Letters to the Editor

NEUROSURGICAL FORUM

Atlantoaxial facet dislocation and Chiari malformation

TO THE EDITOR: We read with great interest the article published by Dr. Goel² (Goel A: Is atlantoaxial instability the cause of Chiari malformation? Outcome analysis of 65 patients treated by atlantoaxial fixation. *J Neurosurg Spine* 22:116–127, 2015, February 2015). We noticed that Goel classified atlantoaxial facet dislocation as Type I (anterior dislocation), Type II (posterior dislocation), and Type III (central dislocation) and that Goel stated, “Such a form of dislocation has not been reported in the literature.” We found that Goel’s classification of 3 types of facet dislocation was similar to ours, which had been published before.⁸ We take objection to Goel stating that this “has not been reported” and suggest that he should have reviewed the literature thoroughly so as to place the material in proper perspective for future readers. We also noticed that Goel believes “the pathogenesis of CM [Chiari malformation] with or without associated basilar invagination and/or syringomyelia is primarily related to atlantoaxial instability.” We have different opinions and wish to express our ideas to the authors and readers of *JNS: Spine*.

We first described the 3-dimensional configuration and the morphological changes of lateral atlantoaxial articulations (facet joints) in 63 patients with basilar invagination and atlas occipitalization and in 20 controls.⁸ We classified the anomalous atlantoaxial facet joints into 4 clinical types (Fig. 1): Type I, characterized by slight anteversion of facet joint; Type II, characterized by partial anterior olisthy of the superior facets over the inferior facets; Type

III, characterized by separation or complete olisthy of the facets; and Type IV, characterized by retroversion of the facet joints.⁸ In our series of 63 cases with basilar invagination and atlas occipitalization we found that 49 patients with anteversion of facet joints (Types I, II, and III) had atlantoaxial dislocation (AAD) and 14 patients with retroversion of facet joints (Type IV) had no AAD. We found that the anterior sagittal inclination of the facet had a close relationship with the severity of AAD and basilar invagination, and thus we hypothesized that instability at the C1–2 junction in congenital anomalies of the craniovertebral junction was likely a direct result of facet joint anteversion and it is aggravated by the increasing obliquity of anteversion of the facet.⁸ In the 14 patients with Type IV findings, the retroversion of the facet joints may have caused the C-1 facet to slide posteriorly over the C-2 facet resulting in partial olisthy of the facets (Fig. 2). However, there was no AAD in Type IV patients; a possible reason could be that the retroversion of the facet joints made the atlantodental distance shorter.

Although the clinical manifestations improved with the treatment of atlantoaxial fixation in Goel’s reported 65 cases, there was no direct evidence that atlantoaxial instability was the main reason. The pathogenesis of Chiari malformation with or without associated basilar invagination and/or syringomyelia is very complex. Decreased volume of the posterior cranial fossa is believed to be the main reason for simple Chiari malformation and/or syringomyelia without ventral compression (usually no AAD/basilar invagination), and foramen magnum decompression with or without duraplasty is the main surgical treatment.^{1,5} Nevertheless, the pathogenesis of Chiari malformation and/or syringomyelia with ventral compression

FIG. 1. Clinical typing of the anomalous C1–2 facet joints in basilar invagination together with atlas occipitalization compared with the normal structure. Reproduced with permission from Yin et al. Three-dimensional configuration and morphometric analysis of the lateral atlantoaxial articulation in congenital anomaly with occipitalization of the atlas. *Spine (Phila Pa 1976)* 37(3):E170–173, 2012. Figure is available in color online only.

FIG. 2. Images obtained in a patient with the retroversion of facet joint (Type IV). **A:** Midsagittal CT image showing platybasia, basilar invagination, scoliosis, and atlas occipitalization, but without atlantoaxial dislocation. **B:** Parasagittal CT image showing the posterior olivary of the facet of C1–2. **C and D:** Parasagittal CT and MR images showed abnormality of the clivus and Chiari malformation. Note the compression of the brainstem and the abnormal angle of the pons-medulla junction.

(usually caused by AAD, basilar invagination, or platybasia) remains controversial,^{1,4,5} and recently Menezes⁴ pointed out that abnormal CSF circulation may be the main reason. In these kinds of patients, if the ventral compression could be relieved, the concomitant Chiari malformation and/or syringomyelia would regress.⁴ Our clinical experience with 30 years of treatment for more than 2000 cases of craniovertebral junction abnormalities also supported this hypothesis (Fig. 3). We believe that the relief of ventral compression and recovery of cerebrospinal fluid circulation are the main reasons for regression of Chiari malformation and/or syringomyelia. As for the treatment options, various procedures exist. Goel preferred the atlantoaxial fixation with or without the use of spacers.² We used the same procedure of C1–2 fixation after atlantoaxial facet joint release to reduce basilar invagination with AAD in Type I and Type II facet joint anomalies.^{3,6,7} Furthermore, some patients still need transoral/transnasal decompression followed by posterior instrumentation, because the posterior approach alone cannot solve the ventral compression in platybasia (Type IV, Fig. 2) or some truly irreducible AAD (mainly cases of Type III facet joint anomalies).⁸

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FIG. 3. Images obtained in a patient with irreducible basilar invagination and atlantoaxial dislocation and atlas occipitalization. **A:** Preoperative MRI showed Chiari malformation and syringomyelia. **B:** Postoperative MRI demonstrated the regression of herniated cerebellar tonsils and disappeared syrinx.

DISCLOSURE

The authors report no conflict of interest.

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Response

Our description in the years 2004 and 2005 was that in cases with basilar invagination and in the AAD previously called “irreducible” or “fixed,” the dislocation is not fixed, but is rather not only mobile but abnormally mobile and can be manually reduced. This has changed the treatment paradigm for these entities, to the extent that transoral (or transnasal) surgical decompression is rapidly slipping into the domain of history.^{1–11} Subsequently, several authors have attempted reduction of basilar invagination in a variety of ways, and surgery via a posterior approach alone has become an accepted form of surgical treatment for basilar invagination. Yin and colleagues have also made

minor modifications to our established technique and published it recently.¹³

We simulated the alignment of facets of atlas and axis in basilar invagination with lumbosacral listhesis.¹¹ We have also used the term “spondyloptosis” in cases with severe basilar invagination, wherein the facets of the atlas were positioned anterior to the facets of the axis.¹² Yin and Yu have used the terms anteversion, anteriorolisthy, and retroversion in their article instead of the more commonly used term “listhesis” described by us, but they have not credited this terminology or the concept.¹¹ Moreover the term “complete olisthy” used by them is nothing but “spondyloptosis,” the term used earlier by us.¹² We have also discussed the issue of progression of listhesis over a period of time³ as a cause of basilar invagination, a feature that has been discussed by Yin and Yu without referring to our work.³

We had earlier classified basilar invagination into 2 groups on the basis of presence or absence of Chiari malformation.⁵ Subsequently, we classified basilar invagination on the basis of presence or absence of instability.⁴ We have now reclassified the “instability” in basilar invagination on the basis of the facet alignment of atlas and axis into 3 groups depending on their sagittal relationships.¹ Such a classification of atlantoaxial instability on the basis of facet alignment has not been described earlier. In their article, Yin et al.¹³ have discussed the alignment of facets in cases of basilar invagination in the presence of occipitalization of atlas. They have identified atlantoaxial instability on the well-described basis of atlantodental alignment and not on the basis of facet alignment. As per our classification, Type I facet alignment is where the facet of the atlas is dislocated anterior to the facet of the axis, or in other words, there is listhesis of the atlas facet over the axis facet. Type II facet alignment is where the facet of the atlas is dislocated posterior to the facet of the axis or where there is retrolisthesis of the facets. This type of alignment is similar to Type IV or retroversion of facets as discussed by Yin et al. However, Yin et al. have identified that in such cases there is no craniovertebral instability or AAD, the region is stable, and the alignment is a kind of “protective phenomenon,” as the odontoid process is placed closer to the anterior arch of atlas.¹³ We have proposed such a form of alignment as a major form of instability of the spinal segment that merits treatment. We have labeled Type III as axial or central instability, wherein there is no facet alignment, but the joint is unstable when assessed intraoperatively. We have labeled such instability as central or axial atlantoaxial facet dislocation. Our classification system is based on the fact that facet alignment is a kind of or a manifestation of instability by itself meriting surgical treatment and has not been hitherto discussed.

Essentially, we have identified atlantoaxial instability in all patients having basilar invagination and Chiari malformation and have stated that both of these entities when present together or in isolation should be treated by atlantoaxial fixation. With our increasing experience with treating Chiari malformation, we are convinced that instability is the cause of the problem and Chiari malformation is only Nature’s protective “airbag-like” effect. We take this opportunity to let Yin et al. and the readers know

that the clinical implications of basilar invagination-related “small posterior cranial fossa” and its effects in the pathogenesis of Chiari malformation were first described by us.⁵ However, based on our observations over a number of years, we now identify that the posterior fossa is not small in these cases and that the cerebellar hemisphere in general and a part of superior vermis are atrophic. In our article, we have mentioned that temporary improvement in clinical symptoms following foramen magnum decompression is akin to deflating a full airbag. In the long run, foramen magnum decompression surgery can be counterproductive.

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Does atlantoaxial dislocation really cause Chiari?

TO THE EDITOR: It has been always a delight for me to go through the enthralling articles published in your esteemed journal. Recently, I read an article by Goel² (Goel A: Is atlantoaxial instability the cause of Chiari malformation? Outcome analysis of 65 patients treated by atlantoaxial fixation. *J Neurosurg Spine* 22:116–127, February 2015). The author, Prof. Atul Goel, is a pioneer in the field of neurosurgery in many aspects, especially in the field of the craniovertebral junction, in which he has made immense contributions with his innovative and time-tested philosophical thoughts and works. In the present article, he has highlighted an aspect regarding Chiari malformation that was not considered before. His recent article regarding a new vision and approach for the management of Chiari malformation with or without basilar invagination has raised some questions for me, which I would be very glad to have answered, to develop insight in managing these patients better in the future in my practice.

First, if atlantoaxial dislocation (AAD) is the inciting factor for the development of Chiari malformation as a protective phenomenon, why don't all patients with AAD have Chiari malformation? In other articles Prof. Goel postulated that Chiari malformation might be Nature's air bag that prevents pinching of critical neural structures between bones, and syringomyelia is formed in an effort to neutralize cranial and spinal pressure and to support the bulk of the Chiari malformation.^{4,5} If the Chiari malformation is due to basilar invagination following AAD, CSF around the craniovertebral junction would be a more effective water cushion/bag than the tonsils as a natural protective phenomenon. When the tonsils descend down around the neural structures, they cause more pinching to these structures than CSF does, even though the tonsils are composed of soft tissue. Therefore, it seems logical that the AAD is not the cause of the Chiari malformation. Rather, a shallow posterior fossa causes the condition, given that it displaces the overcrowded but less vital neural structures, which are comparatively easy to displace. Moreover, Prof. Goel's study contains cases in which there was no clinical or radiological evidence of AAD preoperatively. In cases of Chiari malformation with established AAD, it is possible that, because of the instability, the tonsils are pulled down with an irregular seesaw-like movement at the atlantoaxial joint.

Second, why don't all patients with Chiari malformation have AAD? It is well accepted that a shallow posterior fossa is the most likely pathology in the development of Chiari malformation.^{1,3,5,6} Given that Chiari malformation is a congenital defect of the occipital enchondrium,⁶ AAD may be one component of the changes occurring due to the anomaly, among others like basilar invagination, short clivus, and platybasia that are found in a number of cases of Chiari malformation.

Third, a congenital shallow posterior fossa is a well-documented explanation for the development of Chiari malformation and is the most accepted pathogenic phenomenon.^{1,3,5,6} In that case how does fixation of AAD work here,

especially in the absence of demonstrable basilar invagination or other bony deformities that might play a role in reducing the posterior fossa volume other than the congenital one, where it does not increase the volume of the existing posterior fossa? And in cases of atlantoaxial instability with basilar invagination—in which it seems to be possible to reduce the posterior fossa volume—does the reduction of basilar invagination by fixation of AAD, really increase the volume in the posterior fossa to such a significant degree as to reduce the Chiari malformation? Rather, it seems that the patients improve because of the brainstem decompression due to reduction of the basilar invagination.

Even with my questions, I must admire Prof. Goel for his new philosophy, which will give us very good food for thought and might help in acquiring newer ideas for the management of patients with Chiari malformation. In the future, with more prospective studies and research, Prof. Goel's philosophy might prove commendable, as no one yet has thought of this possibility for this complex clinical situation.

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DISCLOSURE

The author reports no conflict of interest.

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Response

I thank Dr. Rahman for his interest in my article. Over the period of my analysis of the subject, it appears that Chiari malformation, syringomyelia, and basilar invagination, when present together or in isolation, have an origin related to atlantoaxial instability, and seem to be a continuum of the same mechanism.

The instability in the craniovertebral junction has traditionally been identified with an abnormal increase in the atlantodental interval and indentation of the odontoid process into the craniocervical cord. In the present and other related articles I have observed that there can be instability of the atlantoaxial region that is identified by atlantoaxial facetar malalignment or incompetence with or without alteration in the odontoid process-related parameters.^{1,2} In Type II atlantoaxial facetar instability, the facet of the atlas is dislocated posterior to the facet of the axis in the sagittal

profile image. The posterior dislocation may sometimes be unilateral. In Type III atlantoaxial facet instability, the facets are in alignment, and the instability is observed only during direct or manual facet handling during surgery. I have termed such instability central or axial instability. Neither Type II or III instability has been identified earlier in the literature. In both Type II and Type III atlantoaxial facet instability, the atlantodental interval remains unaffected, and there may be no odontoid process-related cord compression. This is probably why the clinical manifestations are late, subtle, and longstanding. Chiari malformation—like Group B basilar invagination and syringomyelia are more often associated with Type II and Type III atlantoaxial instability.^{4,5} Although the exact timing of the beginning of the instability can only be speculated, it could be during the late fetal life or in early infancy. During this period, organogenesis is still in progress and structural innovations by Nature are probably dictated by functional needs. Essentially longstanding nature, subtleness of instability, and, more importantly, the characteristics of dislocation determine the musculoskeletal and neural alterations. Acute-onset AAD or dislocations occurring late in life may not be associated with Chiari malformation or syringomyelia.

Short neck, torticollis, Klippel-Feil abnormalities, platybasia, basilar invagination, Chiari malformation, and syringomyelia all seem to be natural events that are secondary and protective.⁹ The type and extent of physical body alterations are probably determined by the nature and laterality of facet instability. The natural alterations can even be self-destructive, as in syringomyelia, but in the overall scenario, they work in the interest of the patient and in the endeavor of preservation of life.³ The very fact that patients with Chiari malformation improve with regard to their clinical symptoms immediately after surgery that involves only atlantoaxial fixation validates the fact.^{7,8} In my earlier series, like in the present one that focused on stabilization of the atlantoaxial joint, improvement was observed in patients in whom foramen magnum decompression had failed.^{2,4}

Basal suboccipital bone structural malformation resulting in a small posterior fossa that is unable to accommodate the volume of the cerebellum is an accepted theory of pathogenesis of Chiari malformation. My colleagues and I were among the first authors who correlated the small posterior fossa volume with Chiari malformation.⁵ Accordingly, posterior fossa or foramen magnum decompression is the standard form of surgical treatment in these cases. In my present study I realized that the posterior fossa volume is not small. Moreover, we (previously) identified atrophy of the superior vermis and the superior aspect of the cerebellum in the majority of cases.^{2,6} It is evident that the cerebellum is not tightly impacted into the posterior fossa. In the postoperative analysis of at least a sizable proportion of cases we observed superior migration of the tonsils and reduction in the size of the syrinx following surgery that involves only atlantoaxial fixation, without any bone or dural decompression. This experience suggests that atlantoaxial instability is the principal cause of Chiari malformation and may indeed be Nature's air bag and syringomyelia may be a protective formation that attempts to

neutralize cranial and spinal pressures and supports the Chiari malformation.

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Cervical disc arthroplasty: nonconstrained versus semiconstrained

TO THE EDITOR: We read with great interest the article by Dr. Heary and colleagues⁸ in the December issue of the *Journal of Neurosurgery: Spine* (Heary RF, Goldstein IM, Getto KM, et al: Solid radiographic fusion with a nonconstrained device 5 years after cervical arthroplasty. *J Neurosurg Spine* 21:951–955, December 2014). The authors reported the case of a patient who underwent cervical disc arthroplasty (CDA) using the DISCOVER artificial disc (DePuy Synthes Spine Inc.). Five years after the surgery, spontaneous fusion at the surgical level was identified, despite excellent clinical outcomes. The authors' article provided valuable information regarding an unexpected radiographic finding that occurred but did not affect the clinical outcome. However, there is a fundamental issue that needs to be clarified before claiming that this is the first identification of fusion around a nonconstrained cervical artificial disc.

In many reports the Bryan disc (Medtronic Sofamor Danek Inc.) has been considered as nonconstrained.^{1,3,6,7,10,12–15} Unlike the ball-and-socket articulating design of ProDisc-C (DePuy Synthes Spine Inc.) or the Prestige disc (Medtronic Sofamor Danek Inc.), the Bryan disc is composed of 2 pieces of titanium alloy shells and a polyurethane nucleus in between, surrounded by a polyurethane sheath with lubricant inside. This bi-articulating metal-on-polymer design naturally allows for translational and unconstrained motion through a physiological range of motion, including flexion-extension, lateral bending, and axial rotation. The fully variable instantaneous center of rotation (COR) of the Bryan disc has led to some concerns over extra loading of the facets and potential kyphosis.^{6,9,17} On the other hand, ProDisc-C and Prestige discs were often considered semiconstrained^{4,10} because of their predetermined COR of the ball-and-socket design and relatively lower allowance for translation.⁵

Spontaneous fusion or development of heterotopic ossification (HO) after CDA with a Bryan disc has been reported many times.^{2,11,16,18,19} It is reasonable to anticipate more incidences of this undesired bone formation after CDA, as the application of this technology increases and the follow-up is extended. Whether or not the HO is related to the biomechanical design remains elusive. Furthermore, the differences between the more- and less-constrained devices for CDA may be less substantial than those for large joint arthroplasty.

The treatment of cervical degenerative disc disease and spondylosis with CDA is a science with ongoing development. The authors are commended for sharing their precious clinical experience with the worldwide readers of the *Journal of Neurosurgery: Spine*. However, it would be more helpful if they provided a detailed explanation of the nomenclature of CDA devices and a comparison of the types of constraint had by Bryan and DISCOVER discs.

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DISCLOSURE

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Response

No response was received from the authors of the original article.

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