Chapter 7
Evolving Concepts of Craniovertebral and Spinal Instability

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7.1 Introduction

"Mobility" defines life. Mobility and stability are essential elements of life. Human beings are additionally "burdened" by their life long-standing posture. The major bulk of human muscles is located on the extensor compartment of the spinal column or on its "back" and caters to movements that facilitate sitting, standing, and running. On the other hand, only relatively "few" strands of muscles are located in the flexor or anterior compartment of the spinal column, flexion movement being essentially of passive nature. The activity of all major extensor muscles is focused on the facetal articulation of the spine that forms the point of fulcrum of all movements. "Essentially" activity of no major muscle group is focused on the disc or the odontoid process, or in other words the disc or the odontoid process does not form a fulcrum point of movements. Our articles have discussed the role of the disc and the odontoid process in human movements [1]. We philosophized that both disc and odontoid process are like opera conductors who regulates all music without holding any instrument in his hands. Whilst muscles are the brawn, disc (and odontoid process) is the brain of all movements. Weakness of muscles related to their disuse,

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abuse, or injury forms the basis of all spinal instability. Instability is the primary process or the nodal point of pathogenesis of majority of the known craniovertebral junction "anomalies" and a number of spinal ailments that include the so-called "degenerative" spinal disease and deformities. In situations with chronic instability, a number of structural musculoskeletal and neural alterations are a part of Nature's protective or adaptive endeavors. These alterations are secondary, naturally protective, and potentially reversible following treatment that involves spinal segmental stabilization. Understanding the fact that these secondary alterations point towards the unstable spine can rationalize and direct the treatment.

7.2 Craniovertebral Junction

Craniovertebral junction has a supremely designed architecture that caters to the most mobile and most stable region of the body in addition to providing safety to the most critical neural and vascular structures that transit in the region. Mobility and stability are the hallmarks of craniovertebral junction. Occipitoatlantal joint is the most stable joint, and atlantoaxial joint is the most mobile joint of the body. Whilst atlantoaxial joint is the most mobile joint, it is potentially most susceptible to instability. Our four-decade-long experience in the field suggests that it may not be erroneous to state that atlantoaxial instability is possibly the most frequent, most neglected and misunderstood and undertreated clinical entity in our subject. "Compression" of the neural structures by the odontoid process is the most feared issue in the subject of medicine in general and in craniovertebral junction in particular. In general, craniovertebral junction instability is synonymous with atlantoaxial instability, and craniovertebral junction stabilization is synonymous with atlantoaxial stabilization. Inclusion of the occipital bone in the fixation construct is unnecessary, adds to the possibilities of complications, and results in suboptimal fixation. Occipitoatlantal instability is extremely rare clinical entity and is encountered in high-speed vehicular injury and also rarely in syndromic multisegmental spinal instability.

7.3 Atlantoaxial Articulation

The atlas and axis vertebral bones are specially designed [2]. The spinous process of axis is largest, transverse processes of atlas are longest, and facets of the atlas and axis are the strongest of the entire spine. The articular surface of the atlantoaxial joint is flat and round, like no other joint in the body. It caters to circumferential movements of the region. Occipitoatlantal articulation has a cup-and-saucer configuration and facilitates attachment for strong and thick ligaments all along its edges.

7.4 Atlantoaxial Dislocation or Instability

Incompetence of the muscles and ligaments at the fulcrum point of facets of atlas and axis at the atlantoaxial facetal articulation results in atlantoaxial instability. The atlantoaxial instability can be anteroposterior wherein the atlantodental interval abnormally increases and there can be dural and neural compression opposite the tip of the odontoid tip. Atlantoaxial instability is diagnosed on dynamic images with the head in flexion and in extension. The atlantodental interval of more than 3 mm in adults and 5 mm in pediatric age group is generally considered to be indicative of atlantoaxial instability. This parameter to diagnose instability is the most frequently used and is probably the only validated parameter to identify instability [3, 4].

Atlantoaxial instability can be **mobile and reducible** (Fig. 7.1) when atlantodental interval returns to normal on head extension and **partially or completely fixed or irreducible** (Fig. 7.2) when atlantodental interval does not change or only partially reduces on head extension [5].

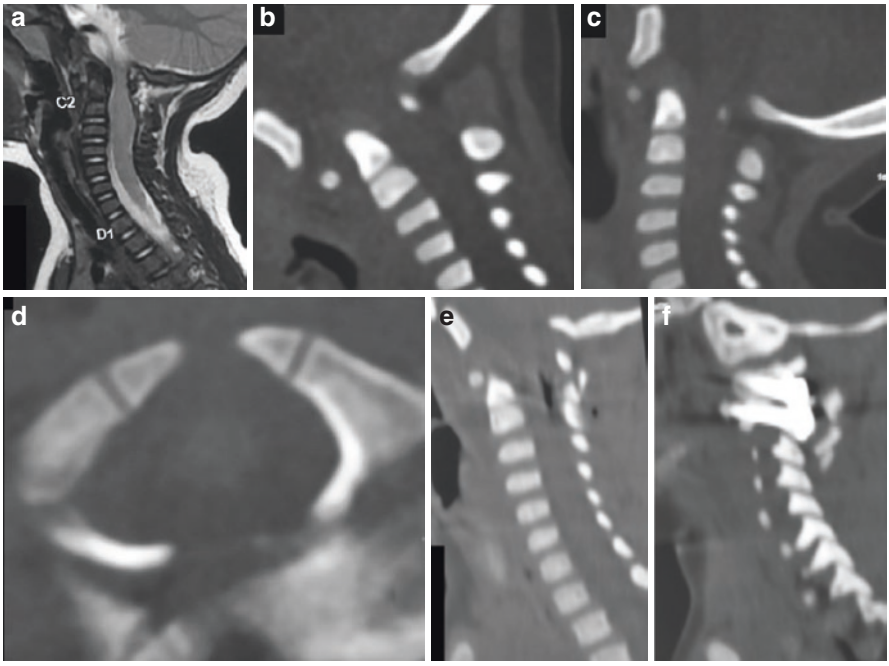

Fig. 7.1 Images showing mobile atlantoaxial instability in a 3-year-old male patient. (**a**): T2-weighted magnetic resonance image showing atlantoaxial dislocation and cord compression opposite the odontoid process. (**b**) Computed tomographic (CT) scan with the head in flexion shows atlantoaxial dislocation. (**c**) CT scan with the head in extension position showing incomplete reduction of the dislocation. (**d**) Axial cut of CT scan showing bifid anterior and posterior arches of atlas. (**e**) Postoperative CT scan showing atlantoaxial fixation in reduced position. (**f**) Image showing the implants in the facets of atlas and axis

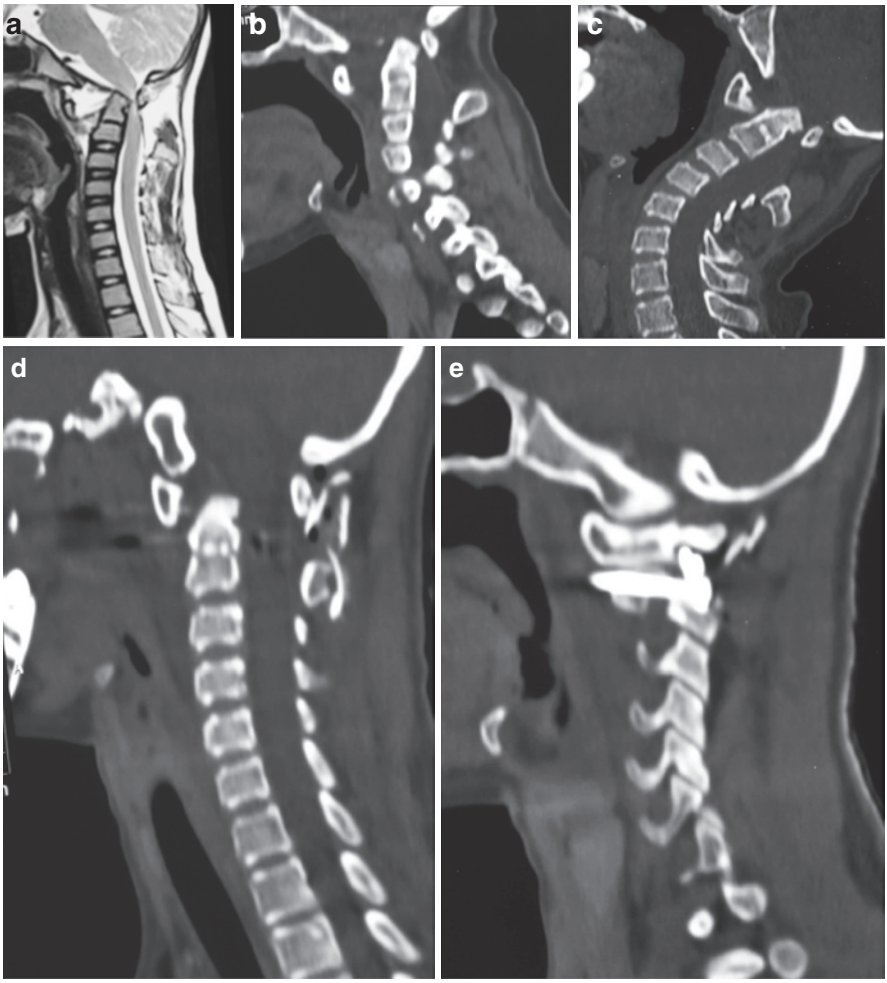

Fig. 7.2 Images of an 18-year-old male patient showing "irreducible" atlantoaxial dislocation. (**a**) T2-weighted MRI showing atlantoaxial dislocation and cord compression by the odontoid process. (**b**) CT scan with the head in flexed position showing severe atlantoaxial dislocation. (**c**) CT scan with the head in extension position does not show any reduction in atlantoaxial dislocation. (**d**) Postoperative CT scan showing realignment of the craniovertebral junction and the atlantoaxial fusion. (**e**) Postoperative image through the facets showing lateral mass plate and screw fixation

In the year 2009, we identified **vertical** atlantoaxial instability (Fig. 7.3) wherein the odontoid process moves up and down in the form of a piston on dynamic imaging that involves flexion and extension of the head [6]. There is no abnormal alteration in the atlantodental interval during these movements.

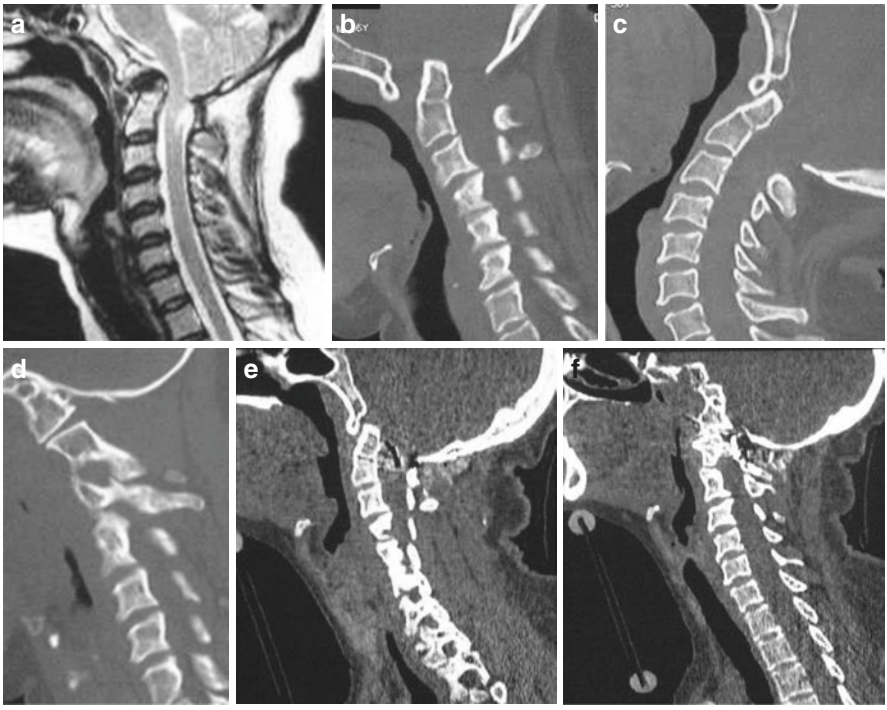

Fig. 7.3 Images of a 23-year-old male patient showing mobile atlantoaxial dislocation in a case with basilar invagination. (**a**) T2-weighted MRI image showing basilar invagination, assimilation of atlas, Chiari formation, and cord compression. (**b**) CT scan with the head in flexed position showing basilar invagination in the form of vertical or superior migration of the odontoid. (**c**) CT scan with the head in extension showing reduction of the dislocation. (**d**) CT scan cut through the facets showing assimilation of atlas and no significant malalignment. (**e**) Postoperative CT scan showing realignment of the craniovertebral junction. (**f**) Postoperative image through the facets showing lateral mass plate and screw fixation

In fracture of the ring of atlas related to trauma, destruction related to tumor or infection like tuberculosis or presence of bifid atlas, the facets of atlas are dislocated laterally in relationship to the facet of axis. Such dislocation is termed as **lateral** atlantoaxial dislocation [7, 8].

Rotatory dislocation is when there is an element of rotation in the facets of atlas and axis with the facet of atlas positioned anterior to the facet of axis on one side and posterior to the facet of axis on the other [9] (Fig. 7.4). A number of types of rotatory dislocation have been discussed in the literature.

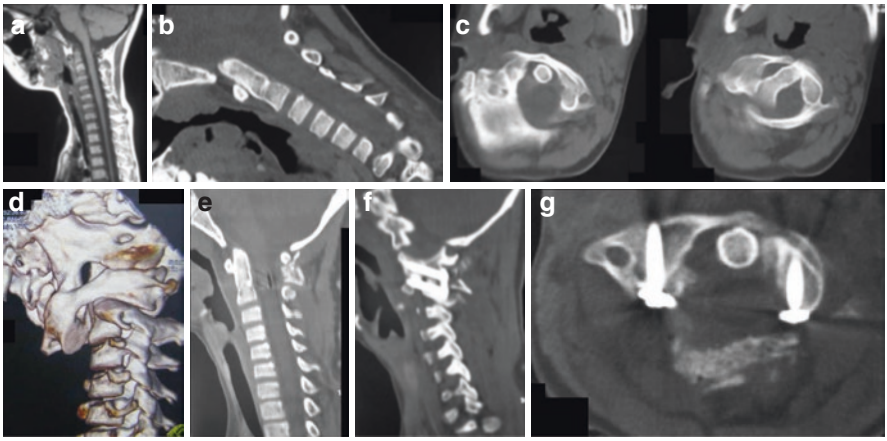

Fig. 7.4 Images a 12-year-old female patient showing rotatory atlantoaxial dislocation. (**a**) T1-weighted MRI showing abnormal tilt of the odontoid process with no change in atlantodental interval. (**b**) Sagittal cut of CT showing abnormal alignment of the odontoid process with no change in atlantodental interval. (**c**) Axial view of the CT scan showing rotatory dislocation. (**d**) 3D reconstructed view of CT showing the rotatory dislocation. (**e**) Postoperative CT scan showing reduction of the tilt of odontoid process. (**f**) Sagittal image of the CT scan showing fixation of the facets of atlas and axis in reduced position. (**g**) Axial CT scan showing screws passing through the facets of atlas. Reduction of the dislocation can be observed

7.5 "Fixed" or "Irreducible" Atlantoaxial Instability

Till about three decades ago, the surgical treatment of mobile and reducible atlantoaxial instability was fixation or stabilization; the treatment of fixed or irreducible atlantoaxial instability was decompression, by resection of the compressing odontoid process by the transoral surgical route and foramen magnum decompression from the posterior surgical route. In the year 2005 for the first time in the literature, we identified that the so-called fixed or irreducible atlantoaxial dislocation is "never" fixed or irreducible, but it is always mobile and pathologically hypermobile and can be reduced by atlantoaxial facetal manipulation and distraction [5] (Fig. 7.2). This concept is now established, and majority of surgeons dealing with craniovertebral junction attempt craniovertebral junction realignment and stabilization in such cases, rather than resorting to decompression by bone resection.

7.6 Central or Axial Atlantoaxial Instability (CAAD)

In the year 2014, we classified atlantoaxial instability on the basis of alignment of the facets of atlas and axis on lateral profile imaging with the head in neutral position [10]. **Type 1** atlantoaxial facetal instability is when the facet of atlas is dislocated anterior to the facet of axis. Atlantodental interval is increased in such

dislocation, and there may be dural and neural compression. **Type 2** atlantoaxial facetal instability is when the facet of atlas is dislocated posterior to the facet of axis. **Type 2 rotatory** atlantoaxial facetal instability is when the facet of atlas is dislocated posterior to the facet of axis on one side and is normally aligned on the contralateral side. **Type 3** atlantoaxial instability is when the facet of atlas and axis are in alignment. Instability in such cases is diagnosed on the basis of telltale radiological and clinical evidences and is confirmed by direct manipulation of bones during surgery. In both type 2 and type 3, there may not be any abnormal alteration of atlantodental interval or any evidence of dural or neural compression by the odontoid process. Such instability is labeled as central or axial atlantoaxial instability (CAAD) [11–13]. Whilst type 1 atlantoaxial instability is usually relatively acute in onset and symptoms are pronounced, CAAD is usually of chronic or long-standing nature, and the symptoms are relatively subtle and relentlessly progressive.

7.7 Acute and Chronic Atlantoaxial Instability

Acute atlantoaxial dislocation is more often related to trauma or injury. Symptoms related to acute atlantoaxial instability are pronounced and sudden and can be disabling and less frequently even fatal. Moderate to severe pain in the nape of the neck, neck stiffness and muscle spasm, and varying range of neurological symptoms and deficits in the limbs are more often the presenting symptoms. On the other hand, atlantoaxial instability can be of chronic or long-standing nature [14, 15]. The duration of instability can be of months or years. Chronic instability is usually of type 2 or 3 atlantoaxial facetal instability or of CAAD. In such potential or manifest instability, there are several and wide-ranging musculoskeletal and neural alterations that appear to be anomalies and "pathological" or compressive but have a protective role and are potentially reversible following atlantoaxial stabilization. These secondary musculoskeletal alterations include the "complex" of basilar invagination. Skeletal alterations include platybasia, Klippel-Feil abnormality, bifid anterior and posterior arch of atlas, bifid posterior elements of C2, os odontoideum, assimilation of atlas, and C2–3 fusion. Neural alterations include Chiari 1 formation, syringomyelia, syringobulbia, external syringomyelia, and external syringobulbia. Our earlier article discuss that in the presence of chronic atlantoaxial instability, there can be short head, short neck, and short spine. Whilst short neck is associated with low hairline and torticollis, short spine can be associated with dorsal kyphoscoliosis. Atlantoaxial instability is indicated when all these naturally protective and secondary alterations are present either discretely or in cohort. More importantly, they suggest the need for atlantoaxial stabilization. All the secondary alterations are potentially reversible following atlantoaxial fixation. Any kind of direct surgical manipulation to any of the abovementioned secondary alterations can only have negative clinical consequences.

7.8 Basilar Invagination

A number of radiological parameters have been described that determine basilar invagination. Amongst these, Chamberlain's line, McGregor's line, and Wackenheim clival line are amongst the more popularly deployed. For several decades, basilar invagination was considered to be associated with "fixed" atlantoaxial instability, and "decompression" of the craniovertebral junction was the accepted form of surgical treatment.

Our understanding of basilar invagination has evolved in three stages. These are briefly discussed.

Stage 1: In the year 1998, we divided basilar invagination into two groups [16]. Group 1 basilar invagination was when the odontoid process migrated into the foramen magnum, and Group 2 was when there was Chiari 1 malformation or tonsillar herniation. As basilar invagination was considered to be a fixed anomaly, decompression was identified to be the treatment. For Group 1 transoral decompression and for Group 2 foramen magnum decompression was considered to be the ideal form of treatment. Role of stabilization was not entirely clear at this time and was considered only because resection of bones from transoral route or by foramen magnum decompression was identified to have potential destabilizing effects in the long run. During this phase of evolution, it was observed that for Chiari malformation and for syringomyelia, there might not be any role for opening the dura after the surgical procedure of foramen magnum decompression [16].

Stage 2: In the year 2004, we divided basilar invagination into two groups [17]. Group A was when odontoid process migrated into the foramen magnum resulting in an increase in atlantodental or clivodental interval (Fig. 7.5). Group B was when there was no alteration in the atlantodental interval (Fig. 7.6). Whilst instability was identified in Group A, Group B basilar invagination was still considered to be a "fixed" anomaly. We identified similarities between lumbar spondylolisthesis and C1 over C2 facetal listhesis that results in Group A basilar invagination [18]. Similarities between the treatment protocol of lumbar spondylolisthesis and basilar invagination were accordingly identified. For Group A, atlantoaxial stabilization and attempts towards craniovertebral junction realignment were advocated, and for Group B, foramen magnum decompression was considered to be the treatment. Distraction of facets of atlas and axis and reduction-stabilization of atlantoaxial articulation introduced a novel concept and a new format of treatment of Group A basilar invagination. The concept that basilar invagination can be reduced and that transoral decompression can be avoided radically changed the treatment of this clinical entity. During the years, the authors have treated several cases of basilar invagination Group A with only stabilization and without any form of decompression.

Stage 3: In the year 2012, it was observed that "chronic" atlantoaxial instability forms the point of pathogenesis of Group B basilar invagination [19]. More often, such patients have central or axial atlantoaxial instability (CAAD). Secondary musculoskeletal and neural alterations are more profound in such cases. Atlantoaxial

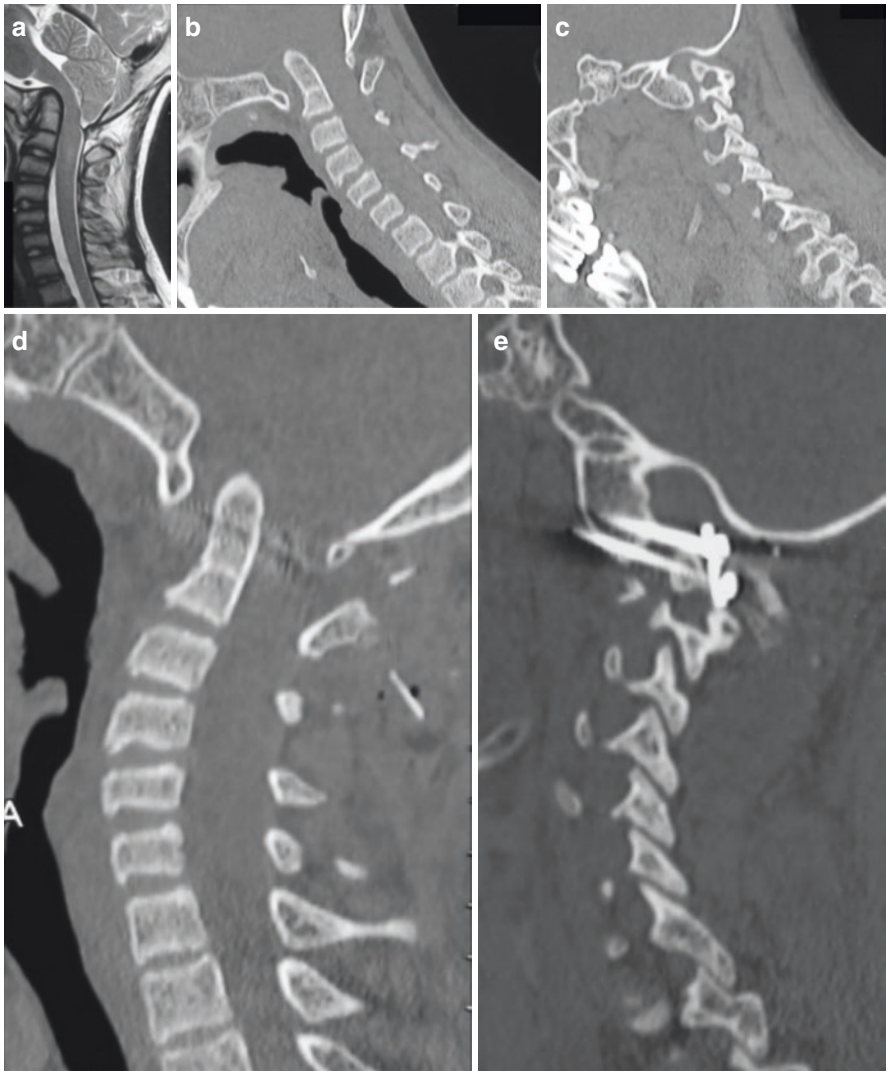

Fig. 7.5 Images of a 30-year-old female patient showing with Group A basilar invagination and Chiari formation. (**a**) T2-weighted MRI showing Group A basilar invagination and Chiari formation and indentation of the brainstem by the odontoid process. (**b**) CT scan with the head in flexion showing the basilar invagination. (**c**) CT scan with the cut passing through the facets showing type 1 atlantoaxial dislocation. (**d**) Postoperative CT scan showing the craniovertebral junction realignment. (**e**) Postoperative image with sagittal cut passing through the facets showing the metal construct

stabilization forms the basis of surgical treatment [20, 21]. Any form of decompression can have negative clinical implications. The author is convinced that foramen magnum decompression in such cases can soon become a historical operation.

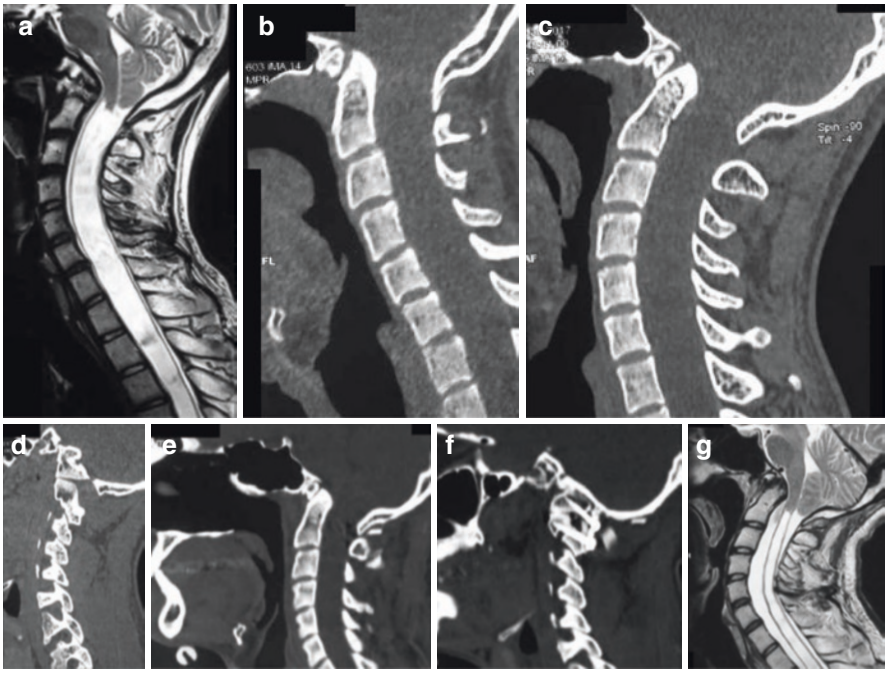

Fig. 7.6 Images of a 23-year-old male patient having Group B basilar invagination. (**a**) T2-weighted MRI showing basilar invagination, Chiari formation, and syringomyelia. (**b**) CT scan with the head in flexed position showing basilar invagination. Assimilation of atlas is seen. (**c**) CT scan with the head in extended position shows so significant alteration in craniovertebral junction bone alignment. (**d**) CT scan cut through the facets showing that the facets of atlas and axis are in alignment. (**e**) Postoperative image. (**f**) Postoperative image with the cut passing through the facets showing the metal implant. (**g**) Delayed postoperative MRI showing reduction in the size of the syrinx

Essentially, the treatment strategy of all types of basilar invagination has changed in the last few years from only decompression to only fixation. Basilar invagination is now identified to be a secondary and protective outcome of chronic atlantoaxial instability, and atlantoaxial stabilization is considered to be the treatment.

7.9 Craniovertebral Junction Alterations

Platybasia, Klippel-Feil alteration, assimilation of atlas, C2–3 fusion, bifid arches of atlas, os odontoideum, and several other so-called pathological clinical entities are secondary to atlantoaxial instability, are naturally protective, and are potentially

reversible following atlantoaxial stabilization [22–28]. Chiari formation, syringomyelia, and basilar invagination are a common clinical triad [29]. When they are present discretely or in cohort or when they are present in association with one or more of other musculoskeletal alteration are indicative of presence of atlantoaxial instability and are suggestive of the need for surgery that involves atlantoaxial stabilization.

7.10 Chiari Formation and Syringomyelia

Chiari formation and syringomyelia are relatively common clinical entities. They are associated with relentlessly progressive clinical symptoms. The symptoms range from neck pain, pain in shoulders and hands, and weakness in the hands that progresses eventually to weakness of all four limbs, sensory dysfunction, breathing disturbances, sleep apnea, and several such symptoms that can eventually lead to crippling neurological deficits. Chronic atlantoaxial instability is the nodal point of pathogenesis. The understanding that Chiari formation is associated with atlantoaxial instability and atlantoaxial stabilization is the treatment has a potential to radically alter the generally followed surgical treatment of foramen magnum decompression (Fig. 7.7) [23, 24, 30–33]. Presence of Chiari, syringomyelia, basilar invagination, and any of the other listed secondary alteration either in a cohort or discretely indicate presence of atlantoaxial instability and suggest the need for atlantoaxial stabilization. Any kind of decompression that involves bone or soft tissue resection in the presence of unstable atlantoaxial articulation can only have negative implications. Dramatic clinical recovery from all symptoms was observed that started on awakening from anesthesia. We identified recovery of motor evoked potential during surgery at the moment when spinal stabilization is completed [34].

We introduced the terms “external” syringomyelia and “external” syringobulbia when “excessive” or more than usual amount of CSF is present around the spinal cord or brainstem [35–37]. Such CSF alteration is Nature’s protective formation and indicates presence of atlantoaxial instability.

Short neck, short head, and short spine are secondary “protective” consequences of chronic atlantoaxial instability [38]. Whilst short neck can be associated with torticollis, short spine can be associated with dorsal kyphoscoliosis [39]. Presence of such spinal alterations is protective, is indicative of chronic atlantoaxial instability, and is potentially reversible following atlantoaxial stabilization.

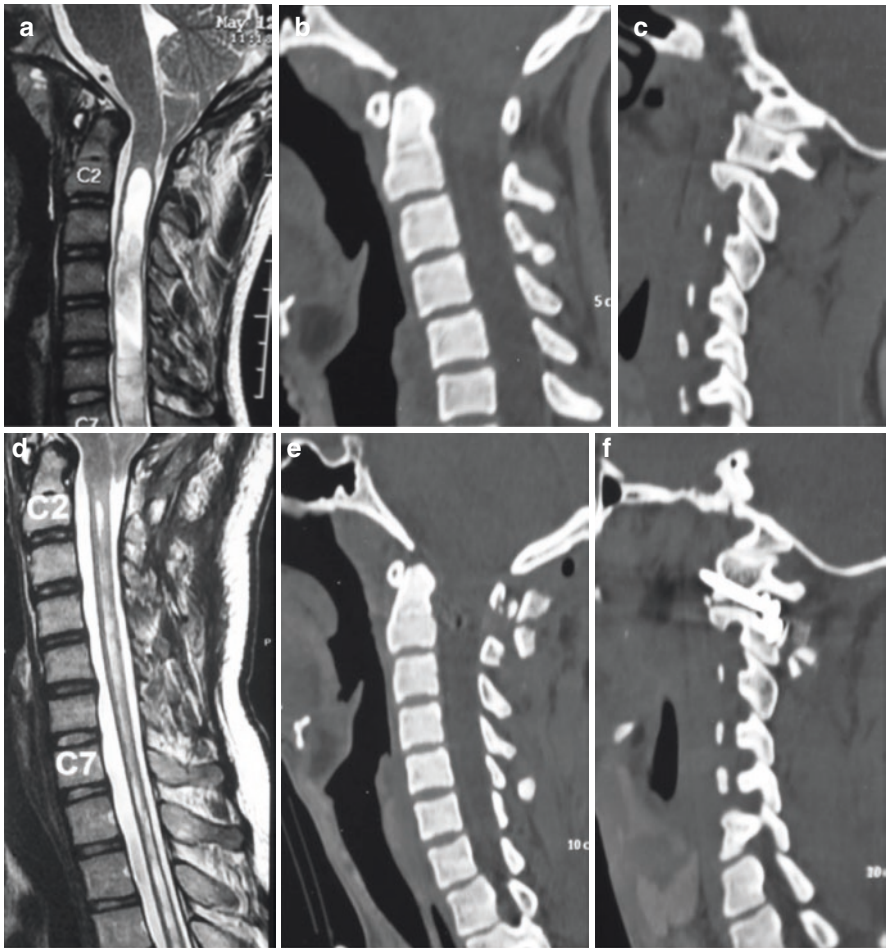

Fig. 7.7 Images of a 19-year-old male patient having Chiari formation and syringomyelia without any significant bone alteration at the craniovertebral junction. (**a**) T2-weighted sagittal MRI showing Chiari formation and syringomyelia. (**b**) CT scan showing no craniovertebral junction bone anomaly. (**c**) CT scan with the cut passing through the facets showing type 2 facetal instability. (**d**) Postoperative T2-weighted MRI showing resolution of the Chiari formation and syringomyelia. (**e**) Postoperative CT image showing no bony decompression. (**f**) Postoperative CT scan showing the implants

7.11 Vertical Spinal Instability

Standing position makes humans unique. This position entails lifelong stress on the extensor muscles located on the "back" of spine. Weakness of these muscles due to disuse, abuse, or injury leads to listhesis of the facets and telescoping of the spinal segments. This retrolisthesis may not be identified on plain or dynamic imaging. Our articles on the subject identify listhesis of the facets as the nodal point of genesis of spinal degeneration [40–46].

7.12 Spinal Degeneration

A number of clinical and radiological features characterize spondylotic disease. Disc space reduction, osteophyte formation, ligamentum flavum hypertrophy, and eventual reduction of spinal and root canal dimension result in symptoms of radiculopathy or myelopathy. Facetal retrolisthesis is included in the gamut of degenerative changes and is considered to be a secondary phenomenon to primary disc space reduction. For several decades degenerative spondylosis has been defined as secondary processes that result from primary disc degeneration, reduction of its water content, and disc space reduction.

Era of computer-based imaging: Advances in the MRI and CT scan technology now provide a clear image of the consequence of spinal degeneration. Compression and deformation of the neural structures by bulging or herniated disc, osteophytes, and thickened ligaments are clearly visualized. Effect on the spinal cord is demonstrated by signal alterations. As cord compression has been considered to be the primary squeal of spinal degeneration, decompression of the spinal cord by anterior decompressive measures like corpectomy and discoidectomy and posterior decompressive measures like laminectomy and laminoplasty have been the prime focus of surgical treatment. The aim of decompression is to provide space for the spinal cord so that the "intruders" could be accommodated and tolerated. Osteophytes and hypertrophy of the ligamentum flavum and other intervertebral ligaments are considered to be the prime factors that result in cord compression and its related ill effects. The more modern treatment focuses on the disc, osteophytes, and thickened ligaments, and the surgical procedure aims to resect these "pathological" entities and provide space for spinal cord and nerve roots.

Issue of spinal instability: The concept that disc degeneration or disc space reduction is not the primary issue in spondylotic spinal disease has a potential to influence or revolutionize the treatment strategies. The issue of instability has never been incorporated as the primary and nodal point of pathogenesis of spondylotic process. The need of treatment by stabilization is generally considered because the surgical treatment by anterior or posterior decompression is likely to have a secondary destabilizing effect on the spine. Considering this possibility, currently decompression-fixation has been the preferred twin operations. Specialized distractor-spacer-fixator placed in the intervertebral space after wide removal of the disc partakes in the process of decompression and provides a background for arthrodesis. Posterior interlaminar and interspinous process spacers have also been popular options.

More recently, some authors prefer to introduce artificial disc with the aim of retaining the movements of the intervertebral joint after wide and appropriate decompression. The possible issue with movement preserving surgery over fusion-fixation option is currently a debated issue.

Goel's concept of pathogenesis of degenerative spine: In the year 2010, Goel introduced an alternative concept regarding the pathogenesis of degenerative spondylotic disease. This concept hypothesized that spinal instability is the

primary pathogenetic issue in the initiation, development, and progression of degenerative spinal disease [41, 42]. Instability is related to and a direct consequence of the weakness of the muscles of the nape of the neck and back. The weakness of the muscles can be due to injury, misuse, or disuse and lack of their proper care by appropriate and full use. The weakness of the muscles is also related to standing human position that lays long-term stress. **Vertical spinal instability** and facetal overriding or listhesis are manifestations of weakness or incompetence of the paraspinal muscles [40]. Even modern images do not show clearly the abnormalities of alignment or instability of the facet joint on dynamic imaging. Due to oblique profile of the facets in the cervical and dorsal spine and a more vertical orientation in the lumbar spine, the dislocation is not horizontal but vertical or oblique when observed from a profile view.

Goel speculated that the primary or the initiation point of spinal degeneration is the facet joint [40–42]. This is the nodal point of initiation and progression of the "spondylotic" disease process. Facetal instability is of vertical nature and results in facetal overriding or listhesis. The facetal instability is manifested by reduction in the intervertebral spaces and buckling of the ligaments. This concept is in marked variation of the earlier hypothesis that suggested disc space reduction is the primary issue and rest of the consequences being secondary. From reduction of the anterior intervertebral space, the concept now places focus on the overriding of the posterolaterally placed facets. The generally identified primary issues in spinal degeneration of disc space reduction, osteophyte formation, ligamentum flavum buckling, and reduction in the spinal canal and neural foramina are secondary and probably protective issues related to primary spinal instability [47–49]. The emphasis on instability as the primary issue has the potential of changing the focus of treatment from decompression to stabilization. The symptom of claudication pain related to lumbar canal stenosis also appears to be secondary to weak back muscles that give way or get fatigued after a period of walking [50]. It seems that the muscles not only play a role in the movements of the spine but also participate in distraction of the intervertebral segments.

Disc herniation appears to be secondary to or can be a cause of focal spinal instability. Instability is the defining issue, and stabilization of the affected spinal segment is the treatment (Figs. 7.8, 7.9, and 7.10). Our studies identify disc herniation to be a protective natural issue, and the related pain or radiculopathy assists in avoiding excessive local movements in the face of focal spinal instability [51–55].

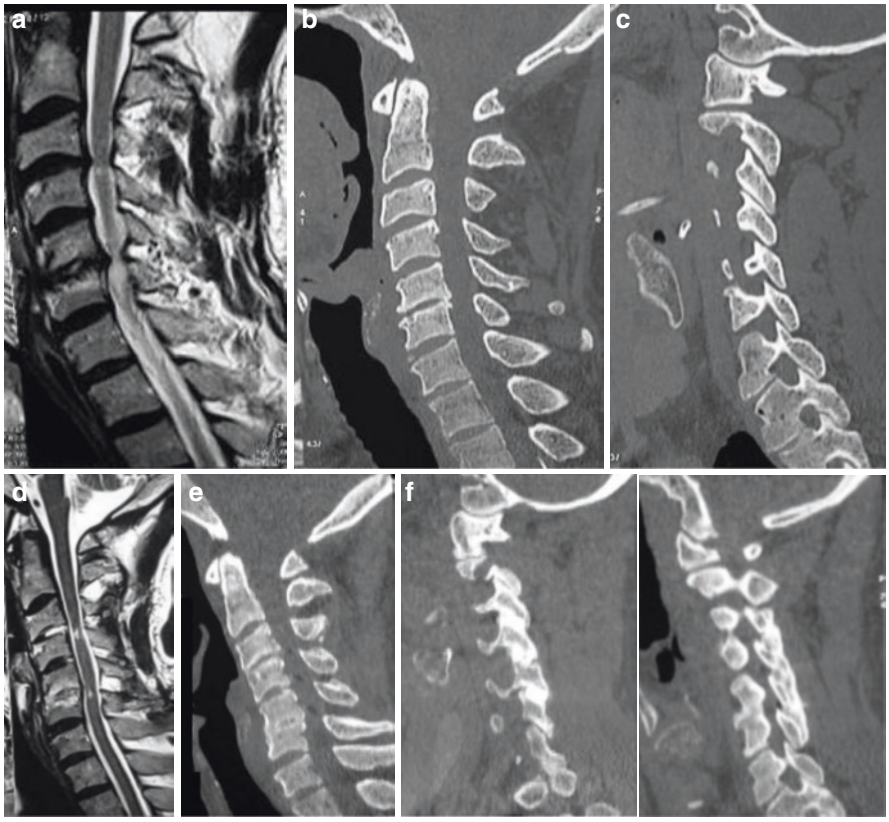

Fig. 7.8 Images of a 48-year-old male patient showing fixation using facet spacers. (**a**) T2-weighted MRI showing evidence of significant spondylotic disease with cord compression opposite C3–4 and 5–6 disc spaces. (**b**) CT scan showing degenerative changes in the spine. (**c**) Sagittal section depicting the facets. (**d**) Postoperative MRI showing reduction in the extent of cord compression. Resorption of the osteophytes and reduction in the buckling of posterior longitudinal ligament and ligamentum flavum at the levels treated can be seen. (**e**) CT scan showing distraction and increase in the intervertebral and interspinous process spaces. (**f**) Sagittal section through the facets showing the spacers with the C3–4, 4–5, and 5–6 facet joints with evidence of arthrodesis

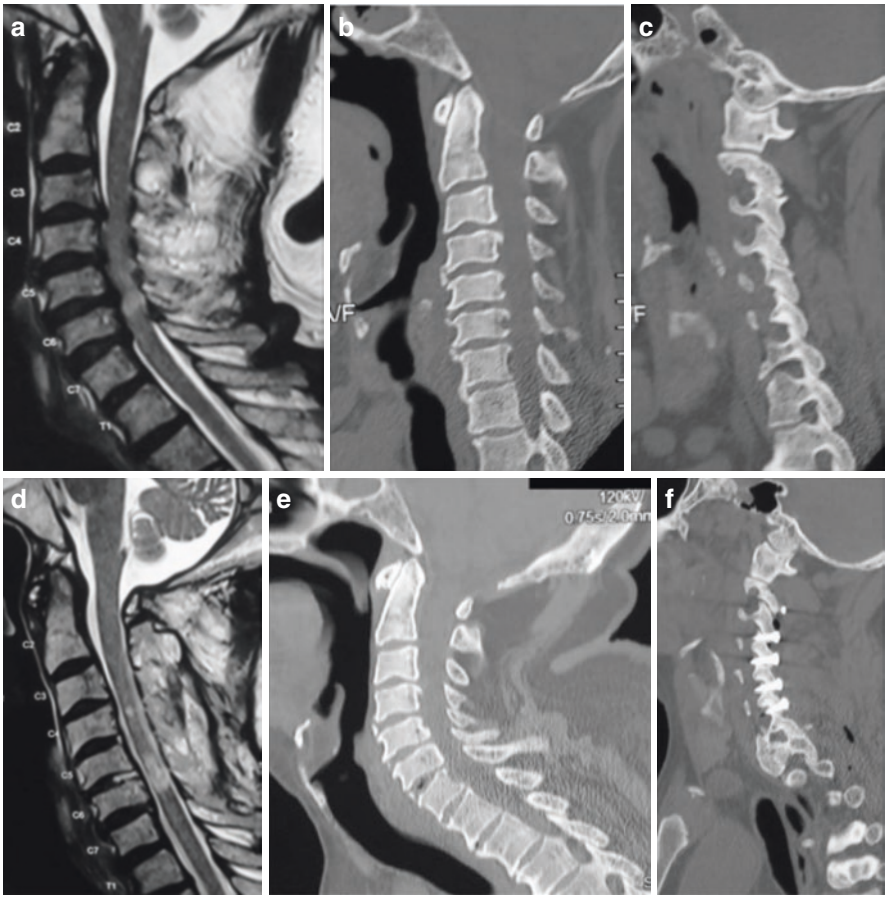

Fig. 7.9 Images a 65-year-old male patient showing fixation using transarticular facet fixation. (**a**) T2-weighted MRI showing evidence of significant multi-level spondylotic disease with cord compression. (**b**) CT scan showing degenerative changes in the spine. (**c**) Sagittal section showing the facets. (**d**) Postoperative MRI showing reduction in the extent of cord compression. (**e**) Postoperative CT showing no bone decompression. (**f**) Sagittal section through the facets showing C2–7 transarticular screw fixation

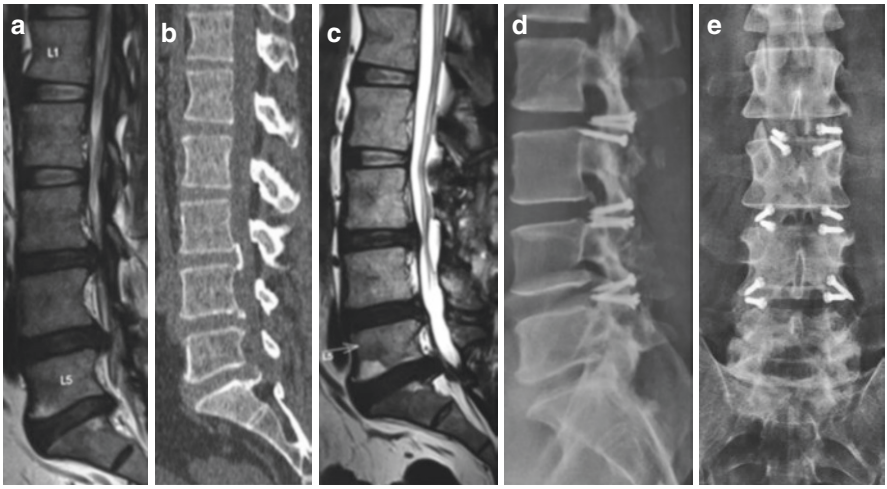

Fig. 7.10 Images showing lumbar transarticular screw fixation. (**a**) T2-weighted MRI showing degenerative changes in the lumbar spine. (**b**) CT scan of the lumbar spine. (**c**) Postoperative MRI (3 months after surgery) showing reversal of the degenerative changes. (**d**) Lateral X-ray showing the double insurance transarticular screw fixation. (**e**) Anteroposterior view of radiograph showing the fixation

7.13 Facet Distraction-Stabilization-Arthrodesis Surgery

Facet distraction and arthrodesis as treatment of single- or multiple-level cervical radiculopathy and myelopathy and lumbar spine degeneration added a new dimension to the treatment and to the understanding of the process of spinal degeneration [43, 44]. Introduction of intra-articular inter-facetal spacers reversed or had the potential of reversal of the entire spectrum of degenerative processes in the spine (Fig. 7.8). Distraction of the facets resulted in an immediate increase of dimensions of spinal canal and neural foramen and also increased the intervertebral distances that included an increase in the intervertebral height. Distraction resulted in stretch to the buckled ligamentum flavum and circumferential intervertebral ligaments that included the posterior longitudinal ligament. There is a potential of regression of osteophytes and restoration of disc fluid volume following facetal distraction. The fact that there is a reversal or potential of reversal of all known pathogenetic factors described in degenerative spinal disease following a single act of facet distraction points towards the site of initiation of the process of degeneration.

The technique of facet distraction involves opening of the joint, denuding of the articular cartilage, introduction of bone chips within the articular cavity, and impaction of the Goel facet spacer. The adjoining posterior surfaces of the laminae of the spine are widely decorticated, and bone graft harvested from the spinous processes or from the iliac crest is placed in the region and forms an additional ground for bone fusion and arthrodesis.

7.14 Only Fixation as Method of Treatment

As we mature further in the understanding of spinal degeneration, we realize that spinal stabilization alone without distraction can be a rational form of treatment [45, 46]. Identification of the unstable spinal segments and their stabilization can form the surgical treatment for single- or multiple-level radiculopathy or myelopathy. This understanding is based on realization that more than neural deformation or compression, it is repeated microtrauma or injury to the spinal cord related to instability that is the cause of symptoms of radiculopathy and myelopathy [48]. Long-term deformation or compression of the neural structures is well tolerated. This fact can be observed in cases with benign spinal tumors and syringomyelia that develop over long periods, and the reduction in cord girth is surprisingly well tolerated by the patient [37]. We resorted to transfacetal or transarticular Camille's technique of screw insertion in the affected spinal segments and identified this as a more effective, safe, and rather simple surgical procedure [56, 57] (Fig. 7.9). Insertion of the screw into the strongest part of the spinal segment provides for firm stabilization of the region with possibility of "zero" movement and a ground for solid arthrodesis. Real-time identification of unstable joints by direct inspection and their stabilization even in the absence of their radiological demonstration can lead to effective treatment of spinal instability. Identification of the level of unstable segment is done by clinical and radiological guides but is finally confirmed by direct visual observation of the facets and by manual manipulation of bones of the region.

7.15 Association of Atlantoaxial Instability in Cervical Spinal Degeneration

Cervical spondylosis is usually considered to involve only the lower cervical vertebral levels and less commonly upper cervical levels. Atlantoaxial joint degeneration is seldom associated with cervical spondylosis. Whilst the special atlantoaxial joint structure facilitates performance of circumferential movements, it also makes it more susceptible to instability. It seems that the instability of the atlantoaxial joint may even be the primary site of degeneration that may be manifested radiologically at the subaxial spinal levels. Instability of the atlantoaxial joint can be identified by direct observation by manual handling of the bones during surgery or can be evaluated by radiological demonstration of facetal malalignment on lateral profile imaging in neutral spinal position.

In chronic degenerative changes, atlantoaxial instability is more often of central or axial type (CAAD). Atlantoaxial instability is more often associated in cases with multisegmental cervical spondylotic disease and particularly when the myelopathy is "severe" [58–61]. Ignoring atlantoaxial instability whilst treating subaxial spinal

degeneration can be a major cause of failure of treatment or can be associated with a poor surgical outcome. A modified form of atlantoaxial fixation involves sectioning of the muscles attached to the C2-spinous process and C2–3 transarticular fixation [62]. The technique allows rotatory movements executed by the muscles attached to the transverse process of the atlas and fixates anteroposterior movements, particularly those that involve the odontoid process.

The treatment of acute and chronic, single- or multiple-level spinal spondylotic disease presenting with radiculopathy and/or myelopathy is thus focused on stabilization of the affected spinal segments. The spinal bones are used for arthrodesis of segments, and their removal for "decompression" in the presence of spinal instability can only have negative consequences. All the so-called pathological "compressive" entities are secondary, naturally protective, and reversible after spinal stabilization surgery. Disc herniation, or prolapse, can regress and resorb, osteophytes can reverse, ligamentum flavum bulge can disappear, disc space height can recover, and there is a potential for bone fusions to un-fuse.

It does seem that a ground has been laid for relegating surgery of spinal canal and foraminal decompression by laminectomy or laminoplasty and corpectomy-discoidectomy into realm of history [63]. The validity and need for osteophyte resection, removal of disc, resection of ligamentum flavum, and enlarging the spinal canal dimensions by laminectomy/laminoplasty/corpectomy that forms the current basis of surgical treatment of degenerative spine can be questioned. Surgical processes that enhance the fixation and arthrodesis should be appropriately adopted in the treatment. It is important to identify the levels that need stabilization, and atlantoaxial joint should not be ignored when treatment is planned and executed. It must be realized that the spinal levels that appear to be affected on radiological imaging may not be the only segments that are actually unstable. Manual physical and visual analysis of stability of the bones of the region on the basis of clinical and radiological guides can have a major impact on guiding the surgeon on the number of spinal levels that need fixation. The concept that there can be instability without any radiological demonstration can expand the scope of surgery.

Ossification of posterior longitudinal ligament (OPLL) and cervical myelopathy: Our studies have identified that like osteophyte formation, OPLL is a manifestation of unstable spinal segment [64–67]. Instability of the spinal segment initiates the process of abnormal ossification. Atlantoaxial instability is frequently associated with subaxial spinal instability in cases with OPLL. The pathogenesis of both spinal degeneration and of OPLL is related to subtle and long-standing spinal instability.

The pathogenesis of **Hirayama disease** has been under discussion. Compression of neural structures by unusually formed dural band has generally been identified to be the causative issue. Our studies have identified multisegmental cervical spinal instability that generally includes CAAD to be the point of pathogenesis of Hirayama disease. Spinal stabilization rather than spinal decompression appears to be a rational form of surgical treatment [68].

7.16 Conclusions

Essentially, our studies identify the validity of spinal stabilization and futility of any form of spinal decompression in cases with radiculopathy and myelopathy for single- or multi-level spinal degeneration. Muscle weakness-related instability of the spinal segments is the cause of spinal degeneration, and correct identification of unstable spinal segments and their strong stabilization form the basis of surgical treatment.

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