

# **ANKYLOSING SPONDYLITIS**

Darren Bergey M.D., Robert S. Pashman. M.D.  
J. Patrick Johnson M.D.

## **INTRODUCTION**

Ankylosing spondylitis is a seronegative spondyloarthropathy which primarily affects the axial skeleton, including ligaments and articulations of the pelvis and spinal column. It is estimated to occur in 0.02 % of the population. Although once believed to affect men predominantly, recent evidence suggests women are affected equally but experience milder symptoms. The HLA-B27 antigen is positive in 80-90 percent of patients with ankylosing spondylitis compared with 8 percent of the general population of American Caucasians (28). This strongly suggests that HLA-B27 antigen is important in the pathogenesis of ankylosing spondylitis. Its precise role remains unclear, however, it is generally understood that an inflammatory response is incited by environmental or infectious agents and hosts are rendered susceptible by HLA-B27 or related antigens (37).

Similar to rheumatoid arthritis, the pathophysiology of ankylosing spondylitis remains unclear. The basic pathologic process is an inflammatory focus, predominantly lymphocytic, that targets both articular joints as well as the insertion of ligaments, tendons and capsules to bone (entheses). Reactive bone formation at these entheses, termed enthesopathy, ultimately results in progressive ankylosis of the axial skeleton, typically involving the sacroiliac, apophyseal, and costovertebral joints. The course of the disease includes progressive enchondral ossification of cartilage, resulting in the characteristic joint stiffness and ankylosis. Symptoms usually begin in at the sacroiliac joints and progress proximally in the spine.

The “Romanus lesion” is an erosion of the anterior and lateral border of the vertebral endplate at the site of vascular attachment of the annulus fibrosus. This lesion represents a focal area of spondylitis, ultimately resulting in syndesmophyte formation and ossification of the annulus fibrosus. These osseous changes result in the classic “bamboo spine” appearance radiographically, which is the hallmark of ankylosing spondylitis.

## **CLINICAL PRESENTATION**

Ankylosing spondylitis typically presents in healthy adults during the second or third decades. Sacroiliitis or low back pain is typically the initial manifestation of the disease. Pain may be unilateral or bilateral and may include radicular symptoms extending into the buttocks or thigh. This radicular pain seldom extends below the knee. Symptoms are usually worse in the morning and improve with activity. This clinical feature distinguishes ankylosing spondylitis from mechanical low back pain, which generally worsens with activity and improves with rest. Night pain relieved by activity is not an uncommon feature of ankylosing spondylitis (28).

In patients with an uncontrolled inflammatory phase of the disease, the lumbar, thoracic, and cervical spine become progressively ankylosed and kyphotic. This usually progresses in a caudal to cranial direction. According to Simkin and colleagues, kyphosis is produced when the patient assumes a "flexed posture in an attempt to unload the facets, thereby reducing joint pressure and alleviating pain (51)." Compensatory flexion contractures of the hips and knees may develop as the patient attempts to maintain an erect posture. Following the inflammatory phase, the patient is typically stiff and kyphotic but relatively pain free. Significant spinal deformity and functional disability may be the end result. Carette and associates studied 150 war veterans for a mean of 38 years and found that, despite severe limitations in spinal motion, 50 per cent of the patients functioned well (13). Those with more severe deformities, however, may be unable to stand upright, lose horizontal gaze, and develop the so-called chin-on-chest deformity.

Spinal fracture and spondylodiscitis are clinical manifestations of ankylosing spondylitis which are of specific interest to spine surgeons. Ossification of the disc space occurs centripetally through the annulus fibrosus, and only rarely is the center of the disc involved. This incomplete ossification combined with stress concentration from loss of polysegmental spinal motion and secondary osteopenia predispose patients to spinal fracture and nonunion (spondylodiscitis) (26). Spondylodiscitis presents as focal pain with coexisting erosive sclerotic changes in adjacent vertebral bodies. It is uncertain whether this is a primary inflammatory process or the result of trauma. Radiographically, the appearance of spondylodiscitis, pseudarthrosis and discitis are very similar.

Neurologic decline in the patient with ankylosing spondylitis is uncommon, exclusive of fractures. However, neurologic injury can be a significant complication of spinal fracture and the diagnosis should not be missed. Severe spinal deformities, together with spondylodiscitis and acute fracture provide the most common indication for spine surgery in the patient with ankylosing spondylitis.

## **SURGICAL MANAGEMENT**

### **General Principles**

As ankylosing spondylitis may lead to severe flexion deformities of the spine the goal in treatment of these patients is early recognition and adequate medical therapy in an attempt to control the disease progress and prevent associated deformities. However, patients may still become grossly deformed and functionally disabled. Spinal osteotomy may be indicated to correct the deformity and achieve upright posture.

Two considerations determine the technique and location of the osteotomy: the region of the spinal deformity that maximally influences sagittal alignment and a surgical procedure that minimizes the surgical risk. It is important to reemphasize that the overall spinal balance as well as the hips must be evaluated to delineate the primary site of deformity. In some patients more than one of these sites may contribute to the deformity. The lumbar spine is the most common site of deformity followed by the thoracic and cervical regions. Accurate measurement of the deformity is required for surgical planning and Simmons (52) advocates the chin-brow to vertical angle as the most effective and reproducible

measurement of deformity (Fig. 1a,b).

Deformities isolated to the lumbar spine are corrected by a lumbar osteotomy procedure. The osteotomy is preferred below the level of the conus medullaris and is usually performed at L3 in order to avoid acute angular correction at the cord level (52). Most lumbar-thoracic kyphotic deformities can also be addressed through a single lumbar osteotomy. The correction should be planned so that the plumb line from C7 falls within the body of S1. Even in cases in which the thoracic kyphosis is greater than normal, a compensatory lumbar osteotomy may correct sagittal plane malalignment and allow the patient to have forward gaze with the hips and knees fully extended. In cases of severe thoracic kyphosis, where the lumbar and cervical lordosis have been at least partially maintained, thoracic osteotomy by a combined anterior and posterior approach may be indicated. When the primary deformity is at the cervical-thoracic junction, resulting in a 'chin-on-chest' deformity, an extension osteotomy of the cervical spine is indicated. The C7-T1 junction is the preferred location as it places the osteotomy below the entrance of the vertebral arteries into the transverse processes at C6 and uses the relatively large spinal canal-to-cord area ratio to safely obtain correction.

The influence of severe hip flexion contractures with or without associated hip joint disease is critical in the preoperative assessment. Soft tissue releases about the hips or, more commonly, total hip joint arthroplasty may be sufficient in itself to allow the patient to stand reasonably upright and see straight ahead, irrespective of the spinal deformity (5). These should be performed prior to any surgical correction of spinal deformity.

Preoperatively, patients should be screened for cardiac and pulmonary abnormalities that can be associated extra-articular manifestations of ankylosing spondylitis. Although pulmonary function abnormalities secondary to decreased thoracic expansion have not carried anesthetic risk for most patients (20), 10 per cent will have cardiac pathology, generally either aortic stenosis or conduction abnormalities.

Although local anesthesia has been reported in the treatment of these spinal deformities (32, 57), general anesthesia is preferred. Intubation is facilitated by the use of fiber optic guidance in cases where cervicothoracic kyphosis complicates easy passage of the endotracheal tube. After the patient has been anesthetized and intubated, the operating table must be modified according to the patient's spinal deformity. The table is flexed into a position where the apex of the table is under the primary spinal deformity. Bolsters are used to free the abdomen and protect bony prominences and peripheral nerves in the extremities.

## **Lumbar Osteotomy**

Smith-Petersen and colleagues' first proposed lumbar osteotomy for the correction of spinal deformity caused by ankylosing spondylitis in 1945 (55). They performed a V-shaped wedge resection osteotomy at the L3-L4 level. The spinous processes were removed at the appropriate angle as were the L3-L4 lamina. The osteotomy was extended laterally to include the bilateral facet joints. The lamina and pedicles were undercut to prevent impingement of the dura or nerve roots upon closure of the osteotomy site. This osteotomy wedge was then closed and the deformity corrected via forceful manipulation through hyperextension. This maneuver caused fracture of the anterior and middle

columns allowing the osteotomy to close. Osteotomes may be used to complete the fracture if the manual maneuver is unsuccessful. Local bone grafts were placed across the osteotomy sites, and the patient was immobilized in a postoperative cast for 2 months followed by a back brace for one year. Six patients were reported and detailed results are not described.

Simmons used the Smith-Petersen osteotomy and popularized the use of local anesthetic for both lumbar and cervical osteotomies, arguing that two thirds of the 8 to 10 percent mortality and 30 percent neurologic complications documented in previous studies were related to the use of general anesthesia (52). In his series of 90 patients he was able to show that correction can be reliably achieved through this posterior osteotomy without a secondary anterior approach, paralleling the experience of other authors (25,26,37,54,63). His series reported a 40 to 104 degree correction with an average of 56 degrees (Fig. 2). The chin-brow to vertical angle improved from an average of 60 degrees preoperatively to 5 degrees postoperatively.

The most common complication associated with this procedure is neurologic compression. In Simmons series seven (8%) patients developed nerve root or cauda equine symptoms postoperatively (52). Such complications can be minimized by adequate decompression and undercutting of the lamina prior to closure of the osteotomy site and rigid stabilization. If these complications occur, prompt reexploration and decompression should be performed.

This osteotomy advocated by Smith-Peterson and Simmons uses the middle column as the fulcrum for closure of the posterior osteotomy and has the inherent risk of placing the spinal cord on stretch. Thomasen reported a spinal column shortening osteotomy via a posterior approach utilizing a decancellation procedure (56) (FIG. 3). The decancellation procedure, also known as an 'eggshell osteotomy' or 'pedicle subtraction osteotomy', is performed by removing a wedge of the posterior elements of L3 as well as bilateral pedicles. This is followed by resection of the posterior vertebral cortex as well as the cancellous bone of the vertebral body. The anterior cortex of the vertebral body is left intact and is the fulcrum for closure, effectively shortening the spinal canal and achieving angular correction. Moreover, removal of the pedicle creates a "super-foramen," which transmits the nerve roots from the adjacent segments and decreases the chance for root compression. Generous undercutting/decompression of the supra and sub adjacent laminar edges are performed to ensure adequate space for the redundant dura that may be produced during closure of the osteotomy. Segmental spinal fixation utilizing multiple pedicle screws and/or hook constructs are used to allow for immediate patient mobilization. Carefully, the table is extended, closing the osteotomy. If necessary, closure can be augmented by pressure on the patient's shoulders or legs and by compression between the pedicle screws once the rods are placed. A wake-up test is routinely performed to assess neurologic function. Finally, a local bone graft is applied and augmented with iliac crest autograft or banked bone, as needed. Thomasen reported 12 to 50 degrees of correction in 11 patients, with 5 of the 11 having a correction of less than 35 degrees (56). He concluded that this small degree of correction was all that was required to obtain an erect posture. This technique is procedure of choice at this time.

## **Thoracic Osteotomy**

Thoracic osteotomies are rarely required in patients with ankylosing spondylitis. As stated previously, if the thoracic kyphosis is mild or moderate and associated with a flat or kyphotic lumbar spine the

deformity can be addressed with a lumbar spine osteotomy. The rare patient will have severe thoracic kyphosis with minimal loss of lumbar or cervical lordosis. This is the patient in whom a thoracic osteotomy may be indicated.

Smith-Petersen pointed out in 1945 that single-stage posterior thoracic osteotomy correction is compromised by stiffness of the costovertebral joints. We favor a two-stage procedure that consists of a first-stage transthoracic approach creating osteotomies through the ossified thoracic disc spaces. Anterior interbody fusion is performed with autogenous cancellous bone graft. This is followed at the same sitting or one week later by posterior, multiple level Smith-Peterson osteotomies with segmental instrumentation. Dural adhesions to the lamina that formed during the inflammatory phase of the disease can be encountered during posterior osteotomy and likewise may make passage of sublaminar wires used in the Luque technique more difficult. We therefore prefer to stabilize the osteotomy with hook-rod compression instrumentation or thoracic pedicle screws. The approach is similar to that used for severe juvenile kyphosis (7,9).

## **Cervical Osteotomy**

Cervical osteotomy may occasionally be indicated when the primary deformity is isolated at the cervical-thoracic junction. In 1953, Mason and associates reported successful correction of flexion deformity of the cervical-thoracic spine in a patient with ankylosing spondylitis (40). They performed the osteotomy distal to C7 in order to avoid damage to the vertebral arteries. In 1958, Urist reported a successful osteotomy at the cervical-thoracic junction in a patient awake under local anesthesia (59).

Simmons has reported experience in 95 patients, consisting of a wide laminectomy from C6 to T1 with osteotomy at the C7-T1 space (53). The entire posterior arch of C7 is resected, as is the inferior half of C6 and the upper half of T1. The lamina are undercut and complete foraminotomies are performed to prevent impingement of the C8 nerve root. The amount of bone to be resected is based on the preoperative chin-brow to vertical angle. This angle is transferred to the lateral radiograph, with the apex of the angle at the posterior edge of the C7-T1 disc space. Following bony decompression Simmons extended the neck and "cracked" the anterior column (FIG 4). An osteotome may be used to perform an anterior osteotomy in a more controlled fashion. Simmons performed the procedure under local anesthesia with halo control and then fixed the halo to a body cast which was worn for 4 months. There were no mortalities and C8 weakness was the primary morbidity occurring in 18 patients, 5 being permanent deficits.

We believe the preferred technique for cervical osteotomy to be that described by Urist and Simmons (59,53). We prefer general anesthetic with controlled halo correction, followed by either an intraoperative wake-up test or spinal cord monitoring. Lateral mass screws are used in the cervical spine with thoracic pedicle screws placed in the upper thoracic spine for interal fixation. Halo and vest supplementation may or may not be utilized.

## **Complications**

Postoperative ileus is common in these patients. Nasogastric drainage is essential. Although aortic rupture has been reported (39, 50, 60) the case in question occurred after closed forceful osteoclasis of severe kyphosis in a patient who had previously been treated with radiation therapy for ankylosing spondylitis. We believe that the fear and likelihood of this complication has been greatly overstated.

It has been stated in review of several series (43-57) that mortality has varied from 8 to 10 percent, and neurologic complications have occurred in up to 30 per cent of patients. However, these quotes may be misleading. In our analysis of the 14 largest series consisting of five or more cases reported, (17-21, 36-39, 43, 53, 54, 56, 57) a total of 427 cases, we find a 4 per cent incidence of neurologic complications and a 5 percent mortality rate. However, and perhaps even more importantly, it appears that in eight of these reports, consisting of 74 patients, there were no neurologic deficits. In nine of these series with a total of 85 patients, (17-21, 39, 54, 56) no deaths were reported. In the single largest study, 177 patients reported by Hehne and associates, there was 2.3 per cent mortality and 2.3 per cent irreversible root lesions (24). Based on our review of the published data and our own experience, we believe that neurologic complications and mortality can be greatly lessened if not prevented altogether by careful attention to four critical factors: (1) avoiding compression of neurologic tissue, (2) monitoring neurologic function during the osteotomy (by wake-up test), (3) using internal fixation, and (4) avoiding translational displacement at the osteotomy site.

## **FRACTURES**

Osteoporosis and stress concentration secondary to long, stiff lever arms enhance the susceptibility of the ankylosing spondylitis patients to acute spinal fracture. Hunter and Dubo (30) and Hyman et al. (31) noted that 75 per cent of fractures occur in the cervical or cervicothoracic junction, 14 per cent in the thoracic spine, and 5 per cent in the lumbar spine.

Cervical fractures commonly involve both anterior and posterior columns, and this fact probably explains the increased rates of mortality and neurologic complication seen in patients with ankylosing spondylitis as compared to fractures in normal spines. (6-9, 44).

A review by Trent and colleagues summarizing the world literature points out that thoracolumbar fractures in patients with ankylosing spondylitis commonly occur between T9 and L2 and are associated with a 25 per cent incidence of neurologic deficit at initial presentation, with subsequent poor prognosis for recovery(58). All authors stress the importance of a high index of suspicion in any ankylosing spondylitis patient with acute onset of new focal pain or deformity. Occult fractures must be suspected and tomography is often required to fully evaluate the symptomatic areas.

Treatment of the ankylosing spondylitis patient with an acute fracture begins with positioning and transport in the prefracture alignment. Extending the neck in the case of cervical fracture or positioning the patient supine in the case of thoracic or lumbar fractures can have serious neurologic consequences(58). Both operative (6, 8, 34, 57) and nonoperative management of these fractures have been described in the literature with similar good outcomes for solid union. Clearly surgery is indicated in cases of progressive neurologic deficit. With current advancements in spinal fixation techniques we believe that aggressive surgical management leads to earlier mobilization and may

avoid the secondary complications of prolonged bed rest.

## **SPONDYLODISCITIS**

Histopathologic features of both inflammatory enthesitis and post-traumatic nonunion (2-5) are noted in the entity termed spondylodiscitis. The true etiology remains controversial. Unlike acute fractures, spondylodiscitis is viewed as a stable lesion because of its lack of involvement of both anterior and posterior columns. The stability of this type of lesion accounts for its low incidence of associated neurologic deficit. In contrast to acute fractures, spondylodiscitis more commonly occurs in the thoracic and lumbar spine. Nonoperative treatment has been associated with spontaneous healing of these defects (3-23, 29, 33, 47, 49, 61, 64). Hehne and associates have reported a 97 per cent fusion rate at 2 years in the operative treatment of spondylodiscitis by pedicle screw fixation in 28 patients(24). Ho and colleagues, reporting the experience at the University of Hong Kong, observed excellent results with anterior spinal fusion in 16 patients (27).

Our current practice for the treatment of both acute fractures and spondylodiscitis in patients with ankylosing spondylitis and no neurologic deficit is early operative treatment with posterior segmental fixation. Patients with neurologic deficit in whom a compressive lesion can be identified may also benefit from anterior decompression. These recommendations are based on an approach that parallels the treatment of fractures in normal spines. Early fixation decreases the chance of progressive deformity as well as the untoward effects of prolonged recumbence and secondary pulmonary and vascular complications in the non-operated patient(22, 45).

## **ACKNOWLEDGEMENT**

The authors would like to acknowledge Helen Cambron, RN, FNP-C for her illustrative contribution to this chapter.

## REFERENCES

1. Adams JC: Technique, dangers and safeguards in osteotomy of the spine. *J Bone Joint Surg* 34B:226-232, 1952.
2. Agarwal AK, Reidbord HE, Kraus DR, Eisenbeis CH Jr: Variable histopathology of discovertebral lesion (spondylodiscitis) of ankylosing spondylitis. *Clin Exp Rheumatol* 8:67-69,1990.
3. Baggenstoss AH, Bickel WH, Ward LE: Rheumatoid granulomatous nodules as destructive lesions of vertebrae. *J Bone Joint Surg* 34A:601 -609, 1952.
4. Ball J: The heberden oration, 1970. Enthesopathy of rheumatoid and ankylosing spondylitis. *Ann Rheum Dis* 30:213-223,1971.
5. Bisia RS, Ranawat CS, Inglis AE: Total hip replacement in patients with ankylosing spondylitis with involvement of the hip. *J Bone Joint Surg* 58A:233-238, 1976.
6. Bohlman HH: Acute fractures and dislocations of the cervical spine: an analysis of three hundred hospitalized patients and review of the literature. *J Bone Joint Surg* 61A:1119-1142,1979.
7. Bohm H, Harms J, Donk R, Zieike K: Correction and stabilization of angular kyphosis. *Clin Orthop* 258:56-61,1990.
8. Bradford DS, Schumacher WL, Lonstein JE, Winter RB: Ankylosing spondylitis: Experience in surgical management of 21 patients. *Spine* 12:238-243, 1987.
9. Bradford DS: Kyphosis: Current Orthopaedic Management. In Kane WJ (ed). New York, Churchill-Uving-stone, 1981.
10. Briggs H, Keats S, Schlesinger PT: Wedge osteotomy of the spine with bilateral intervertebral foraminotomy. *J Bone Joint Surg* 29:1075-1082, 1947.
11. Broom MJ, Raycroft JF: Complications of fractures of the cervical spine in ankylosing spondylitis. *Spine* 13:763-766,1988.
12. Calin A, Fries JF: Striking prevalence of ankylosing spondylitis in "healthy" W27 positive males and females: A controlled study. *N Engl J Med* 293:835-839, 1975.
13. Currence S, Graham D, Little H, et al: The natural disease course of ankylosing spondylitis. *Arthritis Rheum* 26:186-190,1983.
14. Carter ET, McKenna CH, Brian DD, Kurland LT: Epidemiology of ankylosing spondylitis in Rochester, Minnesota, 1935-1973. *Arthritis Rheum* 22:365-370,1979.

15. Cawley MID, Chalmers TM, Kellgren JH, Ball J: Destructive lesions of vertebral bodies in ankylosing spondylitis. *Ann Rheum Dis* 31:345-358, 1972.
16. Detwiler KM, Loftus CM, Godersky JC, Menezes AH: Management of cervical spine injuries in patients with ankylosing spondylitis. *J Neurosurg* 72:210-215,1990.
17. Ernneus H: Wedge osteotomy of spine in ankylosing spondylitis. *Acta Orthop Scand* 39:321-326, 1968.
18. Fang D, Leong JCY, Ho EKW, et al: Spinal pseudoarthrosis in ankylosing spondylitis. Clinicopathological correlation and the results of anterior spinal fusion. *J Bone Joint Surg* 70B:443-447, 1988.
19. Fast A, Parikh S, Marin EL: Spine fractures in ankylosing spondylitis. *Arch Phys Med Rehabil* 67:595-597, 1986.
20. Feltelius N, Hedenstrom H, Hillerdal G, Hallgren R: Pulmonary involvement in ankylosing spondylitis, *Ann Rheum Dis* 45:736-740, 1986
21. Goel MK: Vertebral osteotomy for correction of fixed flexion deformity of the spine, *J Bone Joint Surg* 50A:287-294,1968.
22. Graham B, Van Peteghem PK: Fractures of the spine in ankylosing spondylitis: Diagnosis, treatment, and complications. *Spine* 14:803-807,1989.
23. Hansen ST, Taylor TKF, Honet JC, Lewis FR: Fracture dislocations of the ankylosed thoracic spine in rheumatoid spondylitis: Ankylosing spondylitis, Marie-Striimpell disease. *J Trauma* 7:827-837, 1967.
24. Hehne HJ, Zieike K, Bohm H: Polysegmental lumbar osteotomies and transpedicled fixation for correction of long-curved kyphotic deformities in ankylosing spondylitis: Report on 177 cases. *Clin Orthop* 258:49-55,1990.
25. Herbert JJ: Vertebral osteotomy: technique, indications, and results. *J Bone Joint Surg* 30A:680-689, 1948.
26. Herbert JJ: Vertebral osteotomy for kyphosis, especially in Marie-Strumpell arthritis. *J Bone Joint Surg* 41A:291-320,1959.
27. Ho EKW, Chan FL, Leong JCY: Postsurgical recurrent stress fracture in the spine affected by ankylosing spondylitis. *Clin Orthop* 247:87-89, 1989.
28. Hochberg MC: Ankylosing spondylitis. *Sem Spine Surg* 2:86-94,1990.

29. Hunter T, Dubo HIC: Spinal fractures complicating ankylosing spondylitis. *Arthritis Rheum* 26:751-759, 1983.
30. Hunter T, Dubo HIC: Spinal fractures complicating ankylosing spondylitis. *Ann Intern Med* 88:546-549, 1978.
31. Hyman SA, Rogers WD, Bullington JC III: Cervical osteotomy and manipulation in ankylosing spondylitis: successful general anesthesia after failed local anesthesia with sedation. *J Spinal Disord* 3:423-426,1990.
32. Jackson RP, Simmons EH: Dural compression as a cause of paraplegia during operative correction of cervical kyphosis in ankylosing spondylitis. *Spine*16:846-848,1991.
33. Kanefield DG, Mullins BP, Freehafer AA, et al: Destructive lesions of the spine in rheumatoid ankylosing spondylitis. *J Bone Joint Surg* 51A:1369-1375, 1969.
34. Krodel A, Stiirz A, Siebert CH: Indications for and results of operative treatment of spondylitis and spondylodiscitis. *Arch Orthop Trauma Surg* 110:78-82,1991.
35. LaChapelle EH: Osteotomy of the lumbar spine for correction of kyphosis in a case of ankylosing spondyloarthritis. *J Bone Joint Surg* 28:851-858, 1946.
36. Law WA: Osteotomy of the spine. *Clin Orthop* 66:70-76,1969.
37. Lawrence JS: The prevalence of arthritis. *Br J Clin Pract* 17:699-705,1963.
38. Uchtblau PO, Wilson PD: Possible mechanism of aorta rupture in orthopaedic correction for rheumatoid spondylitis. *J Bone Joint Surg* 38A:123-127, 1956.
39. Mason C, Cozen L, Adelstein L: Surgical correction of flexion deformity of the cervical spine. *Calif Med* 79:244-246,1953.
40. McMaster MJ: A technique for lumbar spinal osteotomy in ankylosing spondylitis. *J Bone Joint Surg* 67B:204-210,1985.
41. McMaster MJ, Coventry MB: Spinal osteotomy of ankylosing spondylitis: Technique, complications, and long-term results. *Mayo Clin Proc* 48:476-486, 1973.
42. McMaster PE: Osteotomy of the spine for fixed flexion deformity. *J Bone Joint Surg* 44A:1207-1216, 1962.
43. Murray GC, Persellin RH: Cervical fracture complicating ankylosing spondylitis: A report of eight cases and review of the literature. *Am J Med* 70:1033-1041, 1981.
44. Osgood C, Martin LG, Ackerman E: Fracture-dislocation of the cervical spine with ankylosing

- spondylitis:Report of two cases. *J Neurosurg* 39:764-769, 1973.
45. Puschel J, Zieike K: Transpedicular vertebral instrumentation using VDS-instruments in ankylosing spondylitis. *Orthop Trans* 9:130, 1985.
  46. Rapp GF, Kernek CB: Spontaneous fracture of the lumbar spine with correction of deformity in ankylosing spondylitis. *J Bone Joint Surg* 56A:1277-1278,1974.
  47. Rogers WA: Fractures and dislocation of cervical spine:An end-result study. *J Bone Joint Surg* 39A-.341-376,1957.
  48. Romanus R, Yden S: Destructive and ossifying spondylitic changes in rheumatoid ankylosing spondylitis (pelvo-spondylitis ossificans). *Acta Orthop Scand* 22:88-99,1952.
  49. Schaberg FJ Jr: Aortic injury occurring after minor trauma in ankylosing spondylitis, *J Vase Surg* 4:410-411,1968.
  50. Seaman WB, Wells J: Destructive lesions of the vertebral bodies in rheumatoid disease. *AJR* 86:241-249,1961.
  51. Simkin PA, Downey DJ, Kilcoyne RF: Apophyseal arthritis limits lumbar motion in patients with ankylosing spondylitis. *Arthritis Rheum* 31:798-802, 1988.
  52. Simmons EH: Kyphotic deformity of the spine in ankylosing spondylitis. *Clin Orthop* 128:65-77, 1977.
  53. Simmons EH: The surgical correction of flexion deformity of the cervical spine in ankylosing spondylitis. *Clin Orthop* 86:132-143,1972.
  54. Smith-Petersen MN, Larson CB, Aufranc OE: Osteotomy of the spine for correction of flexion deformity in rheumatoid arthritis. *J Bone Joint Surg* 27:1-11, 1945.
  55. Sutherland RIL, Matheson D: Inflammatory involvement of vertebrae in ankylosing spondylitis. *J Rheumatol* 2:296-302,1975.
  56. Thomasen E: Vertebral osteotomy for correction of kyphosis in ankylosing spondylitis. *Clin Orthop* 194:142-152,1985.
  57. Thompson WAL, Ingersoll RE: Osteotomy for correction of deformity in Marie-Striimpell arthritis. *Surg Gynecol Obstet* 90:551-556,1950.
  58. Trent G, Armstrong GWD, O'Neil ): Thoracolumbar fractures in ankylosing spondylitis: High risk injuries. *Clin Orthop* 227:61-66,1988.
  59. Urist MR: Osteotomy of the cervical spine. *J Bone Joint Surg* 40A:833-843,1958

60. Weatherley C, Jaffray K, Terry A: Vascular complications associated with osteotomy in ankylosing spondylitis: A report of two cases. *Spine* 13:43-46, 1988.
61. Wholey MA, Pugh DG, Bickel WH: Localized destructive lesions in rheumatoid spondylitis. *Radiology*.74:54-56,1960.
62. Will R, Edmunds L, Elswood J, Calin A: Is there sexual inequality in ankylosing spondylitis? A study of 498 women and 1202 men. *J Rheumatol* 17:1649-1652, 1990.
63. Wilson MJ, Turkell JH: Multiple spinal wedge osteotomy: Its use in a case of Marie-Strumpell spondylitis. *Am J Surg* 77:777-782, 1949.
64. Yau A, Chan RN: Stress fracture of the fused lumbodorsal spine in ankylosing spondylitis. A report of three cases. *J Bone Joint Surg* 56B:681-687, 1974.

FIGURES

Figure 1a (left) and 1b (right). Measurement of sagittal plane deformity with chin-brow to vertical angle.

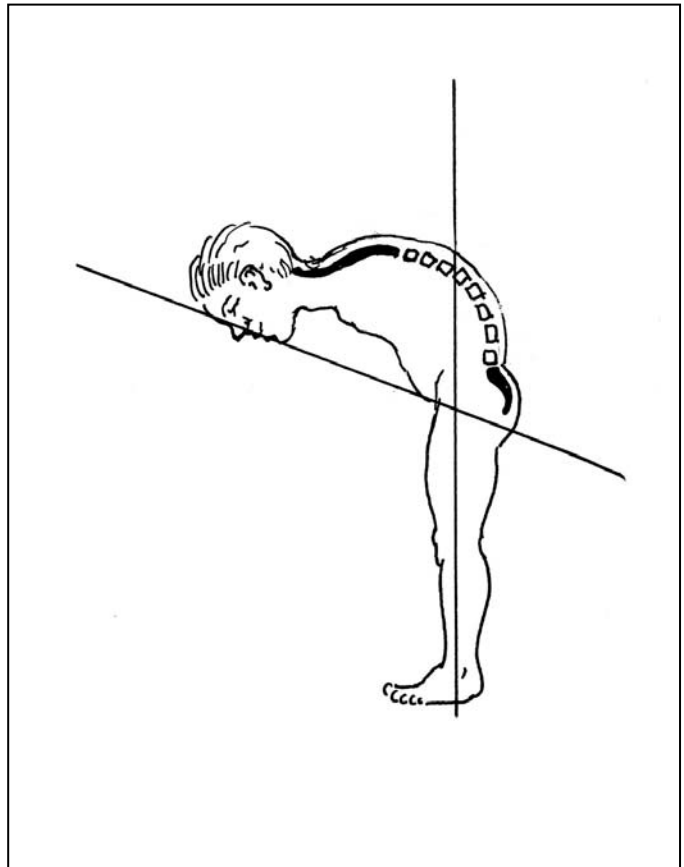
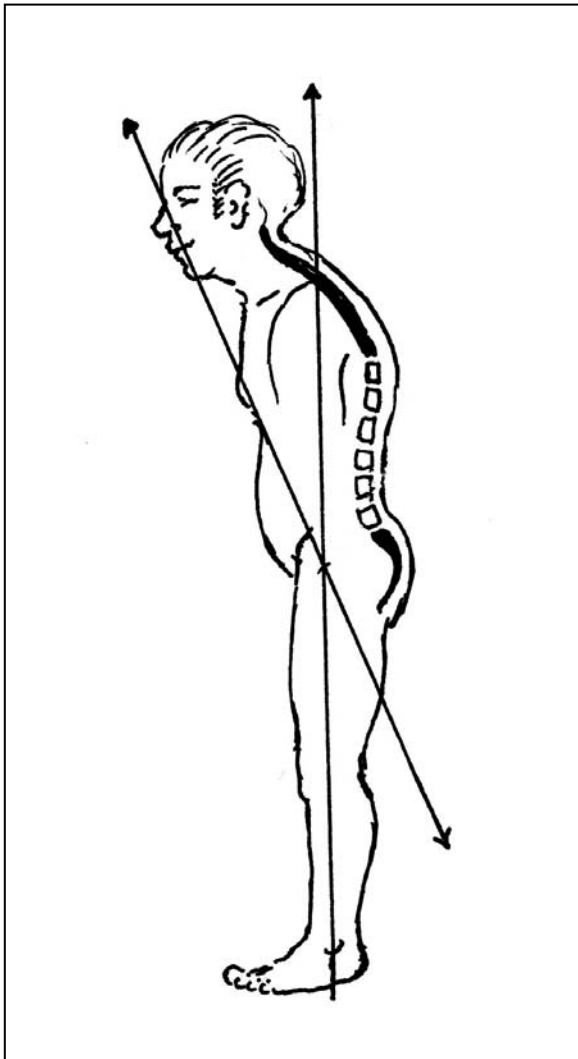


Figure 2. Illustration of Smith-Petersen osteotomy technique.

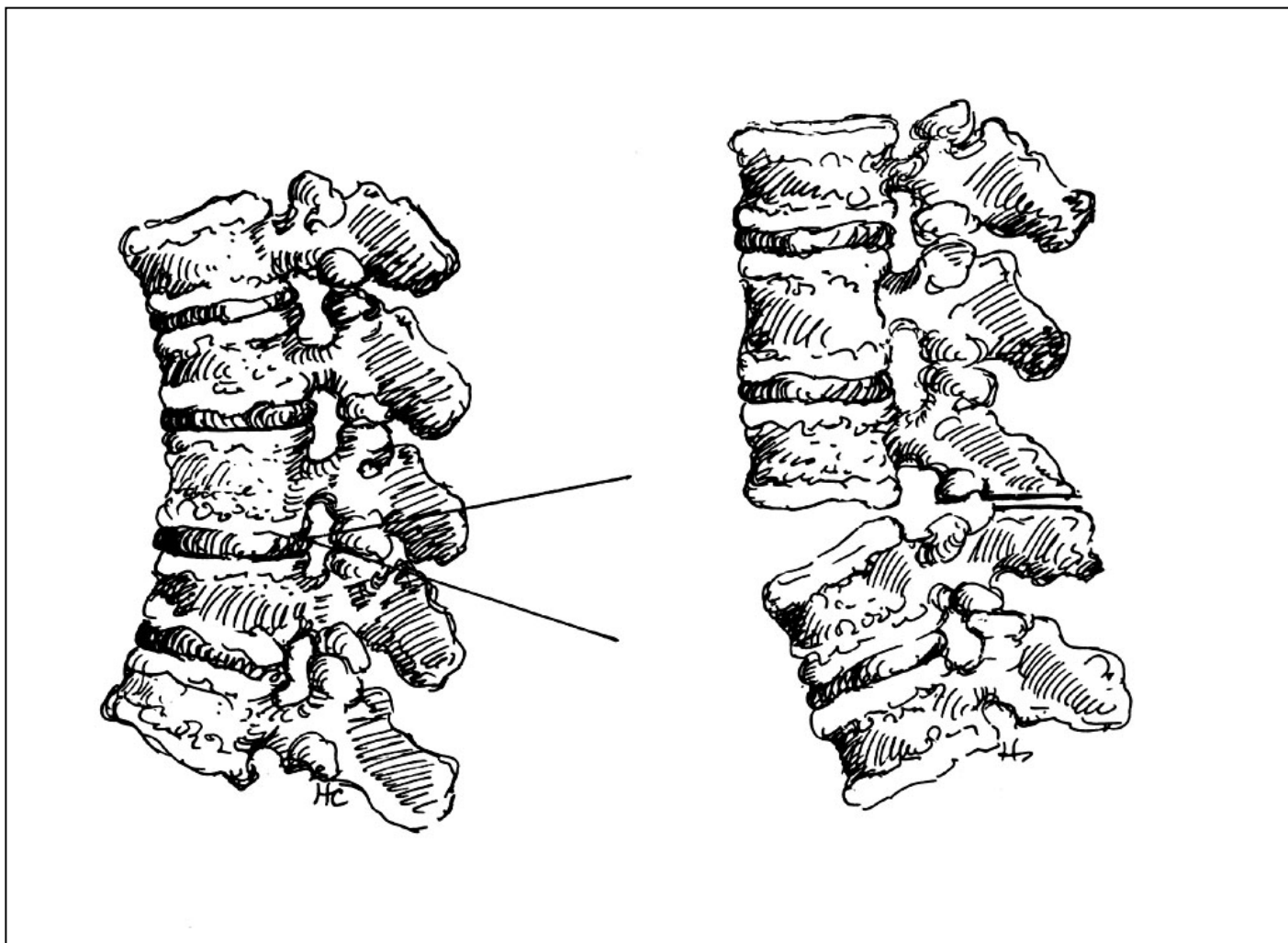


Figure 3. Illustration of pedicle subtraction osteotomy (PSO) technique.

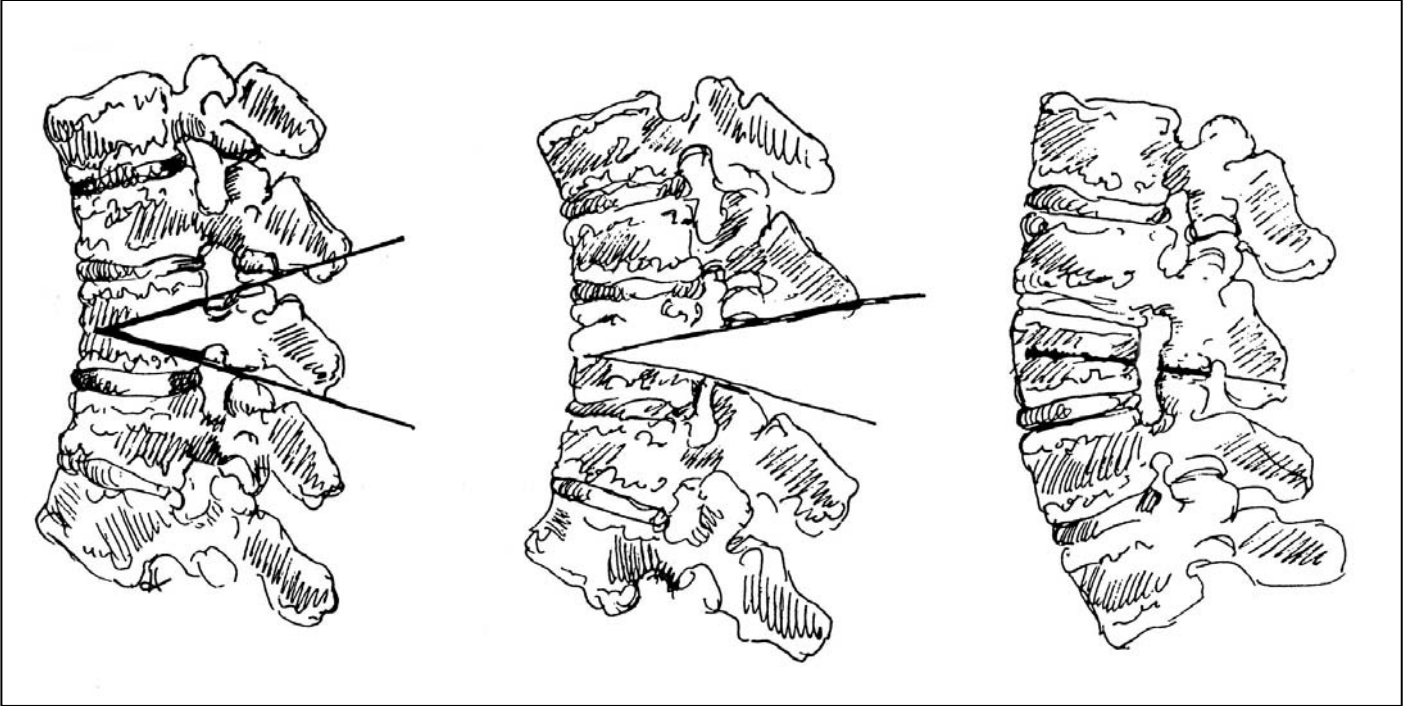


Figure 4. Cervical illustration of Smith-Petersen osteotomy technique.

