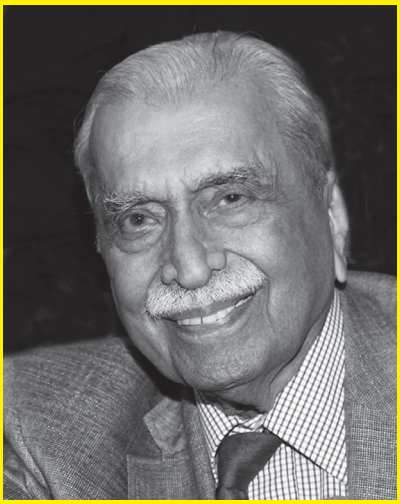
Classics in Indian Medicine



Noshir Hormusjee Wadia

Professor Noshir Hormusjee Wadia, MD, FRCP (London), DSc (Hons), FAMS, FASc, served as Registrar in Neurology to Lord Brain, National Hospital for Nervous Diseases (1952–56), and later at the London Hospital, where he was also Tutor at the Medical School. In 1957, he was appointed as Honorary Assistant Neurologist at the same place. On his return to India, he was appointed as Lecturer in Neurology (1961–68) and Honorary Professor (1968–82), Grant Medical College (GMC), Bombay (present Mumbai). On retirement, he was appointed 'Consultant for Life' at the Grant Medical College and Sir Jamshedji Jejeebhoy Group of Hospitals, Mumbai. Concurrently, he has since 1973 been Lifetime Director, Department of Neurology at Jaslok Hospital and Research Centre, Mumbai. He also received DSc (Honoris Causa, 1999) from Banaras Hindu University, Uttar Pradesh.

Professor Wadia has been a doyen of Clinical Neurology and teacher to undergraduates and postgraduates at the Grant Medical College and Sir J.J. Hospital, and Jaslok Hospital and Research Centre, Mumbai. He has trained over 100 neurologists. He has described several diseases not previously reported/prevalent in India, such as myelopathy of congenital atlanto–axial dislocation, Wilson's disease, tuberculous spinal meningitis, nutritional deficiency disorders, subacute myelo-optic neuropathy, higher prevalence of multiple sclerosis in Parsees and disseminated cysticercosis. He has also described two diseases not previously reported in the world: (i) a new form of heredo-familial spinocerebellar degeneration with slow eye movements, and (ii) the neurological complications associated with epidemic conjunctivitis (Enterovirus 70 disease). He has authored over 130 articles, and a book *Neurological Practice: An Indian perspective.*

Professor Wadia has also been credited with establishing one of the first Neurosciences department of India at the Sir J.J. Hospital, Mumbai. In 1973, he also helped in organizing the Department of Neurology at the Jaslok Hospital and Research Centre. He has served as Member (1981–2002), governing and institute bodies of the Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram and also as Chairman (1995–2002), Governing Body and President (Chancellor), in the same institute. He has also served on the Neurology Expert Committee (1963–66) and on the Scientific Advisory Board (1989–91) of the Indian Council of Medical Research (ICMR).

Dr Wadia has won several awards. He has been conferred the Certificate of Appreciation for Services to Neurology by the World Federation of Neurology (1993), First Rameshwardas Birla National Award for an Outstanding Practising Clinician in Modern Medicine (1999), Lifetime Achievement Award in Medical Excellence by Harvard Medical International and Wockhardt Limited for 'Pioneering and Immense Contributions in Specialty of Neurology', S.S. Bhatnagar Medal for Excellence in General Science by the Indian National Science Academy (INSA, 2003), Dhanvantari Award by Dhanvantari Medical Foundation for Outstanding Contribution in Medicine (2003), and Shree Dhanwantari Prize by INSA (2006). He was elected Fellow of the Indian Academy of Medical Sciences (1972), Indian Academy of Sciences, Bangalore (1983), and Royal College of Physicians, London (1970). He has also been an Honorary Member of the Sociedad Neurologica, Argentina (1961), Sociedad Chilena de Neurologica (1961), American Neurological Association (1977), Association of British Neurologists, UK (Foreign Member) (1979); and President, Neurological Society of India (1963–64) (*http://www.insaindia.org/detail.php?id=N86-0952*).

MYELOPATHY COMPLICATING CONGENITAL ATLANTO–AXIAL DISLOCATION*
(A Study of 28 Cases)

BY

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Atlanto–axial dislocation: The impact

After the initial description of congenital atlanto–axial dislocation by List in 1941[1] and by MacRae[2] as well as McCrae and Barnum in 1953,[3] attention was drawn to this entity in India by the articles written by Wadia in 1960,[4] Barucha and Dastur in 1964,[5] Srinivasan *et al.* in 1967[6] and Singh *et al.* in 1969,[7] where its neurological manifestations were briefly described. In 1965, Dastur, Wadia, Desai and Sinh presented a pilot report on the clinical correlation, pathology and pathogenesis of this entity and stated that sudden haematomyelia occurs following posterior decompression in patients with medullospinal compressison due to atlanto–axial dislocation.[8] In this scenario of developing interest towards clinical manifestations and treatment of congenital atlanto–axial dislocation, emerged this seminal paper by Professor Wadia. This article was a landmark paper due to several features.

1. It was then the largest published series of systematically studied patients of congenital atlanto–axial dislocation. In 28 patients (and in 6 additional patients), it described this entity with clinical signs, natural history and the characteristic that this entity may be of a reducible as well as an irreducible variety.
2. The article brought to light the fact that congenital atlanto–axial dislocation had a high prevalence in the Indian subcontinent.
3. The natural history of the disease and various types of clinical manifestations were described systematically including the presence of delayed myelopathy associated with this entity, even with minor trauma or exaggerated neck movements; the presence of transient bilateral visual obscuration due to posterior circulation stroke; and, Horner syndrome.
4. The high incidence of the association of an atlanto–axial dislocation with an occipitalized atlas and C2–C3 fusion, and an irreducible posterior directed odontoid was established, thus pointing towards the congenital aetiology.
5. A radiological classification was presented for the first time, which described the reducible and irreducible varieties of atlanto–axial dislocation in three distinct categories, those with the odontoid process attached to the C2 body, with or without occipitalization of the atlas; and, those with the odontoid absent or detached from the body of C2 (describing a hypoplastic odontoid or an os odontoideum, respectively). In these subgroups, the high association of irreducible atlanto–axial dislocation with occipitalization of the atlas and C2–C3 fusion; and, the high association of reducible atlanto–axial dislocation with the non-occipitalized atlas, and with hypoplastic odontoid and os odontoid, were described.
6. Autopsy findings established that the neurological manifestations were due to the compressive effects of the existing atlanto–axial dislocation and not due to any other spinal cord or brainstem disorders coexisting with bony anomalies.
7. Autopsied specimens showed that asymmetrical and abnormal C1–C2 joints are the main culprits in the pathogenesis of atlanto–axial dislocation.
8. An interesting finding on histological examination suggested that the point of maximum compression of the cord in atlanto–axial dislocation was often at the C2–C3 level (and not the C1–C2 level!) by the posteriorly directed odontoid.
9. It was established that canal compromise at the foramen magnum occurs when its diameter is reduced to less than 19 mm. An absolute value of 9 mm of spinal canal diameter in patients with atlanto–axial dislocation with an occipitalized atlas or with an hypoplastic odontoid or os odontoideum; and, a diameter of 11.5 mm in atlanto–axial dislocation without an occipitalized atlas represents the critical diameter that may precipitate myelopathy. However, no correlation of the advancing diameters with myelopathy was found.
10. The histological examination of the spinal cord at that level showed the classical antero-posterior flattening, the shortened anterior median fissure, the destroyed anterior horn cells and the almost unrecognizable posterior columns with oedematous vacuolation below this level.
11. A management protocol of attempting to reduce the apparently irreducible atlanto–axial dislocation by using cervical traction, utilizing progressively increasing traction weights, was established.
12. The most remarkable contribution of this paper was the detailed analysis of the results at follow-up and the honesty with which the results were published. At that time, only posterior decompression of the foramen magnum, the posterior arch of the atlas and the lamina of the axis were done to improve the canal diameter, with or without posterior stabilization by onlay bone grafting. However, Professor Wadia found that a number of patients in whom a posterior decompression had been done for congenital atlanto–axial dislocation deteriorated due to further aggravation of the medullospinal posterior distortion (that was already subjected to anterior compression by the posteriorly directed odontoid). This led to the concept of addressing the anteriorly based pathologies at the craniovertebral junction.
13. The article not only discovered that posterior decompression often resulted in an aggravation of myelopathy (and often a decompressive haematomyelia) in these patients but also described anterior grafting (with clinical improvement) in a patient whose condition had deteriorated following the posterior procedure.

All medical personnel dealing with patients with atlanto–axial dislocation will acknowledge that describing all these findings in a single paper in the 'era of plain radiographs', was a remarkable achievement. This paper is indeed a 'Classic'. An internet search in early 2016 revealed that this paper had been cited 91 times. This paper was written by a single author in the pre-PubMed and pre-internet era, when the number of journals on neurosciences were limited. The number of citations that this publication has received, therefore, points to the relevance of this paper in the clinical management of patients with congenital atlanto–axial dislocation even in the current 'computed tomographic/magnetic resonance imaging era'.

THE EVER-EXPANDING IMPLICATIONS

This paper served as the foundation for many of the prevailing concepts in the diagnosis and management of atlanto–axial dislocation and other associated craniovertebral junction anomalies. These include:

Recognition of the high prevalence of atlanto–axial dislocation in India: The included clinico-radiological findings and protocols in this article revived interest in the diagnosis and management of atlanto–axial dislocation in India. Several subsequent papers reporting on a large number of cases from India established the dominance of Indian neuroscientists in dealing with this entity. Many of them have discussed clinical and radiological parameters,[9–14] subtle differences between reducible and irreducible atlanto–axial dislocation,[15] atlanto–axial dislocation with Chiari malformation,[16–18] basilar invagination,[14,19–21] paediatric atlanto–axial dislocation[15,22–24] syndromic versus non-syndromic atlanto–axial dislocation[25] and rotatory atlanto–axial dislocation.[9,26–28]

Emphasis on the role of facet joints in the pathogenesis of atlanto–axial dislocation. This article evaluated the role of abnormally oriented C1–C2 facet joints in precipitating both atlanto–axial dislocation and basilar invagination. Atlanto–axial facet joints are the centre of mobility and instability of the C1–C2 vertebrae. They are one of the most mobile joints of the body. Several studies on C1–C2 facet joints were inspired by this article. The C1–C2 facets have been described as having a translational, lateral, vertical or rotatory dislocation.[29] It was found that in reducible atlanto–axial dislocation, the C1–C2 joints are relatively symmetrical when compared to irreducible dislocation, where the joints were nearly always asymmetrical with the joint surface often vertically oriented relative to each other.[15,30] Genetic polymorphisms in the genes encoding for the enzyme methylene tetrahydrofolate reductase were also found in much higher proportions in patients with irreducible rather than reducible dislocation. As the enzyme is involved in folic acid metabolism, this study pointed towards a congenital origin of irreducible atlanto–axial dislocation, indicating that both congenital and nutritional factors were involved in its genesis, and that irreducible atlanto–axial dislocation also differs from reducible atlanto–axial dislocation at a genetic level.[31] Likewise, in syndromic C1–C2 dislocation, the joints were more symmetrical and the C1–C2 dislocation was often found to be reducible when compared to the irreducible dislocation.[32] Degenerative osteoarthritis has also been associated with C1–C2 dislocation.[33] The emerging concept related to the C1–C2 facet joints is that by applying adequate traction or by causing C1–C2 joint distraction, the C1–C2 dislocation often reduces even in patients with an apparently irreducible atlanto–axial dislocation. This has rendered the distinction between a reducible and irreducible atlanto–axial dislocation nebulous.[34]

Emphasis on the role of studying the vascular anatomy of the posterior circulation and the relationship of the vertebrobasilar system to the C1–2 joints while dealing with congenital atlanto–axial dislocation. In this article, transient visual loss indicated the association of atlanto–axial dislocation with vertebrobasilar insufficiency and bilateral occipital lobe ischaemia. This finding inspired several studies. An elegant anatomical study has established various associations of the vertebral artery with the C1–C2 facet joints.[35] It was also found that in patients with posterior circulation strokes precipitated by atlanto–axial dislocation, the contralateral vertebral artery also showed evidence of severe stretching. Perhaps, this stretching of the vertebral artery led to its occlusion or dissection in the V3 segment on the involved side in the presence of atlanto–axial dislocation.[36] Other papers have suggested an increased vulnerability of the vertebral artery to injury during surgery for congenital atlanto–axial dislocation. Thus, a heightened risk of vertebral artery injury was present in patients with a persistent first intersegmental artery, a fenestrated vertebral artery, and a low-lying posterior inferior cerebellar artery, where the vertebral artery crossed the C1–C2 facet joint. A vertebral artery with an anomalous medial deviation; a high-riding vertebral artery at the level of the axis vertebra associated with a narrow axial isthmus; and, a vertebral artery in association with rotation/tilt at the craniovertebral junction was also at increased risk of getting injured either spontaneously or during surgery.[37,38]

CHANGES IN TREATMENT PARADIGMS BASED ON THIS ARTICLE

The greatest influence of this article has been on establishing the management protocol of patients with atlanto–axial dislocation. This influence was exhibited in many ways. First, the role of preoperative traction in stabilizing the C1–C2 joints in patients with an atlanto–axial dislocation was validated. Further studies established the role of traction in distracting the odontoid from the foramen magnum; as well as in transforming the natural lordotic curvature of the upper cervical spine to a straighter one, thus changing the direction of the posterior directed odontoid to a more vertical one. This helped in increasing the canal diameter at the foramen magnum and in relieving pressure on the cervicomedullary junction. It also laid the foundation for future studies which established that starting with a traction weight that is 4%–5% of the body weight with gradual increments of up to a maximum of 7 kg would be a useful norm for escalation of the traction weights in patients with irreducible atlanto–axial dislocation and basilar invagination.[12,39]

Second, the beneficial role of traction also paved the way for placing an internal C1–C2 distraction and fixation device that helped in aligning the upper cervical spinal curvature to exactly the same beneficial position as achieved following the application of traction.[40,41]

Third, the article, using a large number of patients, and after systematically classifying patients into various categories, provided details of the clinical outcome after a sufficiently long follow-up. This classification is still used in the day-to-day management of patients with congenital atlanto–axial dislocation.

Fourth, an anterior approach towards stabilization of the C1–C2 joints was attempted with a successful long-term outcome.

Finally, in patients who deteriorated and died, detailed autopsy and histopathological examination of the bony and soft tissue structures irrefutably established that (i) the deterioration was due to a bony compression that would have been amenable to an adequate surgical decompression had the patient survived; and (ii) the atlanto–axial dislocation constitutes an anteriorly placed cervicomedullary compression

which causes posterior distortion of the upper cervical cord. Performing a posterior decompression not only does *not* address the anteriorly-based pathology but also causes considerable neurological deterioration by further exacerbating the posterior distortion of the cord due to the removal of the posterior support structures. This led to major changes in management strategies, and surgeons shifted their attention to alleviating the anterior cervicomedullary compression by direct reduction and stabilization in cases with a reducible atlanto–axial dislocation; and, by transoral decompression of the odontoid and posterior stabilization in cases with an irreducible atlanto–axial dislocation.[42,43] The currently prevailing philosophy regarding congenital atlanto–axial dislocation is that distinguishing between an irreducible or reducible atlanto–axial dislocation is no longer relevant as all dislocated C1–C2 joints (however, distorted and vertical they might be) are amenable to reduction either by traction,[15,23] by C1–C2 distraction,[19,34,40] by multiplanar translational and angular manipulation,[44] by direct C1–C2 joint drilling to render joint surfaces horizontal,[45] or by replacing the joint surfaces with artificial atlanto–axial joints.[46]

Thus, barring exceptional circumstances, the pendulum has swung again to the posterior approaches. Despite this trend, the underlying philosophy propounded by the article written by Drs Dastur, Wadia, Desai and Sinh in 1965,[8] as well as this article by Dr Wadia in 1967 (especially the latter by establishing the findings systematically in a large number of patients with a well-defined classification), has remained unchanged. The philosophy essentially states that whatever be the approach, whether anterior, posterior or combined, a sustained C1–C2 reduction and a long-term improved patient outcome are obtainable only by focusing on the anterior cervicomedullary compression.

W.E.B. Du Bois stated, 'A classic is a book that does not have to be written again.' This landmark article by Dr Wadia has withstood the test of time for more than 50 years and has been a model for several generations of scholars, for the breadth of its engagement and the ripple effects it produced, and for the depth of the insightful and systematic research that it contains.

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