

## Complications of the spine in ankylosing spondylitis with a focus on deformity correction

MATTHEW L. MUNDWILER, M.D.,<sup>1</sup> KHAWAR SIDDIQUE, M.D.,<sup>2</sup> JEFFREY M. DYM, M.D.,<sup>3</sup> BRIAN PERRI, D.O.,<sup>2</sup> J. PATRICK JOHNSON, M.D.,<sup>2</sup> AND MICHAEL H. WEISMAN, M.D.<sup>1</sup>

<sup>1</sup>Division of Rheumatology; <sup>2</sup>Institute for Spinal Disorders; <sup>3</sup>Department of Radiology, Cedars-Sinai Medical Center, Los Angeles, California

✓ Ankylosing spondylitis (AS) is a systemic inflammatory disorder with frequent spinal axis symptoms. In this paper, the authors explored the spinal manifestations of AS and its characteristic anatomical lesions, radiological findings, and complications. They also offer a comprehensive report of the medical and surgical treatments with a focus on deformity correction. (DOI: 10.3171/FOC/2008/24/1/E6)

**KEY WORDS** • ankylosing spondylitis • osteotomy • pseudarthrosis

**A**NKYLOSING spondylitis is a systemic inflammatory disease of unknown origin. Affected patients are predominantly but not exclusively male. Chronic inflammatory back pain is the most common presenting symptom and typically develops between the ages of 20 and 40 years.<sup>43,65</sup> Alternating buttock pain caused by sacroiliitis is a less frequent presenting symptom but is often regarded as a classic initial indication of AS.<sup>65</sup> Patients with AS can also have extra-articular manifestations such as ocular, cardiac, pulmonary, gastrointestinal, and renal involvement.<sup>43</sup> These manifestations typically develop after the onset of axial symptoms but on rare occasions can precede them.

A patient's susceptibility to the disease is largely genetically determined.<sup>67</sup> The strong association with *HLA-B27* is well known,<sup>26</sup> and other genetic markers are currently being identified. Apart from *HLA-B27* typing, there are no specific laboratory tests or inflammatory markers that provide diagnostic clues. A pelvic radiograph for the detection of sacroiliitis is usually ordered for diagnostic purposes, but the sensitivity and specificity of this procedure are 70 and 80%, respectively.<sup>60</sup>

Because there is no single suitable laboratory test, clinicians must know as many of the characteristic signs and symptoms as possible to make a diagnosis. Even though AS is a systemic disease, the presenting symptoms, treatment, and morbidity are largely dependent on how the dis-

ease affects the spine. Thus, we believe that a review on spinal disease in AS will be of great value. In this review, we first describe the latest published algorithm to diagnose early disease and the classic inflammatory lesions. We then explore the diseased spine's susceptibility to noninflammatory lesions such as microfractures and deformity. We also describe other sequelae of AS, such as early osteoporosis and CES. Both the medical and surgical approaches to treatment are summarized. There is a special focus on osteotomy techniques. By the conclusion of the article, the clinician should have a better understanding of the diagnostic and treatment possibilities in AS spinal disease.

### Diagnosis of Inflammatory Back Pain and AS

Because AS can markedly respond to the newer biological agents (discussed later), effective treatment of the disease requires early diagnosis. However, the high prevalence of back pain in the general population and the lack of radiographically demonstrated characteristic lesions in early AS often delay recognition of the disease. To make an early diagnosis, it is important to distinguish inflammatory from mechanical back pain on presentation. Factors consistent with inflammatory back pain include morning stiffness lasting longer than 30 minutes, onset of chronic back pain at an early age (before 35 years of age), improvement in pain with physical activity rather than with rest, awakening with back pain during the 2nd half of the night, alternating buttock pain, and a prolonged period of back pain.<sup>48</sup> One factor by itself does not have sufficient sensitivity or specificity to determine if the back pain is inflammatory. Note, however, that in a study of European patients with AS in which only 4 factors were considered, if 2 symptoms

*Abbreviations used in this paper:* AS = ankylosing spondylitis; CES = cauda equina syndrome; DEXA = dual energy x-ray absorptiometry; HLA = human leukocyte antigen; MR = magnetic resonance; NSAID = nonsteroidal antiinflammatory drug; PSO = pedicle subtraction osteotomy; SPO = Smith-Peterson osteotomy; TNF = tumor necrosis factor.

were present, the sensitivity of these factors for the diagnosis of AS was 70.3%, the specificity 81.2%, and the likelihood ratio 3.7.<sup>48</sup> If 3 of 4 factors were present, the likelihood ratio increased to 12.4, specificity increased, and some loss of sensitivity occurred. The exact time period for chronic back pain was poorly defined, but patients were more likely to have AS if the pain persisted for ~1 year.

When back pain is determined to be inflammatory, additional studies can reveal whether AS or another axial spondyloarthropathy is present. Based on published analyses, Rudwaleit et al.<sup>49</sup> proposed an algorithm for the diagnosis of AS. At baseline, a patient with any type of back pain for 3 months has a 5% probability of having an axial spondyloarthropathy. The probability of AS increases to 14% if such pain is characterized as inflammatory. At this point, having inflammatory lower-back pain and radiographic findings consistent with AS equate with a diagnosis of AS. If no typical radiographic findings appear, the patient should be assessed for other features of spondyloarthropathies such as enthesitis (particularly heel pain), dactylitis, uveitis, Crohn disease, alternating buttock pain, psoriasis, asymmetrical arthritis, positive response to NSAIDs, elevated acute phase reactants, or a family history. If the patient has 3 of these features, the probability of axial spondyloarthropathy is 80–90%. If only 1 or 2 factors are present, then the presence of *HLA-B27* raises the probability of spondyloarthropathy to 80–90%; if *HLA-B27* is absent, then the probability is < 10%. Even without any clinical features, the presence of *HLA-B27* increases the probability of a spondyloarthropathy to 59%. At this point, an MR image depicting inflammatory characteristics in the presence of inflammatory back pain increases the probability of AS or another spondyloarthropathy to 80–95%. If *HLA-B27* is present, MR imaging results are negative, and there are no clinical features, then the probability of a spondyloarthropathy is < 15%.<sup>49</sup>

### Anatomical Lesions of the Spine in AS

Although radiographically demonstrated spinal lesions in AS were described in the literature decades ago, confusion still exists over the nomenclature and significance of such lesions. Most lesions in patients with AS can be categorized as either inflammatory or mechanical (noninflammatory). Inflammatory lesions include the historically descriptive Andersson and Romanus lesions, enthesitis, synovitis, enthesophytes, and syndesmophytes. Noninflammatory, or mechanical, lesions include trauma-induced lesions, microfractures, pseudarthrosis, deformity/kyphosis, and infection. Other possible lesions include those resulting from osteoporosis, degenerative spine disease, radiculopathy, spinal stenosis, and CES.

### Inflammatory Lesions in AS

#### *The Historical Andersson and Romanus Lesions*

The first 2 spinal lesions in AS were described by Andersson<sup>2</sup> and Romanus and Yden.<sup>47</sup> On radiographs, Andersson lesions appear as a spondylodiscitis that destroys the central portion of the intervertebral disc and adjacent vertebral body.<sup>29</sup> Romanus lesions are erosive changes at the anterior and posterior vertebral endplates that appear as

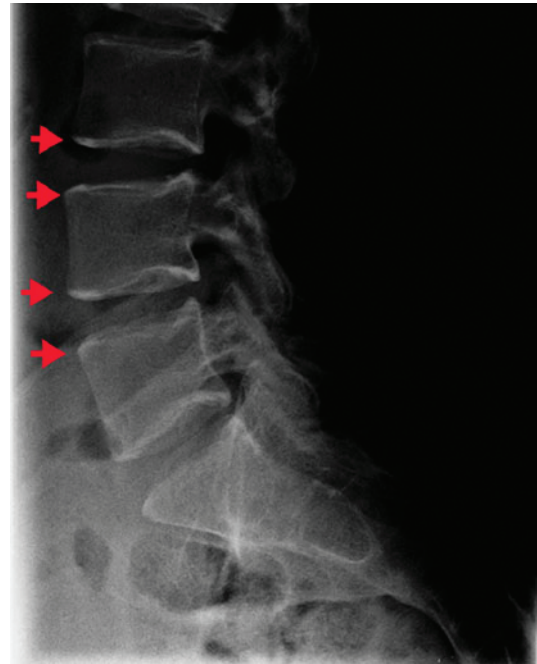


FIG. 1. Radiograph demonstrating the lumbosacral spine in a patient with AS. Arrows indicate Romanus lesions or “shiny corners.”

“shiny corners” on radiographs (Fig. 1).<sup>29,40,47</sup> In an early study cohort, Romanus lesions were present in 53% of patients; Andersson lesions were more rare and occurred in ~ 4%.<sup>54</sup> On MR imaging, short tau inversion recovery sequences show hyperintensity of the involved areas for both lesions, before the radiographically demonstrated changes appear.<sup>29</sup> In late disease, Romanus lesions cause a characteristic squaring of vertebral bodies.<sup>47</sup> Andersson lesions are often mistaken for an infectious process, but cultures and histological studies reveal no causative organism.<sup>15,29</sup>

#### *Enthesitis, Synovitis, Syndesmophytes, and Enthesophytes*

Enthesitis, inflammation of the insertion points of ligaments and tendons that are ubiquitous in the spine, is considered the characteristic inflammatory lesion in AS. Andersson and Romanus lesions result from enthesitis. Synovitis also occurs and affects the zygapophysial, costovertebral, and costotransverse joints. Magnetic resonance imaging reveals enhancement in these tissues when inflammation is present.<sup>29</sup> It is hypothesized that the inflammatory lesions of AS lead to new bone formation as opposed to the erosive changes that occur in rheumatoid arthritis. Ossification occurs in both entheses and synovial articulations.<sup>3,26</sup> The structures resulting from ossification are referred to as “syndesmophytes” or “enthesophytes.” Both share the common histological features of woven bone within a matrix of fibrous tissue. Syndesmophytes form bridging lesions between structures that normally do not connect; enthesophytes simply appear as osseous outgrowths that do not bridge structures.

Syndesmophytes in the spine lead to the permanently restricted motion seen in AS. Only in AS, however, do syndesmophytes cause ankylosis across both zygapophysial

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joints and between vertebral bodies. It is believed that this combination is why patients with the disorder have more spine movement restriction than that seen in other diseases with syndesmophyte formation such as diffuse idiopathic skeletal hyperostosis.<sup>12,13</sup> It has been demonstrated that zygapophysial ankylosis is necessary for bridging syndesmophytes to form between vertebral bodies.<sup>13</sup>

### *Histological Features of Inflammatory Lesions*

Past investigations have revealed that AS lesions in the spine and periphery share a common pathophysiology. Initial biopsy specimens taken from the iliac crest, trochanter, and patella of patients with AS have revealed varying combinations of lymphocytes, plasma cells, and neutrophils at the site of the bone/ligament attachment.<sup>3</sup> Erosion of the cortical bone has also been observed.<sup>3</sup> Biopsy specimens of Andersson and Romanus lesions have revealed a similar process.<sup>15,47</sup> Lesions that progress to ankylosis feature fibrous tissue, cartilage, and reactive bone formation.<sup>7</sup> More recent work by François et al.<sup>19</sup> has shown that inflammation in the subchondral areas of the bone marrow precedes the findings of enthesitis described earlier.

### **Noninflammatory Lesions**

Inflammatory lesions that affect the integrity of the spine are accompanied by other noninflammatory pathological processes. Noninflammatory lesions include those from acute and chronic fractures, deformity/kyphosis, osteoporosis, CES, degenerative disc disease, and spinal stenosis. Although some noninflammatory lesions such as those from stenosis are not specific to spondylitis, such lesions provide unique diagnostic and therapeutic challenges in the patient with AS.

#### *Chronic Microfractures*

Insufficiency fractures, or noninflammatory type Andersson lesions, occur late in the disease.<sup>15</sup> These lesions are thought to occur in areas of the spine that are still capable of mobility when the rest of it is ankylosed. Thus, the spine becomes over-dependent on these sites for movement. These segments are presumably already weakened by chronic inflammatory disease and “forced” to move in ways in which they were not intended. These biomechanical stresses cause microfractures and lead to pseudarthrosis. Repeated micromotion at sites of pseudarthrosis causes progressive osseous destruction. In contrast to the inflammatory lesions that ossify in AS, new bone does not form at these pseudarthrosis sites.<sup>15</sup> Histologically, fibrous tissue is abundant and reflects healing at the site of a microfracture.

#### *Acute Traumatic Fractures*

Although the incidence of traumatic spine fractures in AS has been poorly defined, there is a significantly increased risk compared with that in the general population.<sup>10,63</sup> Approximately 5.7% (prevalence) of patients with AS have reported a history of clinically significant spine fracture, although that number was derived from a self-reported questionnaire and included stable as well as unstable fractures. The lifetime incidence has been reported as

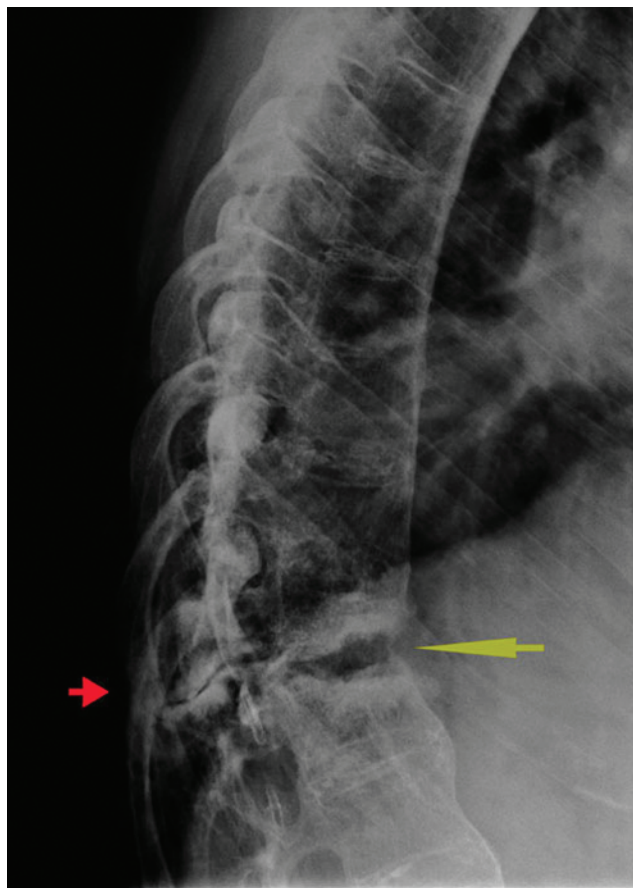


FIG. 2. Radiograph showing a severely ankylosed spine. Large arrow indicates fracture through an ossified disc. Small arrow indicates the extension of the fracture through the posterior elements.

14%, with a range between 4 and 18%.<sup>30,41</sup> The incidence of fractures reportedly increases with disease duration and is typically associated with minor trauma such as ground-level falls.<sup>18,64</sup> Even relatively minor trauma can result in major structural deformity and neurological deficit. Consequent pain can lead to disability, especially if the deformity or pain interferes with physical function. Furthermore, the majority of AS-related spinal fractures are associated with neurological injury. The rate of fracture-related neurological deficit varies from 53 to 83%.<sup>63</sup>

Cervical spine fractures are frequently reported in patients with AS and are more common than thoracolumbar injuries.<sup>46</sup> Typical trauma features include fractures through the ossified disc or an osteoporotic vertebral body with extension into the posterior ligaments (Fig. 2). Most traumatic and pseudarthrosis lesions are centered at the disk space, indicating increased vulnerability of an ossified disc to trauma. This susceptibility to fracture is probably the result of the lower resistance at the disc segment compared to the vertebral body. In addition, these 3-column fractures are typically associated with an extension deformity.<sup>30</sup> Neurological injury can result from buckling of the ossified ligamentum flavum as well as retropulsion of bone and epidural hematomas.<sup>63</sup> Unfortunately, the complication rate after spinal fracture has been noted to be as high as

50%. Weinstein and colleagues<sup>63</sup> reported a mortality rate of 2.8% due to spinal fractures.

#### *Flexion Deformity and Kyphosis*

Even in the absence of trauma, the spine becomes increasingly kyphotic as lesions progress. Objectively, kyphosis is measured as the occiput to wall distance.<sup>57</sup> In a recent study cohort, 49% of the patients with AS had kyphosis.<sup>61</sup> The loss of anterior vertebrae height, wedging of intervertebral discs,<sup>20</sup> disease activity, and the degree of radiographically demonstrated damage<sup>61</sup> have been found to be major contributors to kyphosis. Even though it seems intuitive that anterior wedging leads to hyperkyphosis, patients with anterior wedging from osteoporosis do not have nearly as severe kyphosis as that seen in patients with AS.<sup>20</sup> The likely pathological differences between patients with osteoporosis and those with AS are the enthesitis and synovitis at the zygapophysial joints. The lack of mobility at the zygapophysial joints may limit the spine extension needed to compensate for deformed vertebral bodies.

#### *Osteoporosis in AS*

Osteoporosis of the spine in AS deserves special attention. Although authors of early literature have asserted that osteoporosis is a late disease finding,<sup>54</sup> later studies revealed significant osteoporosis in the lumbar spines of patients with early AS, good mobility, and no peripheral osteoporosis.<sup>66</sup> Thus, axial osteoporosis is linked to early inflammatory events. Other studies have demonstrated that syndesmophyte formation correlates with lower bone mineral density in the spine.<sup>31</sup> Notably, certain things must be considered when assessing osteoporosis in a patient with syndesmophytes. Donnelley and colleagues<sup>16</sup> revealed that syndesmophytes cause overestimation of bone mineral density on DEXA scans in patients with advanced AS. The increased mineral concentration in the syndesmophyte leads to erroneous assessment of spinal osteoporosis. Gilgil and associates<sup>21</sup> showed that the effects of syndesmophytes on DEXA scans can be compensated for by using a lateral method. Dual-energy quantitative computed tomography is also available for measuring bone mineral density and can correlate osteoporosis with disease duration.<sup>31</sup> Thus, it is important to consider the effects of a patient's anatomy on the methods applied when assessing spinal osteoporosis in a patient with AS.

The early osteoporosis that appears in the axial skeleton has clinical consequences. Patients with early AS and mild osteoporosis have a fracture prevalence rate 5 times greater than that in the normal population.<sup>16</sup> Other factors that contribute to the increased rates of vertebral fractures include male sex, longer disease duration, and decreased mobility. At this point, it has not been established if these clinical consequences can be prevented with bisphosphonates.<sup>5</sup>

#### *Spinal Stenosis and CES*

Symptomatic spinal stenosis has been reported in a minority of patients with AS.<sup>63</sup> Neurogenic claudication symptoms from stenosis should be treated nonsurgically followed by surgical decompression if needed. Although rare, CES must be recognized in patients with AS. Symp-

toms include sensory deficits, loss of bowel and bladder control, lower-extremity pain, and weakness,<sup>22</sup> although it must be remembered that patients with tumors, spinal stenosis, and disc herniation can present with similar signs.<sup>14</sup> Cauda equina syndrome usually develops late in the course of AS when inflammatory activity has subsided. One theory of the pertinent mechanism includes spinal arachnoiditis-related compression, although structural compression must be recognized and treated promptly if present. Case reports of spinal block/CES from chronic inflammation and granulation have been published.<sup>24</sup> Spinal meningeal disease in AS can also progress to compression of the thecal sac or roots. Meningeal changes in AS include dilation and widening of the dural sacs with diverticula along the nerve root sheaths.<sup>25</sup> Histologically, these meningeal changes consist of intradural fibrous tissue and epidural "fatty tissue."<sup>25,36</sup> Surgical observations include thickened dura mater, arachnoiditis, adherent ligamentum flavum, and intradural adhesions.<sup>23,44</sup>

#### *Degenerative Axial Spine Pain and Radiculopathy*

The presence of axial spine pain or radiculopathy in the late stages of AS can be due to an active and/or compressive lesion. Causes of axial spine pain include trauma, pseudoarthrosis, kyphosis, and infection. Trauma can certainly be one cause of noninflammatory spine pain, but discovertebral destruction as a result of pseudoarthrosis should always be considered as well.<sup>33</sup> Potential mechanisms of single-level pseudoarthrosis include a stress fracture through a partially fused segment and a fracture through a completely fused segment. Both acute and stress fractures have a propensity for nonunion as a result of mechanical stress.<sup>18</sup> Axial pain in a stiff, kyphotic spine can be caused by pseudoarthrosis when stress is concentrated at the thoracolumbar junction. Infection is another, but less likely, cause of axial pain.

Even though lesions specific to AS can cause nerve root compression and degenerative disc disease, these disorders in this context are treated the same as when they appear in the general population. Most importantly, in the absence of a neurological deficit, treatment is initially nonsurgical; however, surgical intervention may be necessary if the issue remains unresolved.

#### *Differentiation Among Inflammatory Lesions, Fractures, and Infection*

The accurate assessment of lesions is important because treatment differs among inflammatory, noninflammatory, and infectious types. Magnetic resonance imaging is helpful in differentiating these processes. Table 1 summarizes the MR imaging signal characteristics of lesions commonly seen in AS. As shown in this table, active inflammatory lesions and acute microfractures have similar signal intensities on MR imaging. To differentiate these 2 lesions, note that a microfracture will more likely extend into the posterior elements than will an inflammatory lesion.<sup>51</sup> Microfractures also are more likely to occur later in the disease and in a more severely ankylosed spine. To differentiate the lesions described in the table from an infectious process, note that an infection will most likely be an enhancing lesion with irregular borders and soft tissue involvement.

If there are still doubts about a diagnosis following radi-

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TABLE 1  
Magnetic resonance imaging signal characteristics of common AS lesions\*

Type of Lesion	Signal on T1-Weighted MRI	Signal on T2-Weighted MRI
active inflammatory lesion	vertebra: low signal intensity; disc: low signal intensity	vertebra: high signal intensity; disc: high signal intensity
postinflammatory lesion	vertebra: high signal intensity; disc: low signal intensity	vertebra: low signal intensity; disc: high signal intensity
acute microfracture	low signal intensity	high signal intensity
chronic microfracture	low signal intensity	low signal intensity

\* Adapted from Hermann KA, Althoff CE, Schneider U, Zuhlsdorf S, Lembecke A, Hamm B, et al: Spinal changes in patients with spondyloarthritis: comparison of MR imaging and radiographic appearances. *Radiographics* 25:559-570, 2005.

ography and MR imaging, other modalities such as a bone scan can be helpful. Chronic inflammatory AS, unlike fracture, typically does not demonstrate an increased uptake.<sup>64</sup> Although increased radionuclide uptake in cases of acute AS is common, such a finding in chronic AS should alert the physician to possible fracture. With the combination of a positive bone scan and symptoms localized to one segment of the spine, the diagnosis of fracture is easier to make. Therefore, any history of trauma or the sudden onset of increased spinal pain may need to be investigated with a combination of the aforementioned imaging procedures.

## Medical Treatment

Obviously, treatment is tailored to the symptoms of each patient. Some have mild disease requiring intermittent NSAIDs, whereas others progress to surgical intervention. The European League Against Rheumatism recommendations for treating AS include the following: NSAIDs with an appropriate exercise program as a first-line therapy, the possibility of local corticosteroid injections in the sacroiliac joints, and opioids if other modalities fail.<sup>68</sup> In some studies, NSAIDs have slowed radiographically demonstrated progression of the disease.<sup>62</sup> Regular use of systemic steroids is discouraged particularly because of the risk of osteoporosis.

More recently, anti-TNF therapy has revolutionized AS treatment from many standpoints. In the past, patients with moderate to severe disease unresponsive to NSAIDs did not have many options. With the availability of anti-TNF agents, patients with a wide range of disease severity are now being successfully treated. Recently published results have shown that etanercept, infliximab, and adalimumab markedly improve symptoms.<sup>4,6,56</sup> Magnetic resonance imaging findings of inflammation are reduced or eliminated along with clinical symptoms,<sup>4,6</sup> but we do not yet have proof that this therapy leads to a reduction in bone fusion and ankylosis in patients with AS. All 3 agents have been approved by the Food and Drug Administration for use in AS. Although the numbers of patients have been too small for this type of analysis, thus far there have been no reports of an increased incidence of malignancies or lymphomas in those with AS who have undergone anti-TNF treatment.<sup>11</sup> Therefore, so far, and based on a risk/benefit analysis, these biological agents have clearly made a difference in the lives of our patients with AS.

## Surgical Treatment in AS

Although medical treatment is prevalent, surgical inter-

vention is necessary in cases of traumatic instability, significant deformity, and persistent degenerative radiculopathy with axial pain. The first 2 categories (trauma and deformity) have unique characteristics among patients with AS, but degenerative radiculopathy has similarities with its manifestation in the general population. Choosing which patients require surgical treatment is based on the degree of deformity, the level of pain and disability, and the medical condition of the patient.

### Treatment of Trauma

Three-column fractures are common in AS and typically are treated surgically. Hitchon and colleagues<sup>30</sup> reviewed 11 cases of thoracolumbar fractures from AS. Nine cases had 3-column fractures and required surgical fixation (on the presumption that these fractures are inherently unstable). More than half of the cases had a neurological deficit from the fractures, and one half improved following surgery. Eight fractures were a result of minor trauma such as ground-level falls.<sup>30</sup> Interestingly, patients in the minor trauma group were significantly older, implying that patients with advanced AS are more susceptible to fractures. Any neurological deficit caused by fracture was typically treated surgically. When surgery was required, all cases were treated with posterior segmental instrumentation (that is, pedicle screws and/or hooks). There was only one surgical complication, whereas 50% of patients improved neurologically after surgery.<sup>30</sup> Admittedly, separating the natural history of recovery from surgery-related recovery is difficult. At our institution, the preference is also a 3-column fixation using pedicle screws. Surgical complications, however, can be common. In 1 series, the condition of 40% of patients with a surgically treated fracture deteriorated, whereas that in patients treated with a halo vest did not.<sup>63</sup> Note, however, that no information on the type of injury and deficit was provided in that series. Therefore, it is impossible to compare outcomes between the surgical and conservative treatment groups.

### Deformity Surgery

As the kyphotic deformity progresses, alteration of the forward gaze (with resulting restriction in the field of vision), chin-on-chest deformity (with consequent swallowing difficulty), kyphosis-related muscular neck pain, and sagittal imbalance-induced thoracolumbar axial pain occur.<sup>38</sup> Identification of the site and cause of a deformity is the first critical step in its surgical management. If ankylosis causes the deformity, common sites include the cervi-

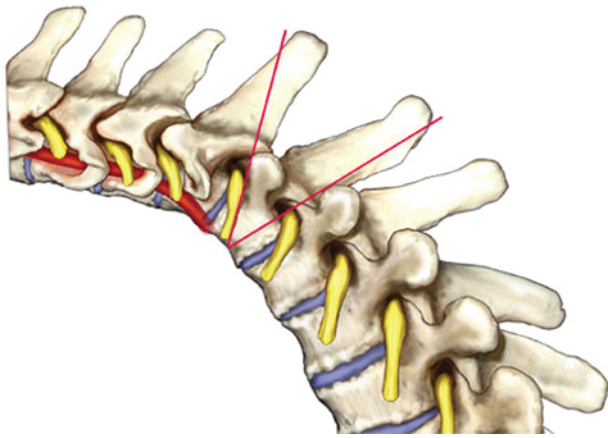


FIG. 3. Drawing illustrating areas subject to a PSO bone resection including bilateral lamina, facets, and pedicles with wedge resection of the anterior vertebral body.

cothoracic junction, midthoracic spine, thoracolumbar junction, and hip joint. Assuming equal degrees of deformity, lumbar correction surgery should probably be performed before correction of the cervical deformity because of a lower rate of complications.<sup>38</sup> Sagittal balance correction must take into account visual angle correction.<sup>35,59</sup> Alteration of lumbar kyphosis can cause overcorrection of the gaze angle. Because patients with AS typically have fixed cervical deformities, overcorrection of the gaze angle can cause significant gait difficulty.<sup>50</sup> Prior to spine surgery, hip flexion contractures must be identified and corrected.

The correction techniques include extension osteotomies such as the SPO,<sup>1</sup> PSO, or polysegmental lumbar posterior wedge osteotomy. The SPO was first described in 1945 for flexion deformity due to rheumatoid arthritis.<sup>53</sup> The procedure originally involved resection of the spinous processes and facets of the lumbar spine but has been modified by many subsequent authors. Resection includes the superior articular facet at the caudal level and the inferior articular facet of the cephalad vertebra. Because each level allows ~7° of correction, multiple levels are often resected to achieve the total correction needed. In 1973 McMaster and Coventry<sup>38</sup> reported on 17 patients treated with an SPO of the lumbar spine. No instrumentation was used; instead a plaster cast with a turnbuckle and hip spica immobilization was applied for gradual postoperative correction. Twelve patients had complications, including 2 deaths and 5 neurological deficits. These patients reported an impressive 39° correction average. Other authors have reported similar results (without instrumentation) following lumbar osteotomy.<sup>28,34</sup> In 1977 Simmons<sup>52</sup> reported on 19 patients who had been treated with an SPO of the lumbar spine while under local anesthesia. These historical articles paved the way for surgical treatment of AS-related deformity, but the high complication rate is prohibitive in the current era. Modern segmental instrumentation is now the cornerstone of treatment. Nonetheless, the SPO is still regarded as a good option in the correction of deformity, especially in the thoracolumbar spine. Unfortunately, these procedures produce an elongation of the anterior column and can theoretically cause stretch injury to the thecal sac and aorta. As a conse-

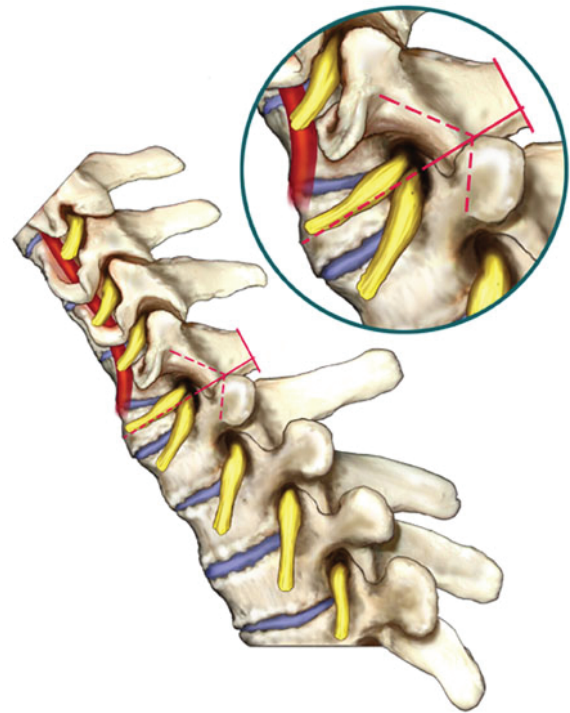


FIG. 4. Drawing showing the exit of 2 nerve roots through 1 foramina opening (C-7 and C-8 if the PSO is at C-7 and C-8 and T-1 if the PSO is at T-1).

quence, many surgeons, including us, prefer the PSO over the SPO.

Pedicle subtraction osteotomy is typically performed at the upper lumbar or cervicothoracic junction, but a midthoracic osteotomy can also be performed (Figs. 3 and 4). At C7–T1 the spinal canal is relatively wide, the C-8 roots are mobile, and the vertebral artery runs anterior to the C-7 transverse foramen. A PSO has been recommended for many disorders, including iatrogenic kyphosis, traumatic kyphosis, and rheumatoid arthritis. It has been used extensively for the correction of AS-related sagittal deformity. In 1953 Mason et al.<sup>37</sup> reported on an AS-related cervical flexion deformity treated with PSO. In 1958 Urist<sup>55</sup> described cervical spine osteotomy without instrumentation for AS (although others had reported cervical osteotomy for traumatic kyphotic deformity). He used an articulated plaster jacket to allow for gradual postoperative correction of the flexion deformity. In 1997 McMaster<sup>39</sup> reported on 15 patients treated with a PSO. A halo vest was applied preoperatively, and a laminectomy of C6–T1 was performed. Internal fixation was not used except in the 3 most recent cases in which he used Luque rods attached to the spinous process. The mean correction of deformity was 54°. Complications included neurological deficits (quadriplegia in 1 patient and transient C-8 palsy in 2 patients), pseudarthrosis requiring anterior fusion (2 patients), and subluxation at C7–T1 (4 patients); there was a mean loss of correction of 6° during the follow-up period in all patients. Simmons<sup>53</sup> reported on 42 patients who had undergone correction. After local anesthesia has been induced and with the patient in a sitting position, a C7–T1 posterior subtraction osteoto-

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my was performed without instrumentation. Patients were then placed in postoperative halo casting for ~ 4 months. The technique was similar to that of Urist, except that the correction was done intraoperatively rather than postoperatively. Similar to authors of many of the historical articles, Simmons had many complications in his study including 3 perioperative deaths and 3 repeated operations.

In addition to the SPO and PSO, a polysegmental posterior wedge osteotomy has been used in the correction of lumbar kyphosis.<sup>45</sup> Essentially, the technique is a variant of the SPO. In contrast to the process in the SPO, elongation of the anterior column in the polysegmental posterior wedge osteotomy is done over multiple segments and without fracturing the anterior column.<sup>58</sup> Van Royen and colleagues<sup>58</sup> reported a mean correction of 9.5° per level (36.3° overall) in 21 patients who had been treated using this osteotomy combined with instrumentation in the thoracolumbar or lumbar spine. At the last follow-up, however, there was a mean loss of 10.7°. In addition, 10% of the cases required an additional anterior release, 33% had a pedicle fracture, 33% had a deep wound infection, and 33% had pseudoarthrosis. Other authors have reported results of polysegmental wedge osteotomies, including Hehne and colleagues<sup>27</sup> who described 177 patients with AS. The average correction was 44° (9.5° per segment), and there was no pseudoarthrosis, with minimal loss of correction in the long term. In 16 patients Chen<sup>8</sup> reported an average correction of 25.8° (5° per level) together with a 25% pseudoarthrosis rate. Overall, this technique has reasonable results and is best indicated for thoracic spine abnormalities and when gradual correction is needed over multiple levels.

Fortunately, authors of more contemporary surgical studies have improved outcomes and lowered complications. For instance, Kim et al.<sup>32</sup> described lumbar PSO in 45 patients with AS. Sagittal imbalance was corrected by 31° with chin-brow vertical angle correction of the same amount. Clinical satisfaction was high, and the complication rates were acceptable. Other PSO studies have shown fusion rates as high as 100% with no neurological complications.<sup>42</sup> The PSO is also our preferred surgical technique in both the thoracolumbar and cervicothoracic spine to correct deformity. Similar to most contemporary authors, we rely heavily on instrumentation (lateral mass and pedicle screws) for immediate rigidity and stability. By using modern and improved surgical equipment, imaging modalities, and anesthesia, the current complication profile has been significantly lowered compared with the historical data.

### *Surgical Treatment of Pseudarthrosis and Radiculopathy*

In instances of pseudarthrosis causing axial pain, most cases occur at the thoracolumbar junction and can be treated surgically. Kuo and associates<sup>33</sup> reported on 2 cases that were treated using anterior spinal fusion with good results. Fang and colleagues<sup>17</sup> reported on 40 patients with pseudoarthrosis. Treatment with antiinflammatory medications combined with bracing was attempted, but many patients required surgery for persistent, localized back pain and/or neurological deficit. Surgical treatment consisted of anterior fusion with an iliac crest autograft. Pathological findings confirmed pseudoarthrosis without inflammation. Fifteen of 16 surgically treated patients had “good to complete” relief of back pain, and 14 of 16 patients had eventual radi-

ographically demonstrated solid fusion. Overall, pseudoarthrosis in patients with AS initially should be treated medically. In the event of persistent pain refractory to medical therapy or accompanied by neurological deficit, fusion surgery is indicated. The preferred technique consists of interbody fusion with segmental instrumentation.

### *Surgical Treatment of CES*

In the absence of a significant compressive lesion, the preferred treatment for CES is nonsurgical. Note, however, that some studies have shown an improved outcome with surgical intervention. Surgical options include resection of diverticula, laminectomy, and shunting of the intrathecal space. Lumboperitoneal shunting improves cerebrospinal fluid resorption in the lumbar thecal sac and is postulated to improve cerebrospinal fluid dynamics.<sup>9</sup> In a metaanalysis of 86 patients with CES, authors divided the patients into no (conservative) treatment, medication, and surgery groups.<sup>1</sup> In the surgical group consisting of 15 patients, 87% had improvement in sensory or motor signs or bladder or bowel symptoms. Comparatively, only 12.5% of patients in the medication group and none of those in the conservative group experienced improvement. We prefer the shunting procedure because of the higher complication rates with laminectomy and dural repair procedures. Although the data are retrospective and uncontrolled, no more complete information exists on the surgical and medical treatment of progressive CES in the AS population.

## Conclusions

The spinal pathophysiology in AS has many dimensions. Early in the disease, inflammatory lesions cause pain. As the disease progresses, the structural integrity of the spine is affected. At these stages, medical treatment and conservative therapies are the first-line treatments. Later in the disease, pseudoarthrosis, traumatic fractures, and other complications can cause pain, deformity, and neurological harm. In these cases, surgery may be necessary for effective treatment and relief. Extensive knowledge of the differential diagnoses and available treatment modalities is necessary for optimal outcomes when treating spinal disease in patients with AS.

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Address correspondence to: Michael H. Weisman, M.D., 8700 Beverly Boulevard, Becker Building #131, Los Angeles, California 90048. email: weisman@cshs.org.