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Adjacent Level Degeneration After Anterior Cervical Fusion: A Clinical Review

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Anterior cervical discectomy and corpectomy for the treatment of cervical spondylosis, cervical disc herniation, and ossification of the posterior longitudinal ligament enjoy favorable rates of fusion and successful clinical outcomes [1–8]. Although the complications from these procedures have been well described, the pathogenesis and clinical development of adjacent segment degeneration (ASD) are not fully understood. Intuitively, the mechanical and shearing motion exerted by the lever moment at adjacent levels after fusion should advance local degenerative changes. Few large series on the treatment of cervical spondylosis with fusion have a long enough follow-up to address ASD, however [1,6,9–14].

Radiographic evidence of degenerative changes in the spine does not necessarily correlate with clinical symptoms of ASD [15]. Several reports have shown radiographic development of degenerative changes in normal patients as well as in patients treated with fusion with little or no clinical symptomatology [16,17]. Therefore, the definition of symptomatic ASD is the development of radicular or myelopathic signs and symptoms referable to a motion segment adjacent to prior cervical arthrodesis [12]. Throughout this article, radiographic evidence is differentiated from clinically symptomatic degenerative changes after fusion. The incidence, pathogenesis, prevalence, and potential treatment strategies for symptomatic ASD are also discussed.

Incidence and prevalence of adjacent segment degeneration

Radiologic evidence of adjacent segment degeneration

Radiologic evidence of ASD can manifest as anterior or posterior osteophytes, disc degeneration, facet hypertrophy, spinal canal stenosis, or segmental hypermobility. Several radiologic reports have addressed the development of degenerative changes after cervical fusion. Hunter and colleagues [18] retrospectively analyzed 9 of 49 patients who underwent fusion for nontraumatic reasons between 1959 and 1971, with a minimum follow-up of 7 years. Of these 9 patients, 8 developed radiologic signs of degeneration in the segments above and below the fusion. However, no clinical information regarding symptoms was reported.

Dohler and coworkers [19] assessed the development of hypermotility at adjacent segments after anterior interbody fusion in 21 patients. Of these, 14 had radiologic evidence of translatory displacement over an average follow-up of 27 months. None of these patients underwent a second surgical procedure. Moreover, there was no correlation between the presence of hypermobile segments and the development of clinical symptoms.

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Baba and colleagues [9] reported on a large series of patients who underwent a single-level, double-level, or multilevel discectomies for myeloradiculopathy, with an average follow-up of 8.2 years. Dynamic measurements on flexion and extension radiographs and the degree of spinal canal stenosis were compared for each patient before and after surgery. Of 106 surgically treated patients, 26 developed new radiologic signs of spinal stenosis at adjacent levels. Hypermotility at adjacent levels, measured by the tilting angle of the disc, and dynamic spinal canal stenosis were statistically significant. Most of these changes involved the upper adjacent level.

Goffin and coworkers [20] studied the development of ASD in posttraumatic patients who underwent fusion with anterior plating. Over a mean follow-up of 7 years, 15 (60%) of 25 patients developed radiologic evidence of degenerative changes. These changes were considered mild in 7 of the 15 cases, and the other 8 cases were considered to be moderate to severe. None of these changes manifested clinically with radiculopathy or myelopathy, and thus were not treated surgically. These investigators reported that the propensity for ASD to develop was increased in patients with multiple fused levels and fusion of lower cervical segments.

MRI has improved the anatomic definition of degenerated disc disease and spinal canal stenosis. Surprisingly, only a few series have used MRI to assess ASD after fusion. Ross and colleagues [21] reported degenerative changes in 40 of 73 patients after cervical surgery. No preoperative studies were included for comparison, however, nor was a control group available to assess degenerative changes in those patients who did not undergo surgery. As this study exemplifies, a limiting factor in the available radiologic series is the lack of control groups to assess for degenerative changes in a similar cohort of patients.

Wu and coworkers [17] used MRI to assess the development of degenerative changes in surgical patients and those patients not undergoing surgery. There were 68 patients in the control and surgical groups. Patients in the control group had no history of cervical symptoms and underwent follow-up MRI after 3 years. The surgical group underwent a single- or a double-level discectomy with fusion. Postoperative MRI scans were obtained after 37 months. In the surgical group, the incidence of degenerative findings at the levels immediately adjacent to the fusion was higher than in those patients not undergoing surgery. The authors concluded that cervical fusion was associated with degenerative changes at the levels immediately above and below a fused segment.

In a similar study using a control group and a fused group, Gore and colleagues [16] employed cervical radiographs to assess the development of degenerative changes in surgical and asymptomatic nonsurgical patients, with an average follow-up of 5 years. The only significant change was the development of anterior osteophytes at the adjacent upper level (C4-C5) in patients who underwent C5 to C7 or C5 to C6 fusion and at the adjacent lower level (C6-C7) in patients who underwent C5 to C6 fusion. The development of degenerative changes over the observed period was similar in both groups. The authors concluded that fusion does not accelerate degenerative changes at adjacent levels.

In contrast, Cherubino and coworkers [10] reported radiographic degenerative changes (eg, decreased disc height, osteophyte formations in segments adjacent to prior fusions in surgical patients) over a mean follow-up interval of 11.5 years. These changes were significantly different compared with those in asymptomatic patients. There was no association between the degree of degeneration and the development of clinical symptoms.

Clinical evidence of adjacent segment degeneration

Radiographic evidence of ASD does not necessarily correlate with clinical manifestations of spondylosis. Several series have studied the prevalence of radiologic degenerative findings in subjects with no evidence of cervical myeloradiculopathy [15,22,23]. Major degenerative changes, such as osteophyte formation, disc abnormalities, foraminal encroachment, and spinal stenosis, have been observed in as many as 19% of asymptomatic patients. These changes in asymptomatic patients become more apparent and have a higher prevalence as age increases. Boden and colleagues [15] reported a 28% rate of significant degenerative changes in patients older than 40 years, whereas Matsumoto and coworkers [23] reported that 89% of patients 60 years and older had evidence of degenerating disc disease. Series with long follow-up periods report late clinical failures related to ASD after anterior decompression and fusion [1,9,11–14,20,24,25].

In their series of 253 patients, Lunsford and colleagues [13] operated on 17 (6.7%) patients for

symptomatic development of ASD. Unfortunately, they were unable to comment on the radiologic incidence of ASD in their population.

Gore and Sepic [11] reported that delayed pain recurred in 23 of 146 patients who underwent anterior discectomy and fusion after an average postoperative interval of 2 years. Of these 23 patients, 18 (12%) had radiologic evidence of degeneration at levels other than the fused levels. Eleven (7%) of these 18 patients required operative decompression for symptomatic relief.

Over a mean follow-up of 8.5 years, Baba and colleagues [9] showed that 26 (25%) of 106 patients with prior cervical fusion developed radiographic evidence of ASD. Only 17 (16%) of the 106 patients required surgical intervention.

Yonenobu and coworkers [25] compared the early and late onset of neurologic deterioration after surgical decompression for the treatment of myelopathy in 110 patients. In the early-onset group, 4 patients deteriorated neurologically as a result of ASD within 6 months of their first operative decompression. Retrospective radiographic analysis of these early failures showed that mild degenerative changes of the involved levels were visible on the preoperative myelogram. Late neurologic deterioration, ranging from 3 to 5 years, occurred in 4 patients with ASD. Of the 8 patients who developed ASD, only 2 required a second surgical decompression. The other 6 patients were treated conservatively. Given the presence of preoperative degenerative segments adjacent to the planned fusion, these investigators recommended addressing suspicious levels to avoid the development of clinically symptomatic spondylosis [25].

In a similar study investigating the causes of failure after anterior decompression and fusion, the incidence of ASD over an average follow-up interval of 6 years was 2.3% (11 of 443 patients) [14]. In this study, 8 (72%) of 11 patients who developed ASD had single-level fusion, whereas the other patients had multilevel fusions (two levels in 2 patients and three levels in 1 patient).

Hubach [24] used preoperative discography to identify symptomatic degenerative discs. The first 23 patients did not undergo discography; 8 of these patients (35%) developed clinically symptomatic ASD after cervical fusion. In all subsequent patients, he performed discography and fused all discs that had positive discograms. Treating positive discs reduced the incidence of ASD to 12%, and only 6 patients (3.5%) required a second surgical decompression at adjacent levels.

Geisler and coworkers [5] retrospectively reviewed 402 patients treated at two separate institutions with anterior decompression and fusion. Although their follow-up was relatively brief (mean of 3.8 years), 8 of 22 patients who required a second operative procedure had clinically symptomatic ASD. All these patients were initially treated without anterior cervical plating. Most of the degenerative changes occurred in the lower cervical levels after initial fusion.

Other large series of anterior arthrodesis have briefly alluded to the development of ASD with an incidence ranging from 2% to 4% [2,3]. Interestingly, in a series of posterior laminoforaminotomies, the incidence of ASD was 3.9% per year [26]. As discussed elsewhere in this article, this observation raised the question of the role of surgery in the development of ASD [12].

Based on their large clinical experience, Bohlman and colleagues [1] and Hilibrand and coworkers [6,12] addressed the incidence and development of ASD. In the initial study [1], they retrospectively evaluated 122 patients who underwent anterior decompression and fusion. Eleven (9%) patients developed ASD and required surgical intervention. The average time between the two surgeries was 5 years [1]. Subsequently, this group [12] retrospectively reviewed 374 patients who underwent an anterior cervical discectomy or corpectomy associated with various forms of postoperative immobilization. Fifty-five patients developed radiographic and clinical evidence of ASD over follow-up, with an annual incidence of 2.9%. Twenty-seven patients were treated surgically. The prevalence of ASD at 5 and 10 years was 11.2% and 19.2%, respectively. Using Kaplan-Meier survivorship analysis, these investigators predicted an 86.4% and 74.4% disease-free survival rate after 5 and 10 years, respectively. The levels with the highest relative risk for developing ASD were C5 to C6 and C6 to C7, with a prevalence of 13.8% and 13%, respectively. The next levels at risk for developing ASD were C4 to C5, followed by C3 to C4, with a prevalence of 9.3% and 7.6%, respectively. Of the 55 patients who developed ASD, the average time of onset depended on the preoperative radiologic conditions of the adjacent discs. For patients with no evidence of degenerative changes, the average time of onset was more than 7 years. In contrast, in patients with severe changes as manifested by spinal cord compression and loss of disc height, the average onset was less than 2 years. Most of the patients with advanced

degenerative changes were older. Thus, age at surgery correlated significantly with degree of degeneration at the adjacent segment. The incidence of ASD was highest in patients who underwent single-level arthrodesis compared with those with multilevel arthrodesis.

Cervical biomechanics and pathophysiology

In vitro studies have addressed compensatory mechanisms seen at adjacent segments after selective experimental arthrodesis. These studies are flawed by different selective mechanical paradigms and anatomic variations within their models. The principles of share loading and compensatory segment motion demonstrated in in vitro studies are widely applicable to clinical conditions, however, and might help to clarify the mechanisms responsible for the pathogenesis of ASD.

Another limitation in the literature exploring biomechanical models of cervical ASD is the mix of lumbar and cervical in vitro studies and their extrapolation to clinical scenarios involving cervical disc disease. The cervical and lumbar regions have distinct physiomechanical loads and demands. Therefore, this discussion concentrates on in vitro cervical studies and avoids intermixing biomechanical studies involving the lumbar and cervical regions.

In the lumbar spine, segmental arthrodesis significantly increases stiffness at the fused level, with concomitant increases in force and motion at adjacent levels [27,28]. Finite element analysis of fused segments in the lumbar region also has shown increased intradiscal pressures at adjacent levels during normal load testing [29]. The combination of increased intradiscal pressure and increased forces at adjacent segments has been postulated as being responsible for hypermobility and for accelerating degenerative changes in the pathogenesis of lumbar ASD.

In 101 cadaveric cervical specimens, Eulderink and ten Have [30] studied compensatory mechanisms of levels adjacent to hypomobile segments. As the stiffness of a level, particularly C5 to C6, increased, there was a compensatory decrease in motion at the immediately adjacent levels with associated degenerative changes. These degenerative changes may have accounted for the decrease in motion but were not significant when compared with other normal segments, perhaps reflecting the general state of the spine specimens.

In the cervical spine, several biomechanical studies have addressed adjacent segment changes after in vitro arthrodesis. Fuller and colleagues [31] simulated one- to three-level fusions on C2 to T1 cadaveric specimens and assessed biomechanical behaviors at adjacent nonfused segments. When C2 to C5 was fused, there was statistically less motion at C5 to C6 compared with increased motion at the C6 to C7 and C7 to T1 levels. Similarly, when C2 to C4 was fused, the increase in motion at C4 to C5 was statistically less than the increase at C7 to T1. For the other fusions performed (C2-C3, C7-T1, C6-T1, and C5-T1), there were no significant increases in angular sagittal displacement in any of the nonfused segments. Applying forced motion through fused segments in the cervical spine resulted in equal and shared distribution of applied moments throughout all nonfused segments. This load-sharing phenomenon was most evident when lower cervical segments were fused. With the equal distribution of loads adjacent to fused segments, Fuller and colleagues [31] concluded that fusion does not necessarily lead to, or accelerate in a disproportionate fashion, degenerative changes at segments immediately adjacent to the fusion. Although they did not evaluate alterations in axial rotation, Fuller and colleagues [31] also pointed out that vertebral axial rotation shifts may be responsible for changes in load sharing after fusion. Higher loads may now be concentrated over smaller areas, precipitating degenerative changes over time.

Local segmental changes after fusion and their potential role in the cervical development of ASD have been partially addressed by other studies [32,33]. Schulte and coworkers [33] performed a one-level fusion (C5-C6) with bone graft with and without a plating system and analyzed motion changes at adjacent levels. Applying pure moments to their construct reduced motion at the fused level by approximately 49% during extension. Similar reductions occurred during lateral bending and axial rotation. With a plating system, all load motions at the fused level were reduced approximately 70%. At adjacent segments, there was a slight increase (15.8%) in extension at C4 to C5 without the plating system. When the construct was supplemented with a plate, extension (29.3%) at C4 to C5 and flexion (18.6%) at C6 to C7 decreased. Otherwise, there were no significant changes in motion at adjacent segments during any of the other load modalities.

Using finite element analysis, Maiman and colleagues [32] performed superior (C4-C5) and inferior (C5-C6) fusions using a variety of techniques and graft materials to assess construct

stiffness and adjacent motion changes. They reported significant changes in the stress on the C4 to C5 disc and C5 vertebral body during compression when C5 to C6 was fused. They concluded that these stress changes in load sharing after inferior fusion might account for clinical and radiographic observations of degenerative changes observed at immediate adjacent levels. Some of the limitations of these studies are the inability to account for the stability afforded by the neck musculature and the inability to assess for true physiologic bony fusion. Moreover, these studies did not perform fatigue loading, which could eventually increase laxity at adjacent fused levels with compensatory osteophyte formation as occurs in the clinical setting. Clinical and biomechanical evidence suggests that the cause of ASD could be multifactorial, with biologic and mechanical processes acting together [31,34].

Predisposing factors

Several factors might aid the treating physician in determining which patients are at risk for developing ASD. One of the most consistent predisposing factors for ASD development in series of anterior cervical fusion is the presence of degenerative changes at adjacent segments before fusion [1,3,9,12,24,25]. In most series of anterior cervical fusion performed to treat cervical spondylosis, the lower segments (ie, C5-C6, C6-C7) are typically the ones to degenerate and to require subsequent surgical treatment. These lower levels may be vulnerable because of their increased segmental motion and loading demands [12,34,35].

In their large series, Hilibrand and coworkers [12] reported that fusions ending at C5 or C6 were at higher risk of developing ASD than fusions ending at other levels. They also concluded that discs associated with some degree of degeneration before fusion were at significant risk of developing clinically symptomatic ASD during follow-up. Several authors have reported similar observations linking ASD to preoperative degenerative changes in the lower cervical segments [1,3,5,25].

Age has also been considered a risk factor for the development of ASD. In most series addressing cervical spondylosis, the patient population is usually older than 50 years. As mentioned, increased age is associated with degenerative changes on cervical spine and routine MRI. After cervical fusion, the combination of increasing age and preexistent degenerative changes sets the stage for the early development of ASD [12,34]. Despite these observations, development of ASD is not only limited to the older adult population. Goffin and colleagues [20] reported ASD in 25 patients, 60% of whom were younger than 30 years. These patients underwent cervical fusion for fractures or dislocation of the cervical spine. Fifteen patients were found to have radiographic evidence of ASD over a follow-up period of 5 to 9 years [20].

Conceptually, one would think that the increased stiffness over longer segments as found in multiple-level fusions would serve as a risk factor for the development of ASD. The contrary has been reported, however. In a series reported by Hilibrand and colleagues [12], the risk of symptomatic ASD was highest in patients who underwent single-level fusion. Eighteen percent of patients who underwent single-level arthrodesis developed ASD compared with 12% of patients who underwent multilevel arthrodesis.

Shinomiya and colleagues [14] also revealed that the incidence of ASD was highest in patients who underwent single-level fusion. A possible explanation for this phenomenon is that the segments at risk were incorporated in the multisegment arthrodesis, minimizing the future development of ASD. Longer follow-up is needed to confirm this notion, however.

The surgical approach used for the treatment of cervical spondylosis seems to have no influence on the development of ASD. Henderson and coworkers [26] performed 846 discectomies through a posterior laminoforaminotomy in 736 patients. They reported a reoperation rate of 10.6% at a new or contralateral segment after 3 years of follow-up. Similarly, Lunsford and colleagues [13] found no significant difference in the development of cervical radiculopathy in patients who underwent an anterior cervical discectomy with or without arthrodesis.

Based on radiographic evidence as well as on clinical evidence of ASD, several authors have speculated that the development of clinically symptomatic ASD is unrelated to surgical intervention [10,12,16,26]. Most of the patients who undergo treatment of cervical spondylosis have degenerative changes throughout their spine. It is enticing to speculate that the clinical development of ASD is a consequence of the natural history of their condition.

Other factors have been implicated in the development of ASD. Dissection and retraction

of the longus colli muscles in conjunction with localization of the disc segment during anterior cervical spine surgery might serve as irritants to ligamentous structures. Ligamentous alterations might lead to local changes in load sharing that affect the dynamic stability of the column [20,34]. Anterior cervical plating provides a significant amount of stiffness not only at the level fused but at adjacent levels [28].

The use of an anterior plating system has not been shown to influence the development of clinically symptomatic ASD significantly, however. In their series of 402 patients, Geisler and coworkers [5] showed that in 8 of the 365 patients who required surgical intervention for ASD, none had undergone anterior cervical plating. In contrast, Goffin and colleagues [20] found that 15 of 25 patients with anterior cervical plating had evidence of radiographic ASD with no need for surgical intervention. With the increasing use of anterior cervical plating, more information should be gained as this clinical entity becomes better recognized and longer follow-up is obtained.

Diagnosis and treatment

The diagnosis of ASD must be based not only on radiographic evidence but, more importantly, on clinical signs and symptoms of radiculopathy or myelopathy. All patients suspected of cervical spondylosis as a result of ASD should undergo careful radiologic and neurologic evaluations. A detailed physical examination should include cervical range-of-motion and provocative maneuvers, such as the Spurling's test, to assess for evidence of radicular impingement. Sensory and motor examinations should be tailored to detect evidence of dysfunction in a dermatomal distribution. Particular attention should be paid to manifestations of myelopathy, such as Hoffman's sign, hyperreflexia, or Babinski's reflex.

In patients with progressive pain, it is important to determine whether it is axial or radicular. In patients with axial pain, evidence for pseudarthrosis at the site of prior fusion should be evaluated carefully. To do so requires plain radiographs to identify bone graft resorption or progressive loss of graft height. Dynamic films, such as flexion-extension radiographs, may provide subtle evidence of instability at the fusion site, increased motion at adjacent levels, or both. In patients with radicular symptoms, careful evaluation is needed to assess concordant physical and radiographic findings. In the absence of pseudarthrosis, if the physical signs and symptoms correlate with the fused level, careful evaluation of imaging studies may reveal evidence of inadequate decompression or of the formation of posterior osteophytes. Other sources for radicular symptoms include brachial plexus pathologic findings and nerve root entrapment syndromes.

Patients who develop ASD from prior fusion usually develop radicular symptoms above or below the fused level that manifest as pain with or without associated sensory and motor disturbances. Radicular pain is often relieved by distraction maneuvers and exacerbated by extension or axial loading. Central disc degeneration with central spinal cord compression and stenosis might be associated with signs of upper motor neuron lesions, including spastic gait, bowel and bladder difficulties, and hyperreflexia.

Imaging studies should include plain, lateral, and anteroposterior (AP) radiographs with flexion-extension views to assess for dynamic instability at adjacent segments. MRI, the study of choice in most patients, can show evidence of central or lateral stenosis (Fig. 1). It also can provide a good assessment of the integrity of the fusion and adjacent segments. Occasionally, CT myelography is obtained to define the bony anatomy and any relation to the nerve root foramen in question. CT myelography is helpful when prior hardware causes scatter artifact that makes MRI difficult to interpret. It is crucial to assess the concordance of imaging studies and the subjective and objective clinical signs and symptoms. In some instances, electromyographic and nerve conduction studies are useful adjuncts to determine and assess objective deficits in dermatomal distributions.

The management of patients with ASD follows the same principles as that of patients with cervical spondylosis. Before surgical intervention, an aggressive regimen of conservative therapy should be instituted. Close follow-up is needed to document any improvement or deterioration in neurologic function. Early surgery should be considered in patients who develop acute or subacute onset of motor deficits, persistent disabling pain, progressive kyphotic deformity, myelopathic signs and symptoms, or clinical instability.

An aggressive course of conservative management should include the use of nonsteroidal antiinflammatory drugs and analgesics. In the event of acute radicular flare-ups, a short course of oral

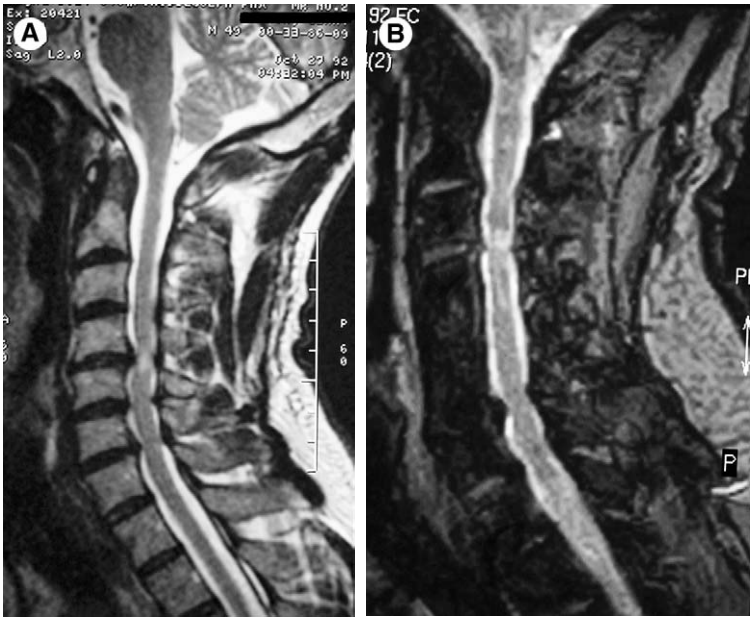

Fig. 1. (*A*) In 1992, a 57-year-old man with myelopathic findings underwent C4 to C6 fusion and his symptoms improved. (*B*) In 2000, he returned with neck pain and problems with hand coordination.

corticosteroids may be recommended to alleviate and control disabling pain. A soft or hard collar might be used to control motion-related exacerbations. Once the acute symptoms are under control, specific physical therapy measures can be implemented. Swimming, walking, and isometric strengthening exercises are usually well tolerated and serve to strengthen the spinal musculature. Epidural or periradicular injections can serve as adjuncts for treating patients with persistent radicular pain. Conservative management for radiculopathy caused by a herniated cervical disc is associated with favorable success rates [36]. Little information is available on the conservative treatment of patients suffering from ASD, however. Hilibrand and colleagues [12] reported that of the 46 patients who developed ASD in their series, only 13 responded to nonoperative conservative measurements. Of the remaining 33 patients, 6 deferred surgical treatment, whereas the other 27 underwent an operative treatment. They suggest that in patients with ASD, nonoperative measurements may not be as effective as in patients with the new onset of radiculopathy without prior fusion. Similarly, Gore and Sepic [11] reported that of the 18 patients who had clinical ASD in their series, only 11 underwent additional surgery.

Surgery should be entertained in patients who fail conservative management and have concordant findings on physical examination and diagnostic imaging. Series treating clinically symptomatic ASD have used an anterior or posterior surgical approach. Unfortunately, given the small numbers of patients who are treated for ASD, the efficacy of these two approaches has not been compared. Consequently, patients' symptoms and imaging findings must be assessed carefully to ascertain the most appropriate approach to treatment.

When a unilateral cervical herniation impinges on the neural foramen, a laminoforaminotomy may be a surgical option. A keyhole laminoforaminotomy offers the advantage of performing selective decompression with minimal surgical morbidity and no need for fusion. A disadvantage of this approach is the difficulty in visualizing, and therefore in performing, the decompression when the symptoms are caused by an osteophyte. Contraindications to laminoforaminotomy include the presence of a kyphotic deformity or radiographic signs of segmental instability that might be exacerbated by the procedure.

A laminectomy also might be a viable treatment for ASD. Some people argue that removing the posterior tension band through a laminectomy

in a biomechanically compromised segment might exacerbate instability and thereby lead to further neurologic deterioration. To prevent this possibility, a laminectomy can be supplemented by lateral mass plates that incorporate the fused segment(s). Laminoplasty also might be a viable option when stenosis is present at or below a fused level or when more than three levels are involved. An advantage of laminoplasty is that the facets are preserved. Therefore, the chances of segmental instability and development of kyphotic deformity are decreased or minimized. Contraindications to laminoplasty and laminectomy include significant anterior spinal cord compression or the presence of a kyphotic deformity.

Most authors who have addressed the surgical management of ASD recommend anterior decompression and subsequent fusion of the newly involved segments (Fig. 2). An anterior approach allows full visualization of the segment(s) involved

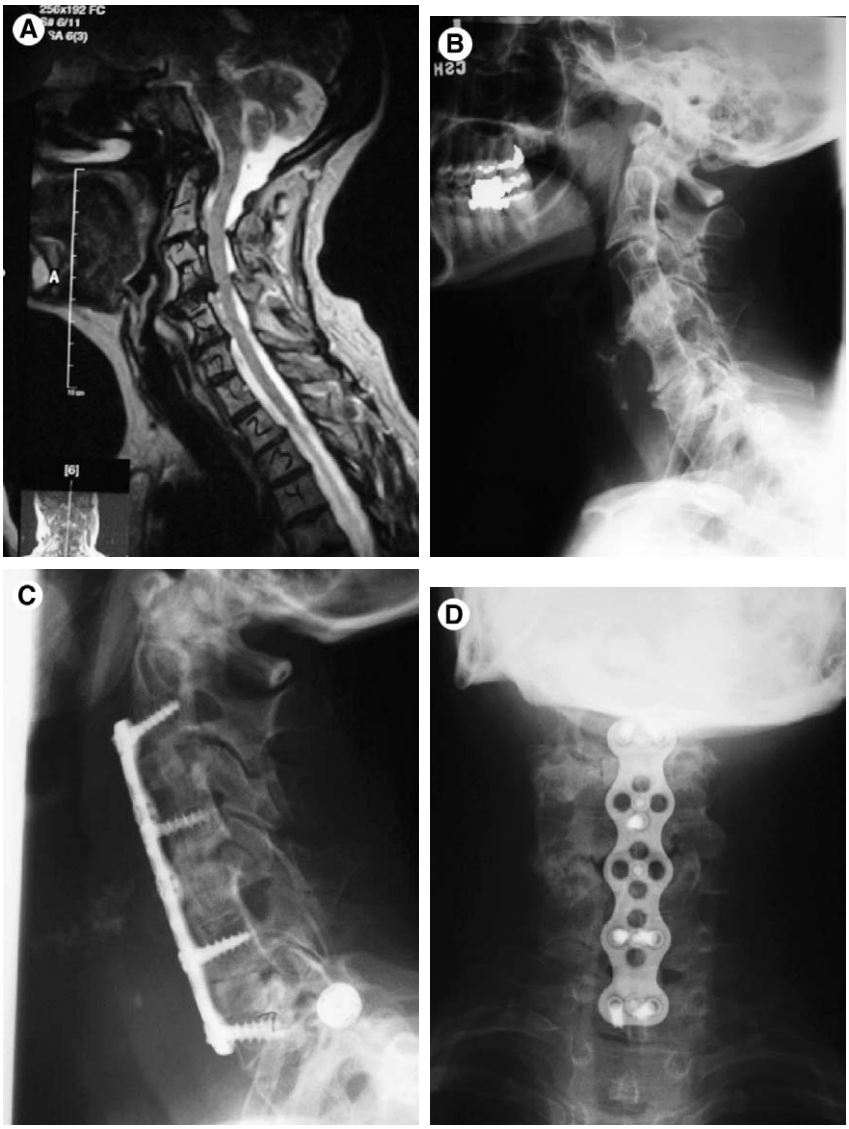

Fig. 2. Preoperative MRI scan (*A*) and lateral radiograph (*B*) of a myelopathic 47-year-old patient with Klippel-Feil syndrome with congenital C4 to C5 and C6 to C7 fusion and severe adjacent level degeneration. Postoperative lateral (*C*) and sagittal (*D*) anteroposterior radiographs are shown.

and allows safe decompression of the spinal cord from one foramen to the other. Placement of a strut graft allows intersegmental distraction, and therefore widening of the neural foramen. Conceptually, the failure mode underlying ASD usually involves the anterior column. A direct approach therefore eliminates the pathologic findings and provides segmental stabilization.

Disadvantages of anterior cervical decompression and fusion include the risk of graft dislodgment, pseudarthrosis, pain at the bone graft donor site, and the propensity to develop ASD at other levels. In patients with prior fusion, these risks are much higher because of the presence of scar tissue. Reoperation in such patients makes the dissection of soft tissue extremely hard and increases the risk of esophageal perforations or vessel injury. Moreover, dissection through prior scar tissue alters the local blood supply, hindering the development of solid arthrodesis.

Long-term follow-up of patients who have undergone treatment for ASD is lacking. Baba and colleagues [9] operated on 17 patients who developed ASD. They performed anterior fusion in 2 patients and an open-door expansive laminoplasty in the other 15 patients. They did not specify which grouped improved, but they reported an overall improvement rate of 51%: four excellent outcomes, six good outcomes, three fair outcomes, and four poor outcomes. Shinomiya and coworkers [14] operated on 11 patients with ASD, 2 anteriorly and 9 posteriorly with laminoplasty, and reported 100% improvement as measured by the Japanese Orthopedic Score for Cervical Myelopathy.

Hilibrand and colleagues [6,12] reported two studies on their experience with treating ASD and arthrodesis adjacent to a prior fusion. In their 1997 article [6], 38 patients who had undergone prior fusion were evaluated for the development of symptomatic ASD. All 38 patients underwent surgical intervention (24 discectomies and 14 corpectomies). All patients received autologous strut grafts and were immobilized with rigid orthosis for 6 weeks or with a halo in patients who underwent a three-level corpectomy. The fusion rate in patients who underwent a discectomy was 62.5%, and it was 100% in patients who underwent a corpectomy. There was no difference in the rate of arthrodesis between single-level and multiple-level procedures. Of the 24 patients in the discectomy group, 20 reported an excellent or good outcome and 4 reported a fair outcome. In the 14 patients who underwent a corpectomy, 12 reported an excellent to good outcome and 2 reported a fair outcome. There were no poor outcomes in this series.

In a prior series from this group, the successful rate of arthrodesis was 89% for single-level fusions and 65% for multiple-level discectomies [1]. The lower rate of arthrodesis in the series by Hilibrand and colleagues [6] illustrates that biomechanical factors adjacent to fused levels interfere with biologic factors responsible for achieving solid arthrodesis.

Although it has not been shown clinically, the potential use of anterior cervical plating in patients with ASD might decrease the rate of pseudarthrosis. In a subsequent study by Hilibrand and coworkers [12], 27 patients who failed conservative treatment for ASD underwent anterior cervical decompression and arthrodesis. Of 29 procedures in these 27 patients, 21 were associated with an excellent to good outcome and 8 with a fair outcome. Pseudarthrosis was not mentioned in this group of patients.

As the clinical entity of ASD is increasingly recognized and treated by specialists, more information should become available on the natural history of this condition. Although there are several potential therapies for rescue in these patients, the current literature does not provide enough data to ascertain the best management strategy. Patients with ASD have different biomechanical demands in their fused cervical spine that might hinder the formation of solid arthrodesis. For this reason, it is important to recognize patients who are at risk and to tailor their initial therapeutic strategies to minimize the chance of ASD developing.

Prevention

The prevention of ASD can be divided into two therapeutic strategies: (1) decompression and alleviation of clinical symptoms with segmental motion preservation and (2) elimination of offending pathologic findings (degenerative segment) with or without preservation of segmental motion. The first strategy can be performed by posterior approaches through a laminectomy, laminoforaminotomy, or laminoplasty. The second strategy can be achieved through an anterior cervical discectomy with or without fusion, through an anterior keyhole uncoforaminotomy, or through the use of an artificial intervertebral prosthesis.

Posterior approaches to the spine are effective for the treatment of multilevel cervical

spondylosis. Posterior decompressive procedures have the advantage of preserving segmental motion while preventing fusion. Theoretically, they also minimize the chances of ASD developing. Late neurologic deterioration after decompressive laminectomy has been attributed to kyphotic deformation rather than to ASD [25,37]. Laminectomy has been associated with neurologic deterioration in as many as 25% of patients, presumably from development of a kyphotic deformity [25]. The 13% rate of early transient postoperative radiculopathy has been attributed to spinal cord migration and to traction on the exiting nerve roots [38]. Patients with straight or reverse lordosis of their spine have a higher risk for developing a kyphotic deformity [37,39].

Biomechanically, in vitro studies have revealed that laminoplasty confers a more stable spine compared with laminectomy [40]. This finding is partially related to preservation of the facets and the interspinous ligaments during laminoplasty. Removing as little as 25% of the facets significantly increases cervical motion [40–42]. In patients with unilateral radiculopathy, a keyhole foraminotomy serves as an alternative posterior decompressive procedure. As alluded to earlier, however, this procedure is associated with an annual incidence of ASD of 3.9% [26]. Moreover, a prospective study treating lateral cervical disc herniations through an anterior cervical discectomy with fusion or through a posterior laminoforaminotomy showed that over a follow-up interval of 4.2 years, the rate of excellent to good results was 90% in patients treated anteriorly compared with 75% in those treated posteriorly [43].

Several authors recommend anterior cervical discectomy with fusion of all degenerated segments with a high risk for developing ASD (ie, C5-C6, C6-C7) [12,14]. This recommendation is partially justified by the fact that patients with multiple-level fusion of degenerated segments experience a notable decrease in ASD. Moreover, the incidence of ASD was reduced by using preoperative discography to identify degenerated discs, which were then incorporated into the fusion [24]. In contrast, Clements and O'Leary [3] advocated not operating on adjacent levels if patients had no clinical symptoms attributable to radiographically degenerated discs. This recommendation was made despite their observation of ASD in some patients who had degenerated discs before surgery.

Clearly, every patient with cervical spondylosis needs to undergo a detailed history and physical examination to identify any signs or symptoms attributable to the degenerated cervical discs in question. If the signs, symptoms, and clinical examination are concordant with the radiologic findings, an anterior cervical discectomy with fusion should be performed on the symptomatic segments.

An argument for preventive surgery can be based on young patients with asymptomatic, degenerated discs adjacent to symptomatic ones and a high risk for developing ASD. In this patient population, the risk of developing symptomatic radiculopathy or myelopathy over their lifetime is significant enough that early surgical intervention to treat asymptomatic adjacent discs might be of some benefit and should be discussed with the patient.

Anterior cervical discectomy without fusion is an alternative for the treatment of cervical myelopathy and radiculopathy. Several studies have found no differences in surgical outcomes when arthrodesis is performed during discectomy [13,44,45]. Others suggest that patients who receive intervertebral bone grafts tend to fare better than those who do not [46].

A theoretic advantage of performing an anterior cervical discectomy without fusion is the potential of continued, albeit limited, motion at the operated unfused segments. This motion reduces stiffness at adjacent segments, thus potentially reducing the chances of ASD developing. Fusion rates after anterior cervical discectomy have ranged between 72% and 100% [7,45,47]. The development of kyphosis after anterior cervical discectomy has ranged between 63% and 83% [7,45]. Kyphotic deformity might confer a biomechanical advantage for the development of ASD. In their large series, however, Lunsford et al [13] reported no differences in the development of ASD between patients undergoing anterior cervical discectomy or anterior cervical discectomy with fusion.

In patients with unilateral radiculopathy, other potential preventive measures for treating cervical spondylosis are a modified far-lateral approach and the implantation of an intervertebral prosthesis. Although promising, the available follow-up intervals are too brief to provide conclusive evidence to support the role of these techniques in preventing ASD from developing.

The far-lateral approach to the anterior cervical spine was first reported by Verbiest [48] in 1968. It was later refined by Hakuba [49], who performed a transuncodiscal approach with

mobilization of the vertebral artery laterally and discectomy with and without intervertebral fusion. Through a similar approach, George and colleagues [50] performed a partial oblique corpectomy for the treatment of radiculopathy and spondylotic myelopathy without the need for fusion. Good to excellent results followed in 79% and 85% of patients with myelopathy and radiculopathy, respectively. A complication of the far-lateral approach is the development of Horner's syndrome caused by significant retraction of the longus colli muscles as needed to expose and mobilize the vertebral artery. In the series by George and colleagues [50], 57% of the patients developed transient Horner's syndrome, 9% of which was permanent. Over a mean follow-up of 3 years, three patients required subsequent fusion for segmental instability.

Further modifications have made this procedure feasible without mobilizing the vertebral artery while permitting direct removal of the offending pathologic findings through a keyhole uncoforaminotomy [51,52]. Although the follow-up period is unclear, Jho [51] used this technique to treat cervical spondylotic myelopathy in 14 patients. No vertebral arteries were injured. Furthermore, neither clinical instability nor Horner's syndrome developed after surgery. Similarly, Johnson and coworkers [52] treated 21 patients with cervical radiculopathy through a keyhole uncoforaminotomy, with a follow-up interval that ranged from 12 to 22 months. Symptoms improved or resolved in 91% of patients, whereas 9% had persistent symptoms that required further surgery. No patient developed radiographic evidence of instability or had major complications.

This corridor provides direct access to the offending pathologic findings with minimal ligamentous disruption and with preservation of the intervertebral disc and segmental motion. Intuitively, partial removal of the uncovertebral joint should not confer significant segmental instability that could predispose the patient to develop ASD. Nevertheless, a recent in vitro biomechanical study of sequential unilateral or bilateral uncoforaminectomies reported a decrease in segmental stiffness by 30% and 36%, respectively [53]. Whether this finding is clinically significant remains to be established as this approach gains acceptance in the surgical community.

Ideally, preservation of disc height, replacement of the disc's viscoelastic properties, and maintenance of segmental motion can prevent ASD from developing. The focus of artificial disc development has primarily been directed to the lumbar spine. Design criteria must consider biocompatibility, endurance, normal kinematic range of motion, disc geometry, and long-term osteointegration [54,55]. Materials used for intervertebral artificial discs have included nontoxic and noncorrosive metal alloys, polyethylene polymers, porous fibrous polyfluorocarbons, polymerized milk sugars, silicone rubbers, thermoplastic elastomers, and polysulfone rubbers [55].

The use of artificial disc materials in the cervical spine to prevent ASD has not been well established. Recently, Cummins and colleagues [56] reported the use of an implantable stainless-steel mobile joint for the treatment of severe cervical spondylosis. In 20 patients, 22 artificial joints were placed (range of follow-up: 1–5 years). All except 1 patient had developed ASD from prior fusions. Early in their experience, a significant number of complications resulted from poor screw placement, uniform implant size despite a patient's unique anatomy, and manufacturing failure. These problems led to design modifications and subsequent improvement in technique. Of 18 patients who underwent late radiographic evaluations, the segmental motion and disc height were preserved in 16. In the other 2 patients, the authors attributed the loss of motion to overdistraction. During their follow-up, 16 of 20 patients reported improvement. None developed ASD or were treated for it.

Future modifications of this instrumentation need to address anatomic variability, the implant's high profile, implant osteointegration, and surface contour to improve the integration of the screw implant–to-bone integration [57]. Preliminary studies using second-generation implants have provided promising results associated with significantly fewer complications than implants of earlier generations [58–60]. Although application of intervertebral prosthesis is in its infancy, this technology opens new avenues and holds enormous potential for treating cervical spondylosis while preserving segmental motion. Long-term follow-up should assess its efficacy in the prevention of ASD.

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