Cervical facet fractures

TO THE EDITOR: We read with interest the article by Aarabi et al.¹ (Aarabi B, Mirvis S, Shanmuganathan K, et al: Comparative effectiveness of surgical versus nonoperative management of unilateral, nondisplaced, subaxial cervical spine facet fractures without evidence of spinal cord injury. Clinical article. *J Neurosurg Spine* 20:270–277, March 2014).

The surgical management of unilateral cervical facet fractures without subluxation remains a topic of debate as the spectrum of injuries within this category is wide and variable. The decision to intervene operatively is usually driven by the presence of discoligamentous disruption seen on STIR MRI, as these fractures are at a higher risk of subluxation, delayed kyphosis, worsening pain, and neurological injury when managed with rigid bracing.

In the absence of discoligamentous injury and neurological deficit, many surgeons elect to brace these fractures. In their retrospective article, Aarabi et al. compared the outcomes of patients with unilateral facet fractures without subluxation that were either managed conservatively or operatively. The integrity of the discoligamentous complex in their patients was either normal or indeterminate. The authors found that operative treatment failed in only 1 (10%) of 10 patients who underwent surgery while conservative therapy failed in 9 (60%) of 15 treated with bracing (failure was defined as progressive translation or kyphosis or not attaining solid fusion). The mean time until failure was 38.4 days (range 5–103 days) from injury. Of note, none of the patients in whom treat-

ment failed presented with a neurological deficit. All of the patients in whom conservative treatments failed then underwent internal fixation.

This high percentage of patients in whom conservative treatment failed might be confounded by the presence of a larger percentage of patients with higher degrees of discoligamentous disruption (assessed by the injury severity score) within this subcategory compared to those patients who were successfully managed with bracing. Moreover, the decision to operate in patients initially treated with brace therapy was driven by radiographic evidence of translation or kyphosis, which in the absence of pain, dislocation, or neurological deficit does not necessarily warrant surgical intervention.

In our experience we find that rigid bracing for at least 3 months is an effective and successful method of managing patients with unilateral facet fractures without subluxation as long as the integrity of the discoligamentous complex is normal. Close follow-up with radiography and assessment of fusion with CT scanning or dynamic flexion and extension radiography is warranted to confirm fracture healing prior to the discontinuation of the hard collar or the crown halo vest (Fig. 1). Since May of 2013, we have assessed and managed 9 neurologically intact patients with unilateral facet fractures without instability. None of these patients has required surgery.

This study further confirms the complexity and spectrum that this injury represents. Spine surgeons should pay careful attention to what seems to be a benign fracture and further assess the severity of this injury with MRI. Furthermore, other risk factors such as age and

Fig. 1. An 18-year-old man who was intoxicated with alcohol fell off a bunk bed. He presented to the emergency room with neck pain. Findings on neurological examination were normal. **A:** Cervical CT demonstrated a left-sided nondisplaced facet fracture of C-6 (arrow). The patient was admitted and was placed in a Miami J collar. **B:** A STIR MR image showed disruption of the interspinous ligaments only with the rest of the discoligamentous complex being intact. The patient was followed up closely with standing lateral radiography (every 2 weeks initially). **C:** Following bracing for 3 months, a cervical CT scan showed complete healing of the fracture. The patient reported no neck pain and findings on his neurological examination were normal.

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bone health should be taken into account. Lastly, future studies directed at the understanding and the assessment of the degree of discoligamentous disruption would help predict successful treatment strategies.

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The authors report no conflict of interest.

Reference

1. Aarabi B, Mirvis S, Shanmuganathan K, Vaccaro AR, Holmes CJ, Akhtar-Danesh N, et al: Comparative effectiveness of surgical versus nonoperative management of unilateral, nondisplaced, subaxial cervical spine facet fractures without evidence of spinal cord injury. Clinical article. **J Neurosurg Spine** 20:270–277, 2014

RESPONSE: We appreciate the critical review of our manuscript by Drs. Dahdaleh, Smith, Lindley, and Hitchon.

The primary objective of our investigation was to define the natural behavior of a nondisplaced unilateral facet fracture under physiological loads. Other researchers have had the same purpose in their studies. Halliday et al.² evaluated the integrity of the discoligamentous complex (facet capsule, interspinous ligament, anterior longitudinal ligament, and posterior longitudinal ligament) in relation to the probability of instability and the need for surgical intervention. Spector et al.³ endeavored to relate the probability of subluxation or kyphotic deformity to the size of the fragment.

We agree with the statement that the “decision to intervene operatively is usually driven by the presence of discoligamentous disruption seen on STIR MRI, as these fractures are at a higher risk of subluxation, delayed kyphosis, worsening pain, and neurological injury when managed with rigid bracing” but could not understand the statement that the “high percentage of patients in whom conservative treatment failed might be confounded by the presence of a larger percentage of patients with higher degrees of discoligamentous disruption.” In our paper we clearly mention that we used MRI to confirm lack of spinal cord injury and to grade the level of injury severity of 7 ligaments of the cervical spine discoligamentous complex (anterior longitudinal ligament/anterior anulus, disc, posterior anulus/posterior longitudinal ligament, ligamentum flavum, joint capsule on right side, joint capsule on left side, and interspinous ligament). The injury severity score of a specific ligament was 0 if the ligament was intact, 0.5 if the injury was indeterminate, and 1 if the ligament was completely disrupted. For all 7 ligaments, the score ranged from 0 to 7. The mean instability score was 3.2 (SD 1.5) (3.6 in the surgical group and 3.0 in the nonoperative group, with no

statistical difference). We were not able to replicate Spector and colleagues’ evaluation of the size of the facet fragment because the fracture lines were basal, transverse, and coronal.³ In addition we did not find any significant differences in Subaxial Injury Classification (SLIC) and Severity score⁴ between the two groups (see Table 2 in our article).

Dr. Dahdaleh and his colleagues opined that “the decision to operate in patients initially treated with brace therapy was driven by radiographic evidence of translation or kyphosis, which in the absence of pain, dislocation, or neurological deficit does not necessarily warrant surgical intervention.” While we understand their concern for unnecessary surgery, our view was that waiting for a neurological deficit or pronounced subluxation to develop might leave the patient with permanent nerve root or spinal cord injury and protracted pain and unhappiness.¹

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References

1. Dvorak MF, Fisher CG, Aarabi B, Harris MB, Hurlbert RJ, Rampersaud YR, et al: Clinical outcomes of 90 isolated unilateral facet fractures, subluxations, and dislocations treated surgically and nonoperatively. **Spine (Phila Pa 1976)** 32:3007–3013, 2007
2. Halliday AL, Henderson BR, Hart BL, Benzel EC: The management of unilateral lateral mass/facet fractures of the subaxial cervical spine: the use of magnetic resonance imaging to predict instability. **Spine (Phila Pa 1976)** 22:2614–2621, 1997
3. Spector LR, Kim DH, Affonso J, Albert TJ, Hilibrand AS, Vaccaro AR: Use of computed tomography to predict failure of nonoperative treatment of unilateral facet fractures of the cervical spine. **Spine (Phila Pa 1976)** 31:2827–2835, 2006
4. Vaccaro AR, Hurlbert RJ, Patel AA, Fisher C, Dvorak M, Lehman RA Jr, et al: The subaxial cervical spine injury classification system: a novel approach to recognize the importance of morphology, neurology, and integrity of the disco-ligamentous complex. **Spine (Phila Pa 1976)** 32:2365–2374, 2007

Please include this information when citing this paper: published online May 2, 2014; DOI: 10.3171/2014.3.SPINE14223.

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Axis fractures

TO THE EDITOR: I found the article by Shinbo et al.⁸ very interesting (Shinbo J, Sameda H, Ikenoue S, et al: Simultaneous anterior and posterior screw fixations confined to the axis for stabilization of a 3-part fracture of the axis [odontoid, dens, and hangman fractures]. Report of 2 cases. **J Neurosurg Spine** 20:265–269, March 2014). The authors described 2 cases of multiple fractures confined to the axis (odontoid Type II fracture associated with a Type IA traumatic spondylolisthesis without involvement of C2–3 disc space). The authors treated both patients with anterior odontoid screws and additional posterior bilateral C-2 pedicle screws, with favorable outcomes and good osseosynthesis. Shinbo et al. also reported that there is no strong consensus for the treatment in such cases, justifying a combined approach.

Although the literature supports surgery for odontoid Type II fractures in patients older than 50 years of age,⁴ fractures of the posterior elements of the axis classified according to the Levine-Edwards criteria as Type IA (without bone translation, significant displacement, or angular deformity) were successfully treated conservatively in most of the reported series of axis fractures^{1,3,5,6} found in literature. Anterior odontoid screw fixation is an accepted treatment for odontoid Type II fractures.⁷ However, I believe that an additional posterior approach with posterior pedicle screw insertion was not necessary for bone healing in Type I Levine-Edwards fractures, even if associated with odontoid fractures. Axis pedicle screws can lead to rare but catastrophic vertebral artery injury, and are inadvisable in up to 20% of cases due to pedicle size and/or abnormal vertebral artery anatomy.² In both presented cases, the posterior approach could be reserved for failure in bone healing, avoiding unnecessary risks and the additional costs of an early combined approach. I thank the authors for their interesting and well-illustrated article.

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Disclosure

The author reports no conflict of interest.

References

- Greene KA, Dickman CA, Marciano FF, Drabier JB, Hadley MN, Sonntag VKH: Acute axis fractures. Analysis of management and outcome in 340 consecutive cases. *Spine (Phila Pa 1976)* **22**:1843–1852, 1997
- Joaquim AF, Ghizoni E, Anderle DV, Oliveira Ed, Tedeschi H: Axis instrumentation: surgical results. *Arq Neuropsiquiatr* **70**:857–863, 2012
- Joaquim AF, Patel AA: Craniocervical traumatic injuries: evaluation and surgical decision making. *Global Spine J* **1**:37–42, 2011
- Lennarson PJ, Mostafavi H, Traynelis VC, Walters BC: Management of type II dens fractures: a case-control study. *Spine (Phila Pa 1976)* **25**:1234–1237, 2000
- Levine AM, Edwards CC: The management of traumatic spondylolisthesis of the axis. *J Bone Joint Surg Am* **67**:217–226, 1985
- Mestdagh H, Letendart J, Sensey JJ, Duquenois A: [Treatment of fractures of the posterior axial arch. Results of 41 cases.] *Rev Chir Orthop Reparatrice Appar Mot* **70**:21–28, 1984 (Fr)
- Ryken TC, Hadley MN, Aarabi B, Dhall SS, Gelb DE, Hurlbert RJ, et al: Management of isolated fractures of the axis in adults. *Neurosurgery* **72 Suppl 2**:132–150, 2013
- Shinbo J, Sameda H, Ikenoue S, Takase K, Yamaguchi T, Hashimoto E, et al: Simultaneous anterior and posterior screw fixations confined to the axis for stabilization of a 3-part fracture of the axis (odontoid, dens, and hangman fractures). Report of 2 cases. *J Neurosurg Spine* **20**:265–269, 2014

RESPONSE: We greatly appreciate Dr. Joaquim's interest in our article. As he pointed out, conservative treatment must be the first choice if the fracture is simple traumatic spondylolisthesis of the axis categorized as a

Levine-Edwards Type IA fracture, and the choice of the odontoid screw fixation is widely accepted for simple odontoid Type II fractures.

However, it might have been unsatisfactory for the patients with the combined fracture presented in our paper if they had undergone only the anterior odontoid fixation and we had not performed the posterior pedicle screw fixation, and if they had been required to undergo the relatively longer period of external fixation that had been supposed to be necessary for the conservative treatment of the simple axis fracture classified as Levine-Edwards Type IA.

In this regard, circumferential operative therapy could result in shortening of the term of external fixation, achieving high primary stability and providing good outcomes in that the range of motion of the cervical spine was preserved. Moreover, recently, more advanced techniques and devices have made pedicle screw insertion easier and safer, although we must pay meticulous attention not to injure the vertebral artery. Therefore, we opted to perform the circumferential operation and were able to achieve the good outcome.

We again thank Dr. Joaquim for making his insightful remarks and hope that our report can help readers arrange their strategy for the combined fracture of axis.

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Please include this information when citing this paper: published online May 2, 2014; DOI: 10.3171/2014.1.SPINE1443.
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Asymmetric patterns of CSF flow in the spinal subarachnoid cisterns in patients with syringomyelia: clinical implications

TO THE EDITOR: We read with great interest the paper of Heiss et al.⁴ (Heiss JD, Snyder K, Peterson MW, et al: Pathophysiology of primary spinal syringomyelia. Clinical article. *J Neurosurg Spine* **17**:367–380, November 2012). In this study, the authors present further empirical evidence from subarachnoid pulse pressure measurements for the theory that the pathophysiology of syringomyelia development and progression involves a block in the normal CSF flow through the spinal subarachnoid space, which leads to increased spinal subarachnoid pulse pressures above the interruption, ultimately producing a pressure differential across the obstructed segment. The results of such study confirm the authors' initial findings of reduced CSF compliance, as well as increased cervical subarachnoid pressure and pulse pressure, obtained in a previous study using phase-contrast cine MRI and CSF pressure measurements.³

In a recently proposed classification of syringomyelia based on postmortem histological analyses and MRI findings in 927 patients, Milhorat divided such lesions into 4 basic groups: communicating syringomyelia, noncommunicating syringomyelia, atrophic cavitations (the

so-called syringomyelia ex vacuo), and neoplastic cavitations.⁹ Similar to the findings of Heiss et al., in the second group (those patients in whom the syrinx cavity did not communicate with the fourth ventricle), the main etiological factor implicated in the pathophysiology of syrinx development and progression was an increase in the arterial pulse wave velocity in the spinal subarachnoid space that was sufficient to force CSF into the central canal of the spinal cord through anatomically contiguous perivascular and interstitial spaces.^{10,12}

Interestingly, although several previous imaging studies have confirmed the presence of significant differences in CSF circulation both around the foramen magnum and adjacent to the syrinx in patients with Chiari I malformation and syringomyelia,^{11,13} only a few recent studies have more consistently mapped the pattern of such changes in the CSF flow, with special attention to a possible antero-posterior asymmetry between the subarachnoid spinal cisterns.^{2,5}

The existence of possible differences between the patterns of CSF flow in the anterior and posterior spinal subarachnoid cisterns acquires further importance when taking into account the much more rare, but also intriguing, clinical situation in which, in the absence of congenital craniocervical or posterior fossa abnormalities (such as a Chiari I malformation), syringomyelia develops concomitantly with (and adjacent to) spinal levels of cervical spondylosis. In our clinical experience we have more than once come across a syrinx cavity in the cervical spinal cord that developed after obliteration of the anterior spinal subarachnoid cistern due to intervertebral disc pathology, even though the total diameter of the cervical canal was preserved (that is, no major cervical canal stenosis). On such occasions, although the degree of compression of the spinal cord was not impressive (taking into account the presence of CSF in the posterior spinal cisterns), the patients presented with progressive clinical symptoms of cervical myelopathy. Interestingly, after such patients were submitted to surgical intervention through anterior cervical discectomy and fusion (ACDF), they exhibited a significant clinical improvement as well as further reduction in the dimensions of the syrinx (Fig. 1). According to the follow-up postoperative images obtained in these patients, although the surgical procedure does not seem to have affected the total anteroposterior diameter of the cervical spinal canal (Fig. 2), a posterior shift of the spinal cord was verified. In fact, the observed increase in the diameter of the anterior spinal subarachnoid cistern and the concomitant clinical improvement seem to suggest that the decrease in the size of the syrinx may be related to some alterations in the CSF flow dynamics, with restoration of the normal flow through the anterior spinal subarachnoid cistern, ultimately leading to the observed clinical improvement (Fig. 2). Such experience has been confirmed by other similar reports in the literature.^{1,14}

Although it must be recognized that such anecdotal reports (there are fewer than 10 reported cases of syringomyelia associated with cervical spondylosis in the literature)^{7,8} provide weak evidence in terms of supporting any therapeutic recommendations in such situations (such as surgical decompression for restoration of the anterior

Fig. 1. **Left:** Sagittal T2-weighted MR image of the cervical spine demonstrating anterior disc bulging at the C4–5 and C5–6 levels and associated syringomyelia extending up to the medulla (black arrow) in a patient presenting with progressive tetraparesis and other clear signs of cervical myelopathy such as hyperreflexia, gait imbalance, and posterior column sensory disturbances. Note the effacement of the anterior spinal subarachnoid cistern (measuring 0.59 mm at C4–5 and 0.79 mm at C5–6). **Right:** Sagittal T2-weighted MRI of the cervical spine of the same patient 15 months after a C4–6 ACDF. The patient experienced significant clinical improvement of the myelopathic symptoms. Note the postoperative reduction in the size and the upward extension of the syrinx cavity (white arrow), as well as an increase in the diameter of the anterior spinal subarachnoid cistern (3 mm at C4–5 and 3.27 mm at C5–6). Interesting to note, according to current paradigms, most surgeons would not have operated on such patients because the posterior spinal cistern was open preoperatively.

spinal cistern even in the absence of significant spinal canal stenosis), in our opinion, they present an interesting scenario for a more thoughtful evaluation of the etiological factors that may be involved in the pathophysiology of syringomyelia, with special reference to the possible presence of a meaningful asymmetry between the CSF flow

Fig. 2. Axial T2-weighted MRI cuts through the level of the most significant anterior compression (C4–5) demonstrating that, although no major change was observed in the total diameter of the spinal canal after decompression, a posterior shift of the spinal cord occurred postoperatively, with a consequent increase in the diameter of the anterior subarachnoid spinal cistern and a simultaneous decrease in the posterior subarachnoid spinal cistern, possibly leading to a new pattern of CSF dynamics, which was responsible for the decrease in the dimensions of the syrinx and the observed clinical improvement. Again, note that the clinical improvement and the decrease in the dimensions of the syrinx correlated with the size of the anterior spinal subarachnoid cistern (which significantly increased) and not with the overall dimensions of the spinal canal at that level (which remained roughly the same).

in the anterior and posterior spinal subarachnoid cisterns and its significance in terms of subarachnoid pulse pressure waves and syrinx development.

Interestingly, a previous study using computational fluid dynamics simulations in order to reproduce the results of phase-contrast cardiac-gated cine MRI in both normal subjects and in patients with Chiari I malformation have demonstrated that the shape and size of the spinal cord (and therefore the final dimensions of the adjacent subarachnoid space) in the simulated model significantly affected the final CSF flow patterns.⁵ According to the results of this study, the models that best reproduced the flow pattern observed on phase-contrast cine MRI were those in which there was an inhomogeneous pattern of flow jets in the subarachnoid spinal space, with significant differences between the anterior and posterior spinal cisterns. By varying the diameters of the spinal cord, such simulations demonstrated that there was a marked increase in CSF velocities when the diameters of the subarachnoid spinal cisterns were reduced to those below a certain threshold. Moreover, such asymmetry of flow between the anterior and posterior subarachnoid spinal cisterns was markedly increased in patients with Chiari I malformation and syringomyelia. In such a group, for example, during the peak diastolic flow, velocities with opposite values (representing CSF flow in opposite directions) were noted when comparing the CSF dynamics in the anterior and posterior subarachnoid spinal cisterns.

Another study using a new form of phase-contrast MR-based imaging, called 4D MR phase-contrast flow imaging, demonstrated that, while a homogeneous distribution of flow in the anterior and anterolateral subarachnoid spaces at the craniocervical junction was observed in all studied healthy volunteers, patients with Chiari I malformation had heterogeneous flow not only at the craniocervical junction but also in the pre-syrinx region.²

Consistent with previous studies using standard axial imaging,⁶ the authors of this study demonstrated that, instead of a complete CSF flow blockage, the observed narrowing in the anterior subarachnoid space caused by the craniocervical abnormality led to a diversion of the CSF flow to each side of the anterolateral subarachnoid space, resulting in uni- or bilateral jets with accelerated flow velocities (Fig. 3). Although such remarkable differences in CSF flow were observed in the anterior subarachnoid space, the presence of a syrinx did not seem to cause any major changes in the CSF flow in the posterior subarachnoid cisterns.

These studies, as well as our clinical experience, provide further support for a special role of the anterior subarachnoid spinal cistern in the development and progression of syringomyelia. We congratulate Heiss et al. for the valuable contribution to the understanding of the pathophysiology of syringomyelia that their study involving direct measurements of mean and pulse pressures in the subarachnoid space represents. Nevertheless, taking into account the possibility of meaningful differences between the CSF flow dynamics and intrathecal CSF pressures in the anterior and posterior subarachnoid spinal cisterns, some questions regarding the methodology of this study (as well as the current standard surgical management of syringomyelia) remain unanswered. Some of these questions are: Would the measured cervical subarachnoid mean and pulse pressures have presented different values if the authors had obtained them from the anterior subarachnoid cistern instead of from the posterior subarachnoid cistern? How would it be technically possible to obtain separate measurements of intrathecal CSF mean and pulse pressures in the anterior and posterior cervical subarachnoid cisterns in patients with syringomyelia in future studies? If several previous experimental studies seem to reveal that obstruction of the anterior CSF subarachnoid cistern

Fig. 3. Coronal 4D phase-contrast CSF flow image (A) and axial 2D phase-contrast image (B) of a patient with Chiari Type I malformation demonstrating left-sided unilateral flow jet in the anterolateral subarachnoid cistern. Paracoronal 4D flow image (C) and axial 2D phase-contrast image (D) of a patient with Chiari Type I malformation demonstrating synchronous bidirectional flow. Notice the bright area in the anterior subarachnoid space between the bilateral dark jets on the axial 2D image (D) indicating synchronous bidirectional flow consistent with right-sided flow vortex formation on the 4D phase-contrast CSF flow image (flow direction indicated by the black arrow). Reprinted with permission from Bunck et al.: Magnetic resonance 4D flow analysis of cerebrospinal fluid dynamics in Chiari I malformation with and without syringomyelia. *Eur Radiol* 22(9):1860–1870, 2012.

has a more prominent role in the pathophysiology of syringomyelia, should anterior approaches for decompression of the cervical spine (which are much simpler than posterior fossa decompression) become an alternative for initial surgical management of patients with Chiari Type I malformation, syringomyelia, and adjacent intervertebral disc pathology (especially in those with syrinx-related myelopathic symptoms) as an attempt to restore normal CSF flow, obtain reduction in syrinx dimensions, and improve clinical symptoms?

In summary, we emphasize that basic science studies, such as the one from Heiss et al., have a crucial role in refining our understanding about CSF flow dynamics in the setting of syringomyelia. Such investigations of important variables related to subarachnoid CSF flow and intrathecal pressure represent a necessary means of paving the way for future guidelines regarding the clinical management of patients with syringomyelia, not only in the setting of craniocervical abnormalities (such as Chiari I malformation), but also in those more rare cases of a syrinx associated with other etiological factors, such as cervical degenerative spondylosis.

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Disclosure

The author reports no conflict of interest.

References

1. Ball JR, Little NS: Chiari malformation, cervical disc prolapse and syringomyelia—always think twice. **J Clin Neurosci** 15: 474–476, 2008
2. Bunck AC, Kroeger JR, Juettner A, Brentrup A, Fiedler B, Crelier GR, et al: Magnetic resonance 4D flow analysis of cerebrospinal fluid dynamics in Chiari I malformation with and without syringomyelia. **Eur Radiol** 22:1860–1870, 2012
3. Heiss JD, Patronas N, DeVroom HL, Shawker T, Ennis R, Kammerer W, et al: Elucidating the pathophysiology of syringomyelia. **J Neurosurg** 91:553–562, 1999
4. Heiss JD, Snyder K, Peterson MM, Patronas NJ, Butman JA, Smith RK, et al: Pathophysiology of primary spinal syringomyelia. Clinical article. **J Neurosurg Spine** 17:367–380, 2012
5. Hentschel S, Mardal KA, Løvgren AE, Linge S, Haughton V: Characterization of cyclic CSF flow in the foramen magnum and upper cervical spinal canal with MR flow imaging and computational fluid dynamics. **AJNR Am J Neuroradiol** 31: 997–1002, 2010
6. Iskandar BJ, Quigley M, Haughton VM: Foramen magnum cerebrospinal fluid flow characteristics in children with Chiari I malformation before and after craniocervical decompression. **J Neurosurg** 101:169–178, 2004
7. Kimura R, Park YS, Nakase H, Sakaki T: Syringomyelia caused by cervical spondylosis. **Acta Neurochir (Wien)** 146:175–178, 2004
8. Lucci B, Reverberi S, Greco G: Syringomyelia and syringomyelic syndrome by cervical spondylosis. Report on three cases presenting with neurogenic osteoarthropathies. **J Neurosurg Sci** 25:169–172, 1981
9. Milhorat TH: Classification of syringomyelia. **Neurosurg Focus** 8(3):E1, 2000
10. Milhorat TH, Nobandegani F, Miller JI, Rao C: Noncommunicating syringomyelia following occlusion of central canal in rats. Experimental model and histological findings. **J Neurosurg** 78:274–279, 1993
11. Sakas DE, Korfiatis SI, Wayte SC, Beale DJ, Papapetrou KP, Stranjalis GS, et al: Chiari malformation: CSF flow dynamics in the craniocervical junction and syrinx. **Acta Neurochir (Wien)** 147:1223–1233, 2005
12. Stoodley MA, Gutschmidt B, Jones NR: Cerebrospinal fluid flow in an animal model of noncommunicating syringomyelia. **Neurosurgery** 44:1065–1076, 1999
13. Struck AF, Haughton VM: Idiopathic syringomyelia: phase-contrast MR of cerebrospinal fluid flow dynamics at level of foramen magnum. **Radiology** 253:184–190, 2009
14. Yaman ME, Eylon A, Ayberk G: Resolution of isolated syringomyelia after treatment of cervical disc herniation: association or coincidence? **Bratisl Lek Listy** 113:500–502, 2012

RESPONSE: We thank Dr. Mattei for his comments on our paper and his thoughts about syringomyelia associated with cervical spondylosis. We agree with him that syringomyelia caused by cervical spondylosis is quite rare. In fact, the MR images presented by him do not show syringomyelia, before or after surgery. Only a very small portion of the cervical portion of the spinal cord, a short segment behind the body of C-4, has a signal intensity similar to the signal intensity of the subarachnoid CSF shown in the same image. The remainder of the abnormal signal affecting the cervical segments of the spinal cord is consistent with edematous tissue or tissue damaged as a result of cervical myelopathy. Although these MRI changes may represent the edematous changes that occur with the “presyrinx” state, syringomyelia so rarely occurs with cervical degenerative disease that the changes seen cannot be considered to indicate the pending development of syringomyelia.

We agree that, in the rare instances in which it occurs, this type of syringomyelia usually occurs “after obliteration of the anterior spinal subarachnoid cisterns due to intervertebral disc pathology” and that “anterior cervical discectomy and fusion” often results in “reduction in the dimensions of the syrinx.” We do not agree with his statement, “According to the follow-up postoperative images obtained in these patients, although the surgical procedure does not seem to have affected the total anteroposterior diameter of the cervical spinal canal, a posterior shift of the spinal cord was verified.” To evaluate if the anteroposterior diameter of the cervical spinal canal was affected by surgery, we drew a dotted line indicating the anterior extent of the spinal canal before surgery on the postoperative image (Figure 1, response). This image and the measurements on Dr. Mattei’s Figure 1 clearly show that the anteroposterior diameter of the cervical spinal canal was expanded by his ACDF. The second part of his statement, “a posterior shift of the spinal cord was verified” is not confirmed in Figure 1, in which the width of the subarachnoid space dorsal to the spinal cord in the midcervical spine is unchanged from before surgery. An increase in the width of the subarachnoid space anterior to the spinal cord after surgery is consistent with removal of the C4–5 and C5–6 intervertebral discs and expansion of the spinal canal (Fig. 1, here). We agree with him that his surgical procedure likely improved flow through the anterior spinal subarachnoid space after surgery.

Dr. Mattei suggests that “flow jets in the subarachnoid spinal space” have a role in syringomyelia pathogenesis. In his Figure 3, the “flow jets” have a velocity of 10–15 cm/sec but are in the region of the vertebral arteries at the foramen magnum, suggesting that the jets may be due to arterial rather than CSF movement. In contrast to these findings at the foramen magnum, within the cervical subarachnoid space CSF movement is fairly regular (Fig. 3, Mattei). The relevance of the phase-contrast imaging shown in Fig. 3 of Dr. Mattei’s letter to his own case is uncertain, because he does not provide phase-contrast imaging studies for his patient or for other patients with syringomyelia associated with cervical spondylosis. Exuberance for the concept of flow jets must be tempered by the fact that the spinal canal is not an open channel but a partially compliant space, resulting in oscillatory CSF motion at the foramen magnum and in the cervical spinal canal during the cardiac cycle that is limited to 0.4–0.6 ml.¹

Dr. Mattei’s letter is a practical reminder that syringomyelia is usually associated with obstruction of normal CSF movement at the foramen magnum or within the spinal canal. Surgical procedures that effectively restore normal CSF flow also resolve syringomyelia.^{2–4}

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References

1. Bunck AC, Kroeger JR, Juettner A, Brentrup A, Fiedler B, Crelier GR, et al: Magnetic resonance 4D flow analysis of cerebrospinal fluid dynamics in Chiari I malformation with and without syringomyelia. **Eur Radiol** **22**:1860–1870, 2012
2. Heiss JD, Patronas N, DeVroom HL, Shawker T, Ennis R, Kammerer W, et al: Elucidating the pathophysiology of syringomyelia. **J Neurosurg** **91**:553–562, 1999
3. Heiss JD, Suffredini G, Smith R, DeVroom HL, Patronas NJ, Butman JA, et al: Pathophysiology of persistent syringomyelia after decompressive craniocervical surgery. Clinical article. **J Neurosurg Spine** **13**:729–742, 2010
4. Oldfield EH, Muraszko K, Shawker TH, Patronas NJ: Pathophysiology of syringomyelia associated with Chiari I malformation of the cerebellar tonsils. Implications for diagnosis and treatment. **J Neurosurg** **80**:3–15, 1994

Please include this information when citing this paper: published online May 23, 2014; DOI: 10.3171/2012.11.SPINE121038.
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