4 lb]). There were 9 spayed females, 1 sexually intact female, and 6 castrated male dogs.

Clinical signs prior to surgery included cervical hyperesthesia (13 dogs, 2 of which also resented palpation of the head), diminished menace responses (7), positional ventrolateral strabismus (7), excessive scratching behavior (6), torticollis (3), abnormal mental status (2), "fly-biting" episodes (2), head tilt (2), chewing at the feet (2), excessive licking (1), excessive eye rubbing (1), generalized seizures (1), and nonambulatory paraparesis (1). Mean duration of clinical signs prior to surgery was 32 weeks (range, 1 to 208 weeks).

Neuroanatomic localization of lesions included multifocal CNS dysfunction (n = 7), isolated cervical myelopathy (6), isolated cerebellovestibular dysfunction (2), and L4-S1 myelopathy (1). All 7 dogs with multifocal CNS dysfunction had evidence of cerebellovestibular and cervical spinal cord disease.

In 15 of the 16 dogs, cervical syringohydromyelia was evident on magnetic resonance images. The dog with L4-S1 myelopathy also had lumbar syringohydromyelia. In 5 dogs, CSF was submitted for analysis. Results were normal in 3 dogs, whereas 1 dog had a high protein concentration (79 mg/dL; reference range, < 48 mg/dL) with a normal WBC count, and 1 dog had mild mixed-cell pleocytosis (10 WBCs/μL; reference range, 0 to 5 WBCs/μL) with a normal protein concentration.

Fifteen of the 16 dogs had received medical treatment prior to undergoing FMD. Thirteen dogs were treated with glucocorticoids PO, and 1 of these also received periodic injections of dexamethasone. In 8 of the 13 dogs treated with glucocorticoids, this was the only treatment. The other 5 dogs were treated with gabapentin (n = 2), felbamate (1), butorphanol and fentanyl (1), or furosemide (1) in addition to glucocorticoids. One dog was treated with carprofen and furosemide prior to surgery, and 1 was treated with phenobarbital. The remaining dog did not receive medical treatment prior to undergoing FMD.

The meninges were marsupialized in 5 dogs. Absorbable gelatin sponge was placed in the foramen magnum defect in 11 dogs. Meningeal biopsy specimens from 6 dogs were submitted for histologic examination. Four dogs had evidence of dural fibrosis; 2 of these 4 dogs had lymphocytic inflammation of the dura, and 1 had areas of mineralization. The remaining 2 dogs had evidence of osseous metaplasia of the dura.

No intraoperative complications occurred in any of the 16 dogs. Postoperative complications occurred in 2 dogs after the initial surgery and in 1 of these dogs during follow-up surgery. One dog that had had a head tilt prior to surgery had a more severe head tilt after surgery; however, the head tilt resolved within 1 month. A second dog developed signs of neck pain 9 days after surgery; signs resolved within 1 month with cage confinement and glucocorticoid treatment. After a second FMD procedure, this dog was nonambulatory tetraparetic. The dog remained nonambulatory until its death during the second week after follow-up surgery.

Clinical signs of neurologic dysfunction resolved in 7 dogs and improved in 6 others following FMD. Persistent scratching activity was the most important clinical sign in 4 of the 6 dogs with residual clinical signs. Three of these 4 dogs had no neurologic deficits at the time of the last follow-up examination, and the remaining dog had a slight right-sided head tilt and decreased conscious placing reactions in the pelvic limbs. One of the remaining dogs with residual clinical signs had had nonambulatory paraparesis prior to undergoing FMD and regained full use of the right pelvic limb, but did not have any improvement in use of the left pelvic limb. The other dog with residual clinical signs had torticollis and hypermetria of the right thoracic limb at the time of the final follow-up examination.

One dog was euthanatized approximately 6 weeks after undergoing FMD because of an apparent increase in the severity of fly-biting and foot-chewing behavior. The dog had been treated with phenobarbital for the fly biting prior to surgery, and administration of phenobarbital had been discontinued after surgery. Because of the increased severity of fly biting and foot chewing, the dog was treated with felbamate. The fly-biting episodes, which had been occurring almost constantly throughout the day, decreased to 5 to 6 episodes/d after treatment with felbamate was initiated, but the severity of fly biting and foot chewing again increased despite continued treatment with felbamate, and the owners refused additional treatment.

One dog did not have any improvement in clinical signs following FMD, and results of follow-up MRI were suggestive of scar formation at the FMD site. A syringosubarachnoid shunting procedure was performed on this dog at another institution; the dog did not have any improvement in clinical signs following this second procedure and was receiving long-term furosemide treatment at the time of final follow-up.

Three of the 16 dogs were clinically improved after undergoing FMD but subsequently developed signs of neurologic deterioration. In all 3 dogs, results of follow-up MRI were suggestive of compressive scar tissue formation at the foramen magnum and a second FMD procedure was performed to remove this scar tissue. Two of the 3 dogs improved clinically following this second procedure and were judged to be clinically improved at last follow-up examination. The third dog had nonambulatory tetraparesis following the second procedure and died 9 days after surgery. The dog apparently went into shock and experienced cardiac arrest. An abdominal fluid wave was balloted in this dog, and rupture of a viscus (eg, colon) was suspected. However, a necropsy was not performed.

Overall, therefore, 14 dogs survived, 1 dog died, and 1 was euthanatized. Clinical signs resolved in 7 of the 14 dogs that survived, improved in 6 dogs, and remained unchanged in 1 dog. Mean duration of follow-up for the 14 dogs that survived was 17 months (range, 6 to 32 months).

Medical treatment was discontinued in all 7 dogs in which clinical signs resolved following FMD and in 3 of the 6 dogs in which clinical signs improved. In 1 of the improved dogs, glucocorticoid administration

was discontinued following FMD, but administration of gabapentin was required to control scratching activity. In 2 dogs, the dosage of glucocorticoids was decreased following surgery, but administration was not discontinued; 1 of these dogs received gabapentin for scratching activity in addition to glucocorticoids after surgery, and the other received furosemide in addition to glucocorticoids.

Mean ± SD age at the time of surgery for the 7 dogs in which clinical signs resolved following FMD (4.4 ± 1.5 years) was not significantly (*t* test[a]; $P > 0.05$) different from mean age for the 6 dogs in which clinical signs improved (3.9 ± 1.4 years). However, mean duration of clinical signs prior to FMD was significantly ($P = 0.03$) longer for dogs in which clinical signs improved (78 ± 31.8 weeks) than for dogs in which clinical signs resolved (4 ± 1.4 weeks).

Discussion

Results for dogs described in the present report suggest that FMD may be an effective treatment for COMS, especially if performed early in the disease course. In human patients, the procedure is generally considered to be of low risk when performed by experienced neurosurgeons,[10,11] and our findings were similar, in that none of the dogs developed intraoperative complications. The most common residual clinical signs among dogs in the present report were sensory in nature (eg, scratching behavior). Similarly, in people with Chiari type I malformation that undergo FMD, clinical signs of motor dysfunction are much more likely to improve or resolve than are clinical signs of sensory dysfunction.[12]

Age at the time of surgery for dogs in the present report in which clinical signs resolved was not significantly different from age for dogs in which signs improved. This was somewhat surprising to us, in that, in general, children with Chiari type I malformation that undergo FMD have higher postoperative improvement rates than do adults.[11–13] However, only 3 of the 16 dogs in the present report were < 1 year old at the time of surgery. Thus, the lack of association between age and outcome may have been reflective of the low number of young dogs that were included.

In human patients with Chiari type I malformation, the degree of clinical success associated with FMD is inversely related to the duration of clinical signs prior to surgery.[7,10,12,13] Similarly, duration of clinical signs was substantially shorter in dogs in the present report in which clinical signs resolved following surgery (mean, 4 weeks) than in dogs in which clinical signs improved (mean, 78 weeks).

Postoperative compressive scar formation at the FMD site necessitating additional surgery has been reported in human patients with Chiari type I malformation, with 8% to 30% of human patients requiring additional surgery.[11,13,14] Of the 16 dogs in the present report, 4 required additional surgery because of confirmed (n = 3) or suspected (1) scar tissue formation at the FMD site.

Although FMD is generally accepted as the surgical procedure of choice for human patients with Chiari type I malformation, there is considerable controversy concerning which variant of the procedure is most appropriate.[9,11–13,15] Central to the differing opinions regarding the best variant of the FMD procedure is the fate of the thickened dura mater and arachnoid at the decompression site. Recommendations vary from partial dural dissection and removal (with preservation of part of the dura mater and all of the arachnoid), to resection of the dura mater and arachnoid with grafting of the resulting meningeal defect, to incision of the thickened dura mater and arachnoid and marsupialization to the surrounding musculature.[9,11–13,15] A major reason for preserving a water-tight dural closure (partial dissection or dural grafting) in people undergoing FMD is a desire to avoid postoperative complications associated with CSF leakage into the peripheral tissues. In people, CSF leakage has been associated with head and neck pain, nausea, and escape of CSF through the skin incision with the attendant risk of ascending meningitis. In general, these complications are self-limiting.[15] In a report[15] of 30 children with Chiari type I malformation that underwent FMD, only 3 developed leakage of CSF through the skin incision, and in all 3, the leakage resolved with bedside skin suturing.

There is some evidence that leaving the meninges partially intact or placing a dural graft over the defect can have negative consequences in human patients undergoing FMD. Preserving the arachnoid and part of the dura mater may allow continued compression, especially if this tissue becomes scarred or hypertrophied. Dural grafts may be pushed against the cerebellum by cervical syringohydromyelia fluid, creating a constrictive effect at the cervicomedullary junction. If fascia is used for dural grafting, this devascularized tissue may develop adhesions or form a scar at the cervicomedullary junction, which may be worse than the original constriction.[13] In the authors' experience, dogs do not have problems with leakage of CSF through the skin incision following intracranial surgery, even with wide resection of the dura mater and arachnoid, and to our knowledge, there are no reports of this complication occurring in dogs following intracranial procedures. None of the 16 dogs in the present report had evidence of CSF leakage through the skin incision. Similarly, the authors have not seen any signs of head and neck pain or nausea in dogs that have undergone intracranial procedures that involved dura mater and arachnoid resection. The authors chose to resect or marsupialize the dura mater and arachnoid because we thought that the potential benefits of removing this constrictive tissue far outweighed the potential risks of not doing so. The authors adopted the marsupialization technique after several dogs had developed scar tissue at the FMD site following meningeal resection. Although only 5 dogs had meningeal marsupialization performed, none of these dogs has yet required additional surgery.

Results for dogs in the present report conflict with those described in a recent report[16] of 4 Cavalier King Charles Spaniels with COMS treated by means of FMD. One of those 4 dogs died shortly after surgery, and the remaining 3 did not have any improvement in clinical signs by 3 months after surgery. All 3 surviving dogs were worse neurologically following surgery, and 2 of them remained so 1 month after surgery. There are several possible reasons for the disparate results between dogs in this previous report and dogs in the present

report. The previous report described only 4 dogs, and it is possible that the success rate would have been higher if more dogs had been treated. In addition, the FMD procedure used by the authors of the previous report was considerably different from the procedure described in the present report. In particular, those authors created a larger laminectomy defect by removing a portion of the dorsal lamina of C2 and placed a fascial graft in the foramen magnum. Finally, the follow-up period was only 3 months for the 3 surviving dogs in the previous report, whereas mean follow-up time for the 14 surviving dogs described in the present report was 17 months.

Adjunctive surgical procedures are occasionally performed in people with Chiari type I malformation that have had a suboptimal response to FMD. Such procedures usually involve placement of a shunt to divert syringohydromyelia fluid from the spinal cord region to another location for absorption.[13,17,18] Syringopleural and syringoperitoneal shunting procedures have been used as adjuncts to FMD in people and may hold some promise in the surgical management of dogs with COMS. However, use of these shunting procedures as a primary surgical solution is generally not recommended in people, as they do not address the cause of the syringohydromyelia.[13,17,19] Most dogs with COMS treated by the authors, including most of the dogs described in the present report, have large spinal cord cavitations that could feasibly accommodate the placement of a small shunting device.

In conclusion, the FMD procedure described in the present report appeared to be an effective treatment for most dogs with clinical signs of COMS. Results for these dogs also suggest that early surgical intervention is associated with an increased likelihood of resolution of clinical signs. Further study is needed to ascertain the value of meningeal marsupialization as well as the role of adjunctive surgical procedures, such as syringopleural and syringoperitoneal shunt placement.

a. PC-SAS statistical software, SAS Institute Inc, Cary, NC.

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