# Sagittal Translation in Opening Wedge Osteotomy for the Correction of Thoracolumbar Kyphotic Deformity in Ankylosing Spondylitis

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**Study Design**. Retrospective.

**Objectives**. To document sagittal translation (ST) of the vertebral column in opening wedge osteotomy (OWO) for ankylosing spondylitis (AS) kyphosis.

**Summary of Background Data**. Surgeons recognize displacement at the hinge of correction in OWO as an accidental event between the vertebrae on the side of OWO rather than ST of the cranial or caudal vertebral column. No report documents ST in this context.

**Methods**. We evaluated radiographic and clinical results in 127 patients (aged 17–49 years) for ST, or displacement of more than 2 mm between the cranial and caudal vertebral columns at the OWO level, over 5 years.

**Results**. Thirty-four (27%) patients had ST. In 31 (94%), the kyphotic apex was at T11 to L4. ST was positively correlated with the correction of lumbar lordosis and preoperative lumbar kyphosis. Five (15%) of 34 patients with ST had neurologic complications compared with 2 (2%) of 93 who did not.

**Conclusions**. ST is a basic mechanism for correcting sagittal imbalance and is likely to occur when the level of OWO is near the apex of deformity. Patients needing more correction of lumbar lordosis than others for best correction of sagittal imbalance need ST more to join the mechanism of correction.

**Key words**: ankylosing spondylitis, opening wedge osteotomy, sagittal translation. **Spine 2006;31:1137–1142**

Ankylosing spondylitis (AS) causes flattening of the normal lumbar lordosis; increased, smooth thoracic kyphosis in which the head and neck are thrust forward; and, occasionally, increased flexion at the cervicothoracic junction. Eventually, the whole spine undergoes bony ankylosis. This deformity is functionally and psychologically disabling.

In 1945, Smith-Petersen et al[1] devised osteotomy by which the lumbar spine is hyperextended, enabling the patient to see straight ahead. Many have attempted the operation.[2–10] In 1993, we began applying opening wedge osteotomy (OWO) to correct kyphotic deformity in AS to improve function. Many patients developed sagittal translation (ST) of cranial or caudal vertebral column at the level of OWO during correction (Figures 1–3). We found no report of this phenomena; its clinical meaning is poorly understood.

Our purpose was to document the frequency and nature of ST during OWO and to consider its implications, predisposing factors, and complications.

## ■ Materials and Methods

Between 1993 and 2002, 127 patients (101 men, 26 women; mean age, 35.2 years; range, 17–49 years) with AS underwent OWO to correct fixed kyphotic deformity. Most could not look straight ahead or lie flat. Patients had restricted interpersonal communication and activity (e.g., driving a car, walking, shaving), compression of the viscera and indigestion, and/or psychosocial impairment.

Their clinical records were reviewed for demographic data and complications. Patients with follow-up longer than 2 years answered a modified Scoliosis Research Society outcome questionnaire[11,12] at final follow-up.

Standing anteroposterior and lateral radiographs were obtained before and after surgery. Sagittal measurements were made on 36-in. lateral views of the spine obtained with the patient's hips and knees fully extended. Sagittal offset was measured as the horizontal distance between the C7 sagittal plumb line and the posterior sacral prominence. Cobb measurements for T3–T12 kyphosis and L1–L5 lordosis were recorded. To evaluate the effect of the magnitude of correction on the incidence of ST, corrections related to lumbar lordosis and sagittal balance were calculated.

We classified kyphosis according to the location of its apex: thoracic, T3–T11; thoracolumbar, T11–L1; and lumbar, L1–L4.

ST was determined by comparing preoperative and postoperative standing lateral radiographs. ST was any measurable displacement more than 2 mm between the posterior inferior edge of the cranial vertebral body and the posterior superior edge of the caudal body at the OWO level.

Statistical analysis included calculation of Spearman correlation coefficients, analysis of variance, multiple linear regression, and measurements of agreement (weighted kappa test).

**Surgical Techniques.** All procedures were performed by monitoring somatosensory-evoked potentials. Under general anesthesia, patients were placed prone on an operating table flexed in a reverse "V" shape. The lumbar spine was exposed through a midline incision. Subperiosteal dissection was performed to expose the posterior elements as far lateral as the transverse processes. Pedicle screws were inserted into segments above and below the osteotomy level.
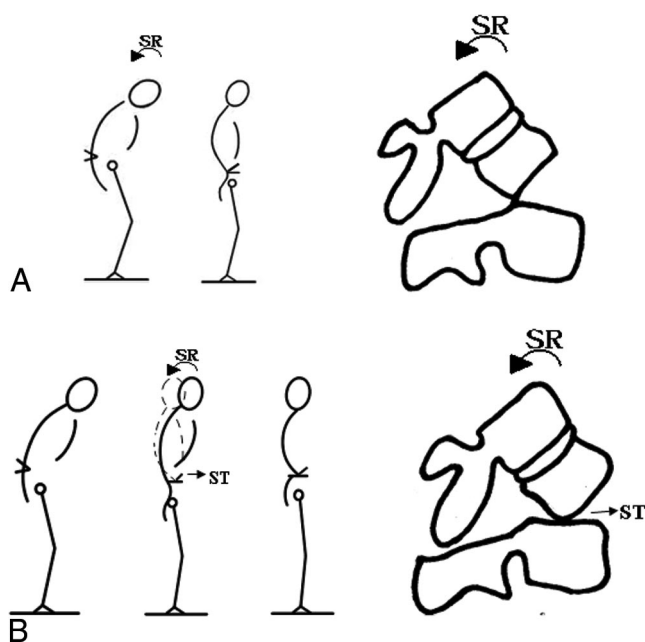
Figure 1. Mechanisms of correcting sagittal imbalance in AS kyphosis by OWO. Top, One mechanism is sagittal rotation (SR) of the cranial and/or caudal vertebral column to create a compensatory lumbar lordosis with a posterior closing wedge and an anterior opening wedge. Bottom, Another mechanism is sagittal translation (ST) of the cranial or caudal vertebral column in addition to sagittal rotation.

The OWO technique was basically that Smith-Petersen et al described.[1] The preferred site was distal to the L1 and usually between L2 and L3 because the spinal canal is relatively spacious here, helping to prevent cord injury. This site was far enough from the sacrum to allow the application of the internal fixation device. An oblique osteotomy was made in the spinous processes one level above and below the central vertebra. Osteotomy with removal of the bony elements delineated an opening equal to the height of two vertebral bodies and wide enough to include the facet joints. The spinal canal was enlarged prox-
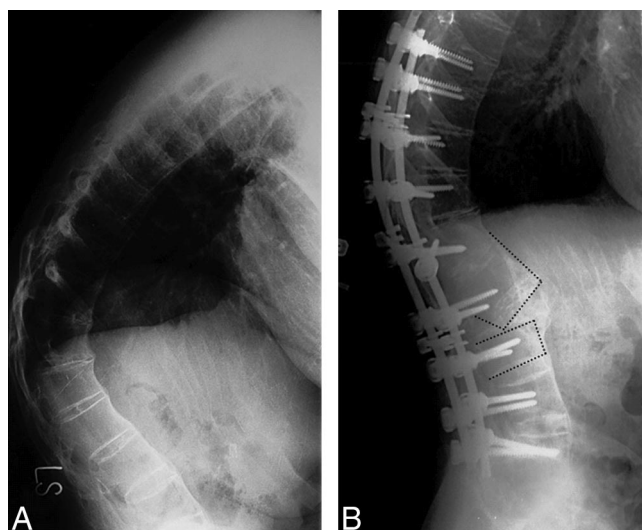


Figure 2. Radiographs in a patient with kyphosis due to AS before (left) and after (right) OWO to correct the deformity. Mechanism of correction appears to be sagittal rotation with translation of the cranial vertebral column.
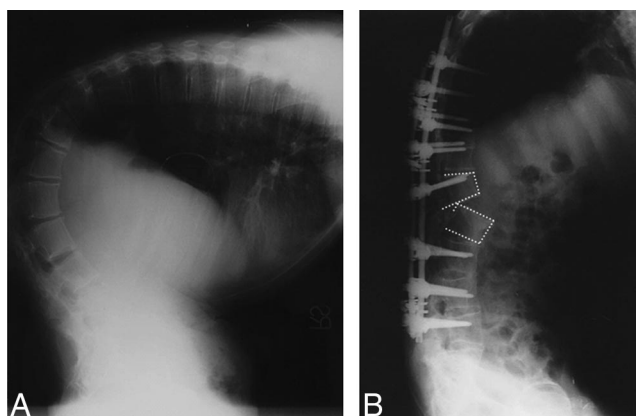


Figure 3. Radiographs in another patient with kyphosis due to AS before (left) and after (right) OWO to correct the deformity. Mechanism of correction appear to be sagittal rotation with translation of the caudal vertebral column.

imally and distally to permit introduction of the tip of the little finger. The bone was carefully cut to avoid spicules and to protect the nerve tissue. We avoided compressing the nerve roots during correction and removed enough bone from the apposing pedicles to ensure that they roots were not pinched as the osteotomy was closed.

Fracture of anterior vertebral column usually occurred by gravity of the patient's trunk or by light pushing on the back after osteotomy. The audible crack was related to the fracture of the calcified anterior longitudinal ligament. When fracture did not occur, a fluoroscopically guided osteotome was cautiously punched through the disc at the osteotomy level to penetrate the anterior longitudinal ligament bilaterally. A light push on the patient's back accomplished the fracture. Sudden force was avoided. Elevation of the head and pelvic ends of the table caused the spine to hinge at the osteotomy level. Closing of the lateral osteotomies and shortening of the vertebral canal opening were observed to ensure that the site of eventual closure, about which correction would occur, was anterior to the osteotomy site. The cauda equina relaxed as the spinal column was extended and the osteotomy closed. After correction, resected bone (cancellous bone chips) was introduced on the raw bone surfaces.

OWO aimed to achieve the best possible sagittal balance. While we closed the osteotomy, the correction proceeded until the patient's shoulder reached the same horizontal line as the pelvis. Thus, we could approximate the C7 plumb line to S1. After we confirmed that the exiting nerve roots were free, we stabilized the pedicle screws. Wake-up testing was performed during closure.

**Postoperative Management.** Patients were allowed to sit up in bed 24 hours after surgery and ambulate with a custom-made plastic thoracolumbosacral orthosis after 3 days. The orthosis was used for approximately 3 to 6 months.

### ■ Results

OWO was performed between L1 and L2 in 8 patients, between L2 and L3 in 112 patients, and between L3 and L4 in 7 patients. Table 1 lists the radiographic results. Thirty-four (27%) of 127 patients had ST (cranial vertebral column in 32, caudal vertebral column in 2).

## Table 1. Radiographic Data

| Data | Preoperative | Postoperative | Correction |
|---|---|---|---|
| Thoracic kyphosis (°) | 56 ± 16 (48–67) | 58 ± 13 (48–71) | NA |
| Lumbar lordosis (°) | −1 ± 11 (−26 to 10) | 31 ± 13 (22–50) | 32 ± 11 (20–68) |
| Sagittal imbalance (mm) | 144 ± 55 (93–274) | 37 ± 8 (13–68) | 107 ± 41 (40–213) |

NA = not applicable. Data are the mean ± standard deviation (range).

The incidence of ST depended on the preoperative curve (Figure 4). Fifteen patients had lumbar deformity, about 60% of whom had ST. Sixty-five had thoracolumbar deformity, 34% of whom had ST, and 47 had thoracic deformity, 6% of whom had ST. OWO at or near the apex seemed to increase the possibility of ST.

Absolute values of preoperative and postoperative thoracic kyphoses, sagittal balance, postoperative lumbar lordosis, and magnitude of correction of sagittal imbalance did not influence the incidence of ST. On linear regression, the magnitude of correction of lumbar lordosis and the degree of preoperative lumbar kyphosis did influence the incidence of ST. The incidence increased with the amount of this correction (Figure 5) and the absolute value of preoperative lumbar kyphosis (Figure 6).

In 34 patients with ST, linear regression did not show ann association between the magnitude of ST and preoperative and postoperative thoracic kyphosis, lumbar lordosis, sagittal offset, and the magnitude of correction of lumbar lordosis and sagittal offset.

### Complications

No vascular complications occurred (Table 2). Eleven dural tears occurred when the ossified ligamentum flavum was dissected away from the midline before the osteotomy was extended and when the dura was adherent to the ligamentum and could not be separated. In 2 patients, the dura was sutured; and in 9 patients, Spongostan was placed on it; they recovered uneventfully.

Among patients without ST, postoperative pneumonia and superficial infection occurred (2 each). Among patients with ST, postoperative pneumonia and superficial infection occurred (1 each). All recovered without adverse effect.

No permanent neurologic deficits were directly due to osteotomy, but 7 patients had early postoperative deficits. All (2 without ST, 5 with ST) had a weak quadriceps on one side. Intraoperative somatosensory-evoked potentials predicted none of these deficits, which were detected during intraoperative wake-up testing in 2 patients and immediate postoperative examination in 5 patients. Five (15%) of 34 patients with ST had neurologic damage compared with 2 (2%) of 93 without ST. Two without ST and 1 with ST had neurologic complications due to nerve root compression by remaining lamina. Four patients with ST had a weak quadriceps due to nerve root compression by a remaining pedicle (Figure 7). All 7 responded to additional central canal enlargement or complete pediculectomy and recovered. Three patients without and one with ST had nonunion and a broken rod at the OWO site; after revision, they healed uneventfully.

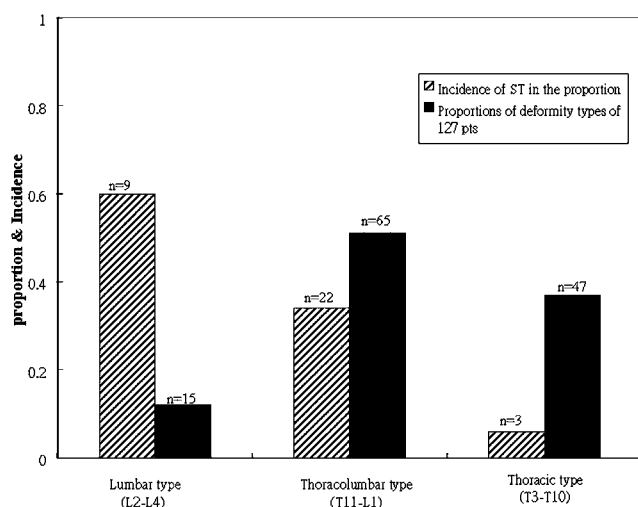Table 3 shows the Scoliosis Research Society scores, which did not differ between groups.



Figure 4. Bar graph shows the proportions of each deformity type in the 127 patients and the incidence of ST of the proportion. Lumbar type, and to a lesser extent thoracolumbar type, had an increased incidence of ST.
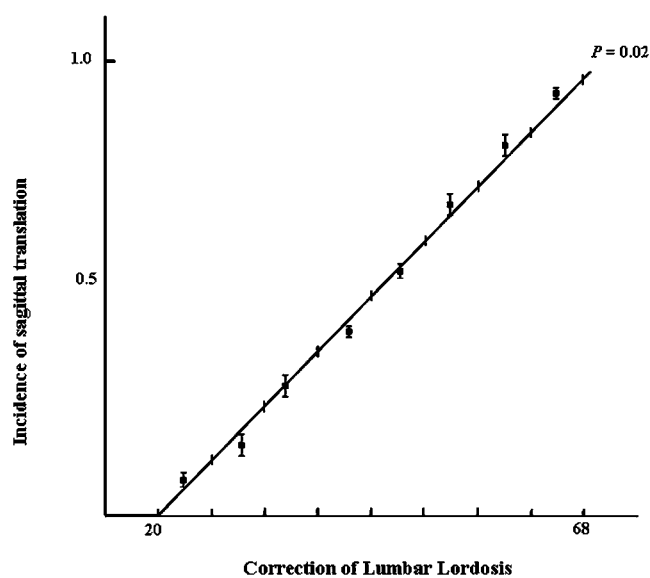


Figure 5. Linear regression showed that the incidence of ST increased as the amount of correction of lumbar lordosis increased. Incidence was the number of patients with ST in the range of correction with increments of every 6° divided by the number of patients in the range of correction with increments of every 6°.
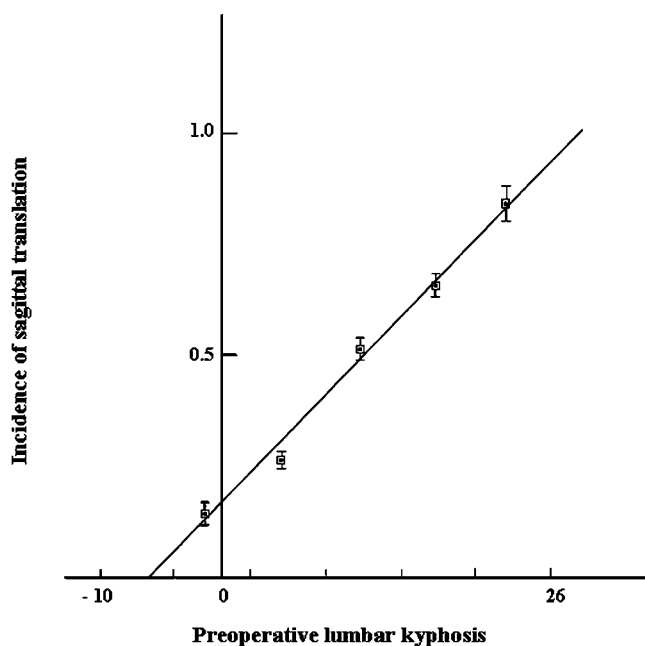
Figure 6. Linear regression shows that the incidence of ST increased as the value of preoperative lumbar kyphosis increased. Incidence was the number of patients with ST in the range of lumbar kyphosis with increments of every 6° divided by the number of patients in the range of lumbar kyphosis with increments of every 6°.

■ Discussion

AS kyphosis is best corrected with lumbar lordosating osteotomy, as thoracic correction is strongly limited by ankylosis of the costovertebral joints. Overall correction is greatest with lumbar intervention. The relatively narrow spinal canal renders the midthoracic spinal cord more vulnerable to perioperative injury than the cauda equina in its spacious canal.

OWO is limited osteotomy involving only the posterior element. Correction of kyphotic deformity was achieved by moving the flexed table to more neural alignment and by forceful manual extension of the lumbar spine to close the posterior edge. This manipulation disrupted the vertebral column and anterior longitudinal ligament, rotating the cranial or caudal vertebral column in the sagittal plane to create an anterior monosegmental intervertebral opening wedge with a hinge at the posterior disc edge at the OWO level and a compensatory lordosis of the lumbar spine. The hinge is not fixed; it is fractured and free to slide. The cranial or caudal vertebral column might develop ST at
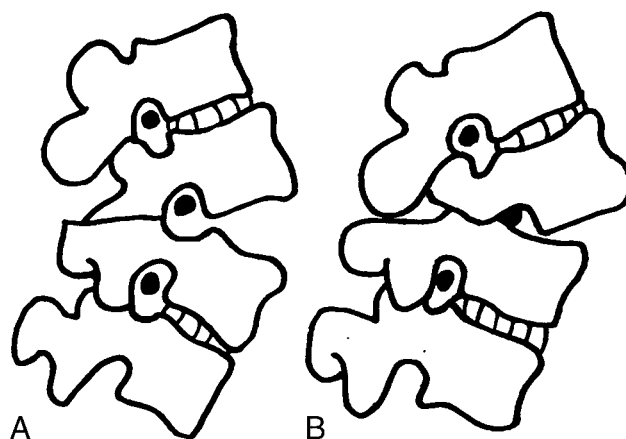


Figure 7. Complications. Left, Rarely, a nerve root is compressed by pedicle in the neural foramen without displacement and rotation of a vertebra. Right, A pedicle can nip a nerve root due to vertebral displacement and rotation.

the hinge to improve sagittal balance if correction achieved by initial closing the posterior wedge is insufficient or inadequate to approximate best possible sagittal balance. ST effectively improves sagittal balance; a few millimeters of hinge displacement may achieve a few centimeters of correction.

Sagittal rotation (SR) of the cranial or caudal column at the hinge is the main mechanism for correcting sagittal malalignment with OWO. Most recognize displacement at the hinge as an accidental, unwanted event between the vertebrae by the side of OWO rather than ST of the cranial or caudal vertebral column. ST is a basic mechanisms by which OWO corrects AS kyphosis. We documented this previously unrecognized ST. Twenty-seven percent of our 127 consecutive patients presenting for OWO developed ST during corrective procedures.

OWO divides the ankylosing spine into cranial and caudal vertebral columns, the curvatures and radiuses of which are not equal. The greater these differences between the two columns, the greater the likelihood of ST. Because lumbar OWO was performed, the rotating radius was longer in cranial column than in the caudal column. OWO at or near the apex of deformity resulted in the largest difference in curvatures and increased the incidence of ST. In 94% of our patients, ST occurred when the apex was at T11–L4 (Figure 4).

### Table 2. Complications

| Complication | OWO Without ST (n = 93) | OWO With ST (n = 34) |
| --- | --- | --- |
| Dura laceration | 8 | 3 |
| Pneumonia | 2 | 1 |
| Superficial infection | 2 | 0 |
| Transient radiculopathy | 2 | 5 |
| Nonunion or rod broken | 3 | 1 |

### Table 3. Modified SRS Outcomes Instrument Scores

| Domain | Patients Without ST (n = 93) | Patients With ST (n = 34) |
| --- | --- | --- |
| Pain | 4.23 ± 0.71 | 4.12 ± 0.81 |
| Self-image/appearance | 4.11 ± 0.51 | 4.01 ± 0.44 |
| Function/activity | 3.98 ± 0.53 | 4.01 ± 0.45 |
| Mental health | 3.97 ± 0.55 | 3.87 ± 0.74 |
| Satisfaction with surgery | 4.65 ± 0.55 | 4.55 ± 0.38 |

Data are the mean ± standard deviation.

Patients who need more correction of lumbar lordosis than others to approximate best correction need more ST to join the mechanism of correction with OWO and initial closing of the posterior wedge. As the magnitude of correction increased, the incidence of ST increased (Figure 5). In patients with AS-related lumbar kyphosis, the apex is usually at or near the level of OWO; these patients typically need more correction and are therefore predisposed to ST (Figure 6).

The incidence of ST and the preoperative sagittal offset and magnitude its correction were not related possibly because sagittal offset depends on the angle of kyphosis and trunk height, which varies. A tall patient with a small kyphotic angle may have a large sagittal offset, but only small angular correction is required for optimal correction of sagittal imbalance. SR is enough, and ST does not have the opportunity to join the mechanism of correction.

In 34 patients with ST, linear regression found no association between the magnitude of ST and preoperative angle of kyphosis, sagittal offset and the magnitude of correction of lumbar lordosis and sagittal offset. SR is the main mechanism of correction with OWO. The magnitude of ST depends on how much correction is accomplished with SR, which varies.

Although the cranial vertebral column is more amenable than the caudal column to the correction of sagittal imbalance because of its longer radius, both columns on the side of OWO can develop ST, depending on which is manipulated more. Correction usually involved the patient's shoulder, which was manipulated and raised more than the pelvis. Therefore, the cranial column generally developed ST. Two patients had ST of caudal column. In both, the pelvis was manipulated and raised more than the shoulder because airway tubes prevented us from elevating the shoulder.

The mechanism of correction by OWO is SR alone or with ST. The distance between the apex and the osteotomy site, the magnitude of correction of lumbar lordosis, and the lumbar-predominant kyphosis determine the mechanism. In addition, how surgeons manipulate the cranial and caudal columns, the resistance and friction at the hinge, and the elasticity and tension of soft tissue around the osteotomy site are other determinants but difficult to analyze. We believe ST joins the mechanism when SR is insufficient or inadequate for optimal correction and cannot be avoided without a fixed hinge by OWO.

Recognizing ST in OWO for AS kyphotic deformity has important implications. The language of deformity correction (opening wedge, closing wedge) must accommodate ST. Furthermore, ST must be appreciated when OWO is used to treat AS kyphosis and when the restoration of sagittal alignment is interpreted. It now seems untenable to report that OWO increases lumbar lordosis to correct sagittal imbalance but does not carefully control for the occurrence of ST. The corollary assumption is that OWO is designed to improve the sagittal malalignment and posture deficit resulting from AS by using SR to create a compensatory lumbar lordosis with a posterior closing wedge and an anterior opening wedge. However, the improvement in sagittal balance in a remarkably proportion of patients with AS kyphosis (27%) was due to ST in addition to SR.

Rupture of the aorta or its branches is associated with reduced elasticity of the aortic wall in older patients and a sharp lordotic angle and elongation of the anterior column. This small risk cannot be ignored.[13–15] Vascular injury occurs when the opening wedge is performed at L1–L2 or L2–L3.[16,17] OWO causes the aorta opposite the anterior opening wedge to stretch during correction. OWO with ST subjects the aorta to an anteriorly directed force in addition to stretching. Therefore, avoiding spike formation at the anterior edge of the opening wedge is critically important to prevent a penetrating injury of the aorta.

Many have used OWO in which the spine is osteotomized through the posterior elements and corrected with direct pressure on the osteotomy site. The patient's upper body and legs are extended to form a hollow cavity between the ventral trunk and the table, and the pressure causes the ossified anterior vertebral column to fracture, often with a sudden snap, which suddenly stretches the aorta opposite the anterior opening wedge. The osteoclasis thus created might avulse a bone fragment from the vertebral body to form a spike, which could injure the aorta while the anterior wedge is opened. In our patients, the osteoclasis usually occurred at the intervertebral disc at the level of osteotomy during posterior osteotomy with gravity or light pressure on the patient's trunk. If the ossified anterior vertebral column was too hard, a fluoroscopically guided osteotome was placed through the intervertebral disc at the level of the osteotomy. Then, osteoclasis at the anterior disc space was formed by gentle manipulation. Correction should not be started without first ensuring osteoclasis; it is then accomplished by a slow and finely controlled closure of the osteotomy. As the posterior wedge is closed, correction occurs in the anterior vertebral column with opening of the anterior disc space, without any avulsed bone fragment to form a spike. None of our patients had vascular injury.

Complications other than neurologic deficits did not significantly differ. The risk of nerve root injury in patients with ST was substantial; ST poses a risk of neurologic complications because of displacement and rotation of a vertebral body. Compression of a dorsal nerve root by the lamina can result from closure of the osteotomy. Enough lamina should be removed to prevent this complication. A nerve root can also be nipped by pedicle (Figure 7). Four of five patients with ST and neurologic deficits had root compression due to a remaining pedicle. The nerve root must be examined during surgery after correction and adequate pediculectomy. Because it is difficult to perform after SR with ST, we suggest routine pediculectomy before the corrective maneuver in patients predisposed to ST (e.g., patients with a predominant lumbar kyphosis or patients who need more correction of lumbar lordosis for optimal correction).

ST significantly increased the incidence of neurologic deficits. All patients responded well to prompt neurologic decompression and recovered without adverse effect. Outcome analysis showed no significant difference between our groups.

Our observations have limitations. Our definition of ST has not been reported and must be standardized. Although definition bias might have alerted the relative frequencies, it does not change our observation that ST is a basic mechanism of correcting sagittal balance with OWO.

ST must be considered when OWO is performed to manage kyphotic deformity in AS and when restoring lumbar lordosis to improve sagittal balance is discussed. The nosology, definition, and epidemiologic study of ST in OWO; the associated complications due to ST; and the distinction of displacement between the vertebrae on the side of OWO and ST of the vertebral column must be reconsidered.

### ■ Key Points

- Sagittal translation of the cranial or caudal vertebral column occurred in 34 (27%) of 127 patients with ankylosing spondylitis who underwent opening wedge osteotomy for kyphotic deformity.
- Sagittal translation is one of the basic mechanisms of opening wedge osteotomy for the correction of sagittal imbalance.
- Sagittal translation is more likely to occur when the level of the opening wedge is near the apex of the deformity than when it is not.
- Patients who need more correction of lumbar lordosis than others to approximate the best possible correction of sagittal imbalance also need more sagittal translation to join the mechanism of correction.

- Sufficient laminectomy and pediculectomy are important to avoid neurologic complications in cases of sagittal translation.

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