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Editorial

The Role of Atlantoaxial Instability on Chiari, Ossification of the Posterior Longitudinal Ligament, Spondylosis and Stenosis

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I would like to congratulate Neurospine for a very interesting and highly informative set of articles. I would like to comment on the 2 articles[1,2] by 2 leading authorities in the field, Dr. Atul Goel and Dr. Sanjay Behari, that take somewhat different positions on the treatment of Chiari malformation. I have heard Dr. Goel's presentations regarding his novel treatment of this condition and therefore read this paper with great interest. Although I consider Dr. Goel a friend as well as a highly respected colleague, I must say that, based on these two papers, Dr. Behari's evidence and reasoning is more convincing to me. Dr. Goel's Figures 1-4 demonstrate four cases of craniovertebral junction abnormalities. All 4 cases have occipitalization of the atlas, as well as platybasia. I do not think that anyone would one disagree with his treatment of these four cases, as a C1–2 fusion essentially fuses the entire craniovertebral junction, since their atlas is already congenitally fused to the occiput. However, as Dr. Behari points out, >70% of patients with a pure Chiari I malformation have excellent improvement of their neurologic function with a simple decompression. If a nonfusion operation works as well as a fusion operation in greater than 70% of cases, the less invasive nonfusion operation is almost always the favored approach.

Dr. Goel also discusses several other interesting approaches to the cervical spine. His Fig. 5A-C shows images of the patient with a cord signal change at C3–4 and stenosis from C3 down to C6. Figure 5B shows that the atlas is slightly posterior to the axis on one cut of the magnetic resonance imaging (MRI). Dr. Goel calls this a type 2 atlantoaxial facetal instability. He treated the patient with a posterior atlantoaxial and C3–6 fusion. While I agree with fusing the patient from C3 down to C6, I would not have fused him at the atlantoaxial joint. An intact ring of the atlas cannot dislocate posteriorly because the dens acts as a hard stop. Therefore, there is a limit to how much the atlas can sublux posteriorly. Although the patient may have a few millimeters of posterior subluxation of C1 on C2 on one sagittal MRI cut, this can be produced in any normal patient by obtaining an MRI with the neck slightly rotated to the ipsilateral side. Even if a patient has such subluxation without rotation, I would want to see incontrovertible proof that the patient's symptoms are related to the subluxation before fusing what I consider to be the most important joint in the cervical spine when it comes to range of motion for activities of daily living. Likewise, for Fig. 6, I see no evidence that the patient needed an atlantoaxial fusion. There is overwhelming evidence in the literature that ossification of the posterior longitudinal ligament (OPLL) that does not involve the C1–2 region can be treated without a C1–2 fusion. It is also well-recognized that fusing a stenotic spine will often resolve any neurologic deficits without the need for a decompression. This is especially the case for mild neurologic deficits. Therefore, I agree that some

patients with OPLL can be treated exactly as Dr. Goel recommends, with the fusion and no decompression. However, I believe it would be dangerous to recommend this as the only treatment for all patients. First, many patients with OPLL are already auto-fused because of their OPLL and still develop progressive neurologic deficits. Second, some OPLL patients who are surgically decompressed posteriorly and solidly fused can nevertheless continue to grow their OPLL and have a recurrence of symptoms despite the solid fusion. Third, OPLL can become so severe that the anterior bone actually fuses to the lamina and encroaches into the spinal canal, leaving less than 1 to 2 mm of space available for the cord. Despite being solidly fused, these patients can be profoundly myelopathic or even quadriparetic. For patients with mild myelopathy, I would agree that a fusion can result in improvement of symptoms. However, in moderate to severe myelopathy, or for those with quadriparesis, I do not believe that a solidly fused OPLL patient with inadequate decompression will improve in any significant way. I fail to see how adding a posterior fusion without decompression can help such patients. Ultimately, only a study of patients with severe neurologic impairment treated without a decompression showing 100% resolution of their neurologic deficits can prove Dr. Goel's claim. However, if those patients do not fully recover their deficits and an MRI still shows cord compression, I do not believe that one can claim that the fusion alone was the definitive operation.

Finally, Dr. Goel posits that, "instability of the spinal segment/segments is the primary event and all the other spinal alterations like osteophyte formation, ligamentum flavum buckling, disc space reduction and ultimate reduction of spinal canal and/or neural foraminal dimensions are secondary alterations." I would agree that there are definitely cases where the facet degenerates first, resulting in its remodeling, with subluxation and disc degeneration occurring as a secondary phenomenon. Most commonly, this occurs from C2 to C4. However, every spine surgeon has seen patients with disc degeneration, whose facets are totally normal. If this were not the case, no patient would be eligible for a cervical disc arthroplasty, since it is contraindicated in patients with facet disease.

In conclusion, I read with great interest all of the papers in this edition of *Neurospine* and commend the authors on their excellent work. Drs. Goel and Behari take differing points of view on a common condition and Dr. Goel presents some novel ideas about various cervical conditions. Although I cannot agree with everything he says, it would be foolhardy not to pay close attention to a leading authority in the field with great expertise, experience and knowledge. Hopefully, future studies by independent researchers on these topics will ultimately prove him to be right or wrong.

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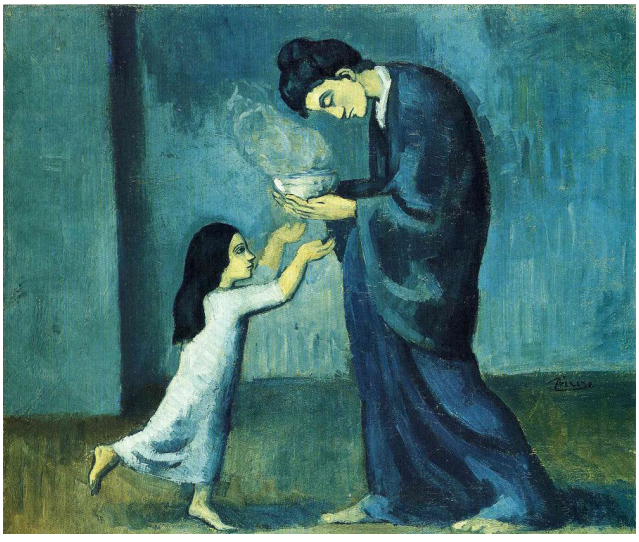

Title: La Soupe
Artist: Pablo Picasso
Year: 1902
Despite his initial success at the Vollard exhibition in 1901, Picasso's funds soon dried up. It was not unusual for the artist to go hungry during this period in Paris, but his own circumstances only heightened his awareness of their desperate poverty that frequently surrounded him. Back in Barcelona in 1902 Picasso produced the painting entitled *La Soupe*.
More information: https://www.pablopicasso.org/the-soup.jsp