


Central Atlantoaxial Instability: A New Clinical Entity?



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“Skepticism is the first step toward truth.”

- Denis Diderot (French philosopher and prominent figure during the Enlightenment)

Dr. Atul Goel, a highly accomplished surgeon and investigator in the treatment of cranio-vertebral disorders, provides a remarkably thought-provoking review of his concept of central atlantoaxial instability (CAAD) in this special issue of *Neurospine*.¹ Dr. Goel has previously published his classification of atlantoaxial “facetal” dislocation which includes 3 types.² Type 1, typically the most apparent form, is when the facet of the atlas is dislocated anterior to the facet of the axis and usually demonstrates alteration of the atlantodental interval and an odontoid process that is angled acutely posteriorly. Type 2 atlantoaxial facet instability is when the atlas is dislocated posterior to the facet of the axis. In type 3, although atlantoaxial facet instability is presumed to be present, dynamic images are unable to identify it. Type 3 instability is only suspected based on clinical assessment (e.g., older patients and those with significant neurological deficits) and imaging findings (e.g., retro-odontoid ‘pseudotumor,’ atlantoaxial facet and odontoid tip osteophytes, unusual cervical lordotic curvature, bone fusions, bifid arch of the atlas, and unusually or abnormally open atlantoaxial joints), and can only ultimately be confirmed based on direct intraoperative manual manipulation.

Type 1 atlantoaxial facet instability is often readily apparent and treatment for this condition is well established and relatively noncontroversial. However, types 2 and 3 atlantoaxial facet instability may have a normal atlantodental interval and lack neural or dural compression by the odontoid process. It is types 2 and 3, collectively labeled as forms of CAAD, that may not be readily apparent on clinical or imaging assessment that have generated skepticism with regard to their existence and their utilization to guide surgical treatment. Dr. Goel has detailed a host of musculoskeletal and neural alterations in response to the presence of CAAD that he attributes to “secondary natural processes that aim to protect the neural structures and life and delay or stall the neurological symptoms and deficits in the event of potential, subtle or manifest atlantoaxial instability, more often of central or axial variety.”^{1,3,4} The implication of Dr. Goel is that addressing the CAAD with stabilization (C1–2 fusion) can readily address and potentially reverse all of these musculoskeletal and neural alterations.

Among the conditions that Dr. Goel associates with CAAD are Chiari “formation” and syringomyelia, basilar invagination, multisegmental cervical spondylotic disease, torticollis, cervical kyphosis, dorsal kyphosis, ossified posterior longitudinal ligament, and Hiramaya disease. For each of these conditions, Dr. Goel’s assertion is that atlantoaxial stabilization is important for treating the underlying problem. Undoubtedly, the ascription of each one of these conditions to CAAD could stimulate a lengthy discussion. Behari and colleagues⁵ offer a counterpoint to the treatment of one of these conditions, Chiari type 1 malformation,

in this special issue of *Neurospine*. They argue, that for cases of pure Chiari type 1 malformation without atlantoaxial dislocation or basilar invagination and with completely symmetrical C1–2 joints, posterior fossa decompression with or without duroplasty is sufficient and that C1–2 stabilization is unnecessary. They note on review of the literature that using this approach results in approximately 70% of patients achieving neurologic improvement. However, the remaining 30% of patients that fail to improve with decompression alone suggest that optimal treatment may not always be this simple. Could it be, as Dr. Goel argues, that instability is the underlying pathology in Chiari type 1 malformation and that simple decompression, while potentially alleviating the symptoms in some patients, does not address the true underlying problem? Dr. Goel's previously published experience⁶ would seem to suggest that this could be the case, but even if it is, does this necessarily provide sufficient grounds for C1–2 stabilization (potentially even without any accompanying direct decompression) in all patients with type 1 Chiari malformation who warrant surgical treatment?

As a true pioneer who has frequently pushed the boundaries in the field of craniovertebral disorders, Dr. Goel's many contributions have not always been met with instant acceptance, but rather some have necessitated converting the skeptics.⁷ Importantly, Dr. Goel's review in this special issue is not simply a discussion of new concepts and proposals, instead it has been carefully built on the findings from his many relevant previously published, peer-reviewed studies. As he readily notes in the introduction of his review, other authors have yet to validate his observations. Indeed, this is the crux. Dr. Goel's claims of amazing clinical success with stabilization for CAAD (and they are quite impressive),⁸ coupled with his talents and track record, suggest that the concept of CAAD warrants further investiga-

tion. The current prevailing skepticism will no doubt stimulate others to test his concepts, and as further reports become available, the truths will emerge.

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Title: *Les Femmes d'Alger*

Artist: Pablo Picasso

Year: 1955

Pablo Picasso had been fascinated by Delacroix all his adult life, and by *Les femmes d'Alger* in particular. In addition to being an homage to Delacroix, Picasso conceived the series as an elegy to his friend and great artistic rival, Henri Matisse. Matisse had died in November 1954, five weeks before Picasso began the series. Matisse viewed Delacroix as his immediate forebear in terms of colour and Orientalist subject matter.

More information: <https://www.christies.com/features/Picasso-5829-1.aspx>

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