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 facetal 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 olisthesis of the superior facets over the inferior facets; Type III, characterized by separation or complete olisthesis of the facets; and Type IV, characterized by retroversion of the facet joints.9 In our series of 63 cases with basilar invagination and atlas occipitalization we found that 49 patients with anteverision 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 anteverision 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 olisthesis 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.13 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.](https://example.com/fig1)
(usually caused by AAD, basilar invagination, or platybasia) remains controversial,\(^1,4,5\) and recently Menezes\(^4\) 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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**DISCLOSURE**

The authors report no conflict of interest.

**References**


**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